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Linkage Map of *Salmonella typhimurium*, Edition VII

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INTRODUCTION

We present edition VII of the linkage map of *Salmonella typhimurium*. We list a total of 750 genes; 680 of these are located on the linkage map, and the remaining 70 are not yet mapped, although mutant alleles are known. The first edition of the map (411) contained 133 genes. Later editions were published in 1967 (408), 1970 (409), 1972 (410), 1978 (412), and 1983 (418).

All genes presently known to us are indicated in Table 1, with formerly used gene symbols in Table 2, but references presented in earlier editions of the map are not normally shown. Therefore, to find all relevant references to a gene, it may be necessary to check earlier editions of the map; where references were given in earlier editions, this edition is shown in Table 1. In a recent summary of the cellular and molecular biology of *Escherichia coli* and *S. typhimurium* (346), edition VI of the linkage map of *S. typhimurium* is presented (413), which contains most of the relevant references published up to and including edition VI.

THE LINKAGE MAP

The coordinate system used in early editions of the linkage map of *S. typhimurium* was determined by F-mediated conjugation. Hfr strains were used to place P22 transduction linkage groups on a 138-min time-of-entry linkage map. In edition V, the map was changed to 100 units to correspond to the 100-min linkage map of *E. coli* K-12. This was done to emphasize the similarity of the two organisms and to facilitate comparisons with this closely related bacterium. The change was justified because the measure derived from F-mediated conjugation, the "minute of entry time," had become less important as a measure of distance than transduction frequency or, more recently, restriction fragment lengths or DNA sequences. The second change was that although the linkage map is circular, for convenience it was not displayed as a circle but as 10 linear 10-min intervals (Fig. 1).

The same system has been retained in this seventh edition (Table 1). The 100 units of the map are based on P22, "phage lengths." P22 can normally encapsulate about 45 kilobases (kb) of DNA; this is approximately 1% of the *Salmonella* chromosome.

Nomenclature

We use the system of nomenclature for genes which was established by Demerec et al. (106). This system has become the de facto standard for bacterial genetics. Authors considering a three-letter designation for a new gene in *S. typhimurium* should check the published maps of *S. typhimurium*

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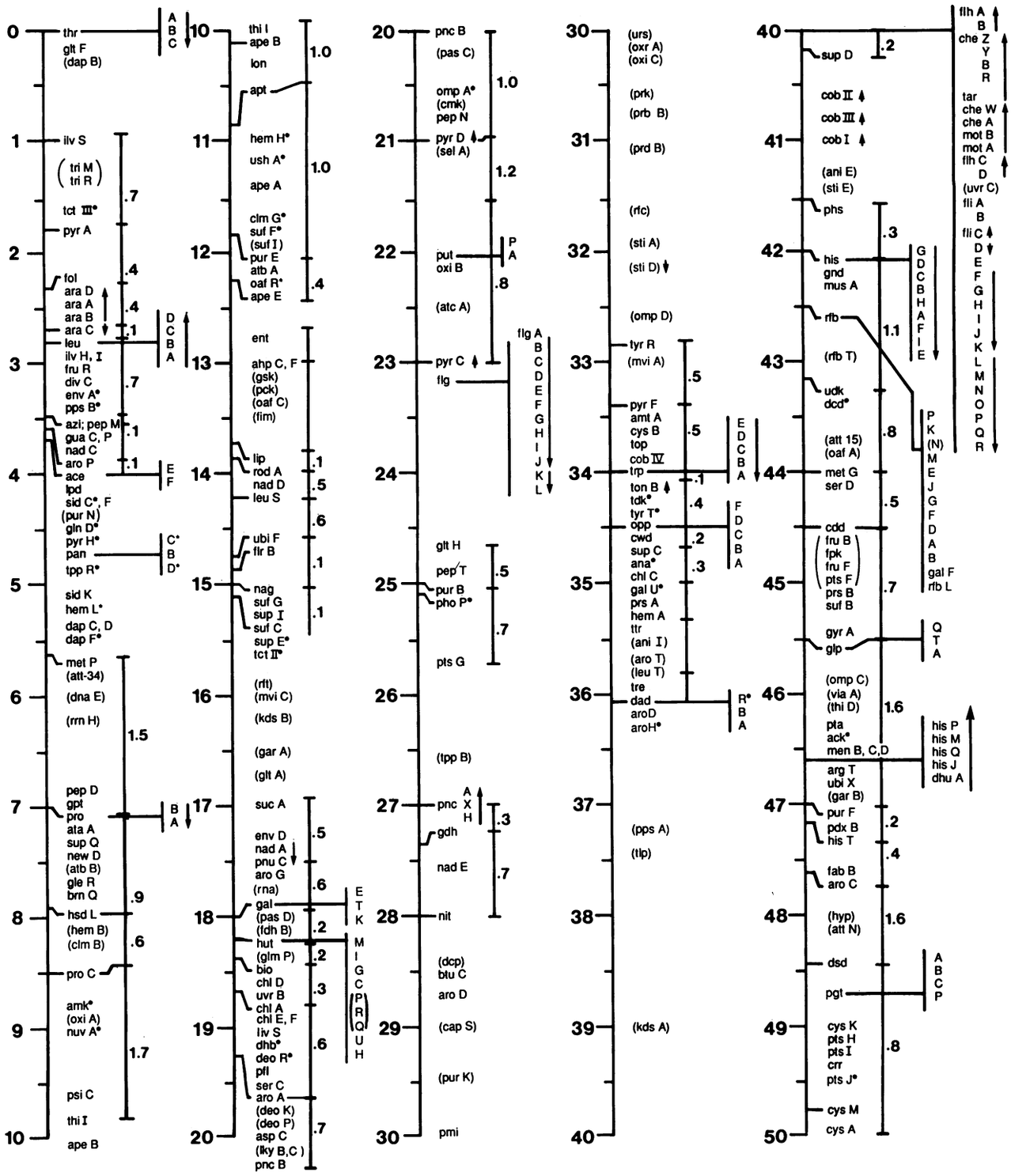

FIG. 1. Linkage maps of *S. typhimurium*, represented as 10 segments. The scale of 100 min begins at zero for the *thr* loci, as in previous maps (418) and in the linkage maps of *E. coli* (14). The segmented line to the right of the gene symbol indicates that the genes are jointly transduced; the numbers to the right of the segmented line indicate the linear distance between genes. This linear distance was determined from the fragment of joint transduction and was calculated by assuming that the length of P22, KB1, and ES18 transducing fragments is 1 min, whereas that of P1 is 2 min, and applying the formula developed by Wu (520) to convert the percentage of joint transduction to map distance. The genetic symbols are defined in Table 1. Parentheses around a gene symbol indicate that the location of the gene is known only approximately, usually from conjugation studies. An asterisk indicates that a gene has been mapped more precisely, usually by phage-mediated transduction, but that its position with respect to adjacent markers is not known. Arrows to the extreme right of genes and operons indicate the direction of mRNA transcription by these loci. Daggers are shown to the right of a few genes; these genes of *S. typhimurium* are carried on an F-prime factor, and this plasmid was shown to complement *E. coli* K-12 mutations of that gene; mutant alleles of *S. typhimurium* have not been tested directly.

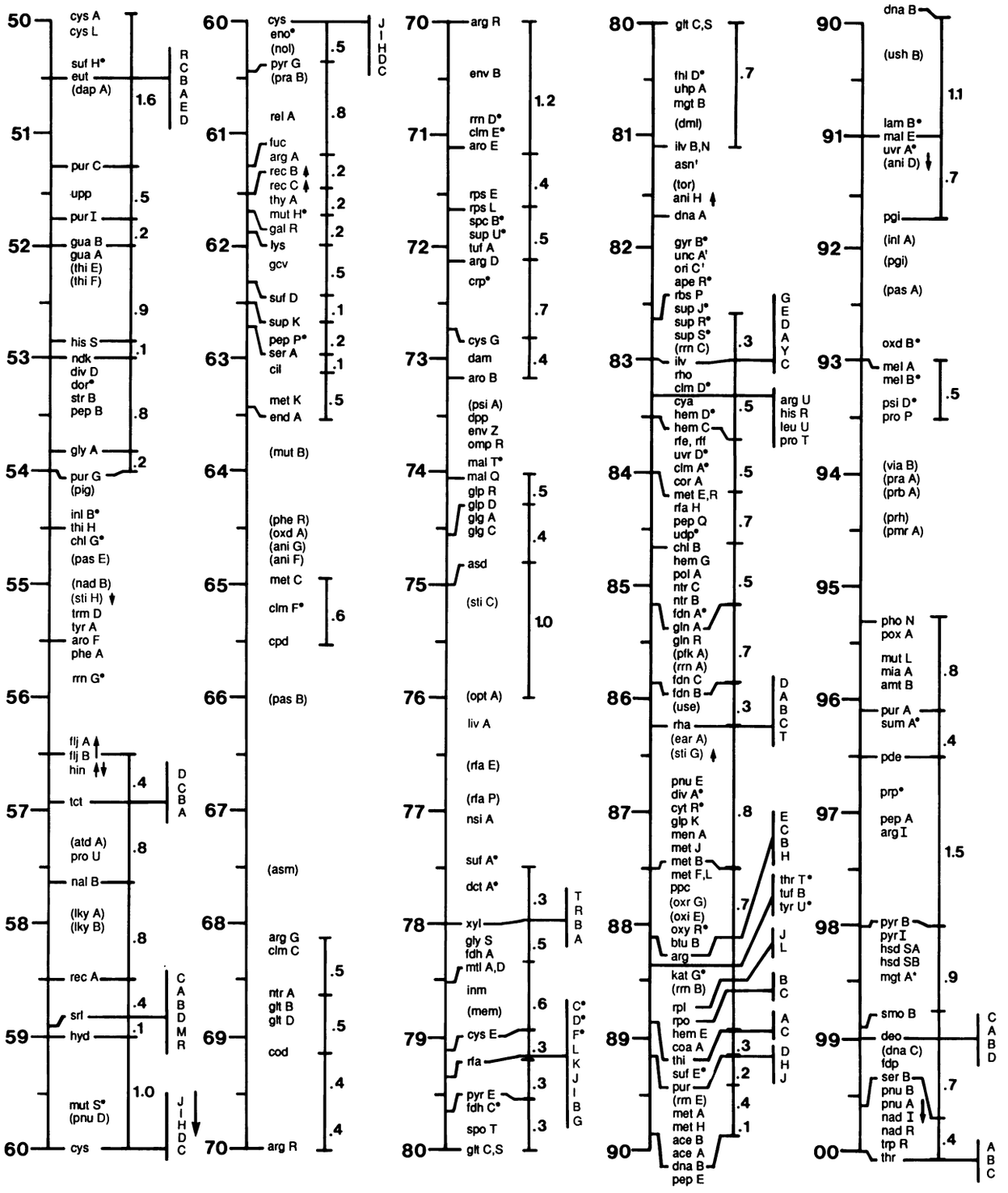
50 cys A
cys L
suf H*
eut
(dap A)
1.6
R C B A E D
51
pur C
upp
.5
pur I
.2
52 gua B
gua A
(thi E)
(thi F)
.9
his S
.1
53 ndk
div D
dor*
str B
pep B
.8
gly A
.2
54 pur G
(pig)
inl B*
thi H
chl G*
(pas E)
55 (nad B)
(sti H)
trm D
tyr A
aro F
phe A
rrn G*
56
flj A
flj B
hin
.4
D C B A
57 tct
(atd A)
.8
pro U
nal B
58 (lky A)
(lky B)
.8
rec A
.4
C A B D M R
srl
59 hyd
.1
1.0
J I H D C
mut S*
(pnu D)
60 cys
60 cys
eno*
(nol)
.5
J I H D C
pyr G
(pra B)
rel A
.8
61
fuc
arg A
rec B
.2
rec C
thy A
.2
mut H*
gal R
.2
62 lys
gcv
.5
suf D
.1
sup K
pep P*
.2
ser A
63 cil
.1
met K
.5
end A
(mut B)
64
(phe R)
(oxd A)
(ani G)
(ani F)
65 met C
clm F*
.6
cpd
66 (pas B)
(asm)
68
arg G
clm C
.5
ntr A
.5
glt B
glt D
69 cod
.4
.4
70 arg R
70 arg R
env B
1.2
rrn D*
71 clm E*
aro E
.4
rps E
rps L
spc B*
.5
sup U*
72 tuf A
arg D
crp*
.7
cys G
73 dam
.4
aro B
(psi A)
dpp
env Z
omp R
mal T*
74 mal Q
glp R
.5
glp D
glg A
.4
glg C
asd
75
(sti C)
1.0
76 (opt A)
liv A
(rfa E)
(rfa P)
77 nsi A
suf A*
dct A*
.3
T R B A
78 xyl
gly S
.5
fdh A
mtl A,D
inm
.6
C* D* F* L K J I B G
(mem)
79 cys E
.3
rfa
.3
pyr E
fdh C*
.3
80 spo T
glt C,S
80 glt C,S
.7
thl D*
uhp A
mgt B
(dml)
81
ilv B,N
asn'
(tor)
ani H
dna A
82
gyr B*
unc A'
ori C'
ape R*
rbs P
sup J*
G E D A Y C
sup R*
sup S*
(rrn C)
.3
83 ilv
rho
clm D*
cya
.5
arg U his R leu U pro T
hem D*
hem C
rfe, rff
uvr D*
84 clm A*
.5
cor A
met E,R
rfa H
pep Q
.7
udp*
chl B
hem G
pol A
85
.5
ntr C
ntr B
fdn A*
gln A
gln R
.7
(pfk A)
(rrn A)
fdn C
D A B C T
86 fdn B
(use)
.3
rha
(ear A)
(sti G)
pnu E
div A*
cyt R*
.8
E C B H
87 glp K
men A
met J
met B
met F,L
thr T*
ppc
.7
tuf B
tyr U*
88 (oxr G)
(oxi E)
oxy R*
J L
btu B
arg
kat G*
B C
(rrn B)
rpl
A C
rpo
89 hem E
coa A
.3
D H J
thi
suf E*
.2
pur
(rrn E)
.4
met A
met H
.1
ace B
ace A
90 dna B
pep E
90 dna B
(ush B)
1.1
lam B*
91 mal E
uvr A*
(ani D)
.7
pgi
92 (inl A)
(pgi)
(pas A)
oxd B*
93 mel A
mel B*
.5
psi D*
pro P
(via B)
94 (pra A)
(prb A)
(prh)
(pmr A)
95
pho N
pox A
mut L
.8
mia A
amt B
96 pur A
sum A*
.4
pde
prp*
97 pep A
arg I
1.5
pyr B
pyr I
98 hsd SA
hsd SB
mgt A*
.9
smo B
C A B D
99 deo
(dna C)
fdp
ser B
.7
pnu B
pnu A
nad I
nad R
trp R
.4
A B C
00 thr

TABLE 1. Genes of *S. typhimurium*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
aceA	Acetate	Growth on acetate or fatty acids; isocitrate lyase (EC 4.1.3.1)	*aceA*	89	418, 517
aceB	Acetate	Growth on acetate or fatty acids; malate synthase (EC 4.1.3.2)	*aceB*	89	418, 517
aceE	Acetate	Acetate requirement; pyruvate dehydrogenase (pyruvate:cytochrome b_1 oxidoreductase; EC 1.2.2.2)	*aceE*	3	412, 418
aceF	Acetate	Acetate requirement; pyruvate dehydrogenase (pyruvate lipoate oxidoreductase; EC 1.2.4.1)	*aceF*	3	412, 418
ack	Acetate kinase	Acetate kinase (ATP:acetate phosphotransferase, EC 2.7.2.1)	*ackA*	46	272, 418, 498
ahpC	Alkyl hydroperoxide	Alkyl hydroperoxide reductase, C22 subunit	NM	13	AB
ahpF	Alkyl hydroperoxide	Alkyl hydroperoxide reductase, F52a subunit	NM	13	AB
alr	Alanine racemase	Biosynthetic alanine racemase (EC 5.1.1.1)	*alr*	NM	127, 129, 145, 146
amk		AMP kinase		9	412, 418
amtA		Resistance to 40 mM 3-amino-1,2,4-triazole in the presence of histidine		33	410, 412, 418
ana		Anaerobic gas production	*ana*	35	192
aniB	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		93	4
aniC	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		93	4
aniD	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		92	4
aniE	Anaerobically inducible	May be the same as *phs*		41	4
aniF	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		63	4
aniG	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		63	4
aniH	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		81	4
aniI	Anaerobically inducible	Induced by anaerobiosis; does not reduce benzyl viologen		35	4
apeA	Acyl peptide esterase	Acyl amino acid esterase (hydrolyzes *N*-acetyl-L-phenylalanine-β-naphthyl ester)		11	412, 418
apeB	Acyl peptide esterase	Acyl amino acid esterase (hydrolyzes *N*-acetyl-L-phenylalanine-β-naphthyl ester)		10	412, 418
apeE	Acyl peptide esterase	Membrane-bound acyl amino acid esterase (hydrolyzes *N*-acetyl-L-phenylalanine-β-naphthyl ester)		12	418
apeR	Acyl peptide esterase	apeD; regulatory gene for *apeE*		82	418
aphA		Nonspecific acid phosphatase II		NM	418
apt		Adenine phosphoribosyltransferase		10	412, 418
araA	Arabinose	L-Arabinose isomerase (EC 5.3.1.4)	*araA*	2	285, 296, 297, 412, 418
araB	Arabinose	Ribulokinase (EC 2.7.1.16)	*araB*	2	285, 296, 410, 412, 418
araC	Arabinose	Regulatory gene for arabinose catabolic enzymes	*araC*	2	284, 285, 296, 412, 418
araD	Arabinose	L-Ribulose-phosphate 4-epimerase (EC 5.1.3.4)	*araD*	2	285, 295, 296, 418
argA	Arginine	*argB*; amino acid acetyl transferase (EC 2.3.1.1)	*argA*	61	342, 410, 412, 418, 443
argB	Arginine	*argC*; *N*-acetyl-γ-glutamate kinase (EC 2.7.2.8)	*argB*	88	410, 412, 418
argC	Arginine	*argH*; *N*-acetyl-γ-glutamyl phosphate reductase (EC 1.2.1.38)	*argC*	88	410, 412, 418
argD	Arginine	*argG*; acetylornithine aminotransferase (EC 2.6.1.11)	*argD*	72	410, 412, 418
argE	Arginine	*argA*; acetylornithine deacetylase (EC 3.5.1.16)	*argE*	88	37, 410, 412, 418
argG	Arginine	*argE*; argininosuccinate synthetase (EC 6.3.4.5)	*argG*	69	410, 412, 418
argH	Arginine	*argF*; argininosuccinate lyase (EC 4.3.2.1)	*argH*	88	410, 412, 418
argI	Arginine	Ornithine carbamoyltransferase (EC 2.1.3.3)	*argI*	98	410, 412, 418
argP	Arginine	Arginine transport	*argP*	NM	412, 418
argR	Arginine	L-Arginine regulation	*argR*	70	147, 412, 418
argS	Arginine	Arginyl-tRNA synthetase (EC 6.1.1.19)	*argS*	NM	412, 418

Continued on following page

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
argT	Arginine	Lysine-arginine-ornithine-binding protein		46	8, 283, 418, 428, 455
argU	Arginine	*argT*; tRNA structural gene for arginine		83	41, 44
aroA	Aromatic	3-Enolpyruvylshikimate 5-phosphate synthetase	*aroA*	19	121, 196, 349, 410, 412, 418, 442, 453
aroB	Aromatic	5-Dehydroquinate synthetase	*aroB*	73	410, 412, 418
aroC	Aromatic	*aroD*; chorismate synthetase	*aroC*	47	195, 410, 412, 418
aroD	Aromatic	*aroE*; 5′-dehydroquinate dehydratase (EC 4.2.1.10)	*aroD*	36	410, 412, 418
aroE	Aromatic	*aroC*; 5-dehydroshikimate reductase	*aroE*	71	410, 412, 418
aroF	Aromatic	Tyrosine-repressible DAHP synthetase	*aroF*	55	410, 412, 418
aroG	Aromatic	Phenylalanine-repressible DAHP synthetase	*aroG*	17	410, 412, 418
aroH	Aromatic	Tryptophan-repressible DAHP synthetase	*aroH*	36	410, 412, 418
aroP	Aromatic	Aromatic amino acid transport	*aroP*	3	410, 412, 418
aroT	Aromatic	Ability to transport tryptophan, phenylalanine, tyrosine	*aroT*	35	410, 412, 418
asd		Aspartic semialdehyde dehydrogenase (EC 1.2.1.11)	*asd*	75	410, 412, 418
asm		Unable to assimilate low levels of ammonia; deficient in glutamate synthase and glutamine synthase		68	418
asn	Asparagine	Asparagine synthesis	*asn*	81	418
aspC	Aspartate	Aspartate aminotransferase (EC 2.6.1.1)	*aspC*	20	497
ataA	Attachment	*attP22 I*; attachment site for prophage P22	*attP22*	7	393, 410, 412, 418
atbA	Attachment	*attP27 I*; attachment site for prophage P27		12	410, 412, 418
atbB	Attachment	*attP27 II*; second attachment site for prophage P27		7	410, 412, 418
atcA	Attachment	*attP221*; attachment site for prophage P221		22	410, 412, 418
atdA	Attachment	*attP14*; attachment site for prophage P14 in group C *Salmonella* spp.		57	410, 412, 418
ats		Arylsulfatase		NM	418
att15	Attachment	Attachment site of phage ε^{15} to chromosome in group E *Salmonella* spp.		43	412, 418
att34	Attachment	Attachment site in phage ε^{34} to chromosome in group E *Salmonella* spp.		5	412, 418
attN	Attachment	Attachment site for prophage N in *S. montevideo*		48	412, 418
avtA		Alanine-valine transaminase (transaminase C)	*avtA*	NM	29, 514
azi	Azide	Resistant to 3 mM sodium azide on L-methionine	*azi*	3	410, 412, 418
bio	Biotin	Requirement	*bioA*	18	412, 418
brnQ		*ilvT*; branched-chain amino acid transport	*brnQ*	7	311, 353, 412, 418
btuB	B_{12} utilization	*bfe*; transport of vitamin B_{12}	*btuB*	88	412, 418, H
btuC	B_{12} utilization	Transport of vitamin B_{12}	*btuC*	28	H
capS	Capsule	Capsular polysaccharide synthesis	*capS*	29	418
cdd		Cytidine deaminase (EC 3.5.4.5)	*cdd*	44	410, 412, 418
cheA	Chemotaxis	*cheP*; chemotaxis	*cheA*	40	268, 412, 418, 456, 521
cheB	Chemotaxis	*cheX*; chemotaxis. Protein-glutamate methylesterase	*cheB*	40	39, 40, 102, 268, 385, 412, 418, 437, 438, 445, 461
cheR	Chemotaxis	Chemotaxis. Protein-glutamate methyl transferase	*cheX*	40	39, 40, 102, 268, 412, 418, 461, Y
cheS	Chemotaxis	Chemotaxis		NM	412, 418
cheW	Chemotaxis	Chemotaxis	*cheW*	40	268, 412, 418, 458
cheY	Chemotaxis	*cheQ*; chemotaxis	*cheY*	40	268, 412, 418, 445, 457
cheZ	Chemotaxis	*cheT*; chemotaxis	*cheZ*	40	385, 418, 445, 459, 460
chlA	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase	*chlA*	18	89, 410, 412, 418
chlB	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, and hydrogen lyase	*chlB*	84	410, 412, 418
chlC	Chlorate	Retains sensitivity to chlorate; affects nitrate reductase	*chlC*	34	273, 410, 412, 418
chlD	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, and hydrogen lyase	*chlD*	18	410, 412, 418
chlE	Chlorate	Resistance		18	410, 412, 418
chlF	Chlorate	Resistance		18	410, 412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
chlG	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase		20	410, 412, 418
cil	Citrate lyase	Deficiency of the essential enzyme in a temperature-sensitive mutant		63	266
clmA	Conditional-lethal mutation	Heat- or cold-sensitive mutation		84	X
clmB	Conditional-lethal mutation	Heat- or cold-sensitive mutation		8	X
clmC	Conditional-lethal mutation	Heat- or cold-sensitive mutation		69	X
clmD	Conditional-lethal mutation	Heat- or cold-sensitive mutation		83	X
clmE	Conditional-lethal mutation	Heat- or cold-sensitive mutation		71	X
clmF	Conditional-lethal mutation	Heat- or cold-sensitive mutation		64	X
clmG	Conditional-lethal mutation	Heat- or cold-sensitive mutation		11	X
cmk		Cytidylate kinase (EC 2.7.4.14)		89	412, 418
coaA	Coenzyme A	Coenzyme A synthesis; pantothenate kinase		89	412, 418
cobI	Cobalamin	Operon encoding synthesis of cobalamide (vitamin B_{12})		41	128, 141, 234, 235
cobII	Cobalamin	Operon encoding synthesis of DMB; defective in vitamin B_{12} synthesis		41	128, 234, 235
cobIII	Cobalamin	Operon encoding functions for joining cobalamide and DMB; defective in vitamin B_{12} synthesis		41	128, 234, 235
cobIV	Cobalamin	Operon including functions required for vitamin B_{12} synthesis		34	L
cod		Cytosine deaminase (EC 3.5.4.1)		69	410, 412, 418
corA	Cobalt resistance	Magnesium transport	*corA*	84	194
cpd		cAMP phosphodiesterase (EC 3.1.4.17)	*cpd*	64	45, 412, 418, 485
crp		cAMP receptor protein	*crp*	72	37, 97, 107, 110, 240, 410, 412, 418, 430, 512
crr		Factor III for sugar transport by phosphotransferase IIB′ (*ptsG*) system	*crr*	48	107, 316, 317, 318, 329, 341, 375, 377, 406, 410, 412, 418, 485
cwd	Cell wall defect	Sensitive to bile salts; mucoid		34	192
cya	cAMP	Adenylate cyclase (EC 4.6.1.1)	*cya*	83	37, 110, 240, 410, 412, 418, 485, 512
cysA	Cysteine	Sulfate-thiosulfate transport (chromate resistance)	*cysA*	49	221, 332, 341, 370, 410, 412, 418
cysB	Cysteine	Cysteine regulation; positive control of L-cystine transport	*cysB*	33	226, 227, 325, 358, 410, 412, 418
cysC	Cysteine	Adenylylsulfate kinase (EC 2.7.1.25)	*cysC*	60	332, 410, 412, 418
cysD	Cysteine	Sulfate adenylyltransferase (EC 2.7.7.4)	*cysD*	60	332, 410, 412, 418
cysE	Cysteine	Serine acetyltransferase (EC 2.3.1.30)	*cysE*	79	410, 412, 418
cysG	Cysteine	Seroheme component of sulfite reductase	*cysG*	72	234, 410, 412, 418
cysH	Cysteine	Adenylylsulfate reductase (EC 1.8.99.2)	*cysH*	60	410, 412, 418, S
cysI	Cysteine	Heme protein component of sulfite reductase	*cysI*	60	412, 418, S
cysJ	Cysteine	Flavoprotein component of sulfite reductase	*cysJ*	60	410, 412, 418, S
cysK	Cysteine	*trz*; resistance to 1,2,4-triazole; *O*-acetylserine sulfhydrylase A (EC 4.2.99.8)	*cysK*	49	65, 221, 332, 338, 410, 412, 418
cysL	Cysteine	Resistance to selenate		50	412, 418
cysM	Cysteine	*O*-Acetylserine sulfhydrylase B (EC 4.2.99.8)		49	221, 332, 338, 418
cytR		Regulatory gene for *deo* operon and *udp* and *cdd* genes	*cytR*	87	410, 412, 418
dadA		*dad*; D-histidine, D-methionine utilization; D-alanine dehydrogenase (EC 1.4.99.1)	*dadA*	36	410, 412, 418
dadB		Catabolic alanine racemase (EC 5.1.1.1)	*dadB*	36	129, 146, 418, 503
dadR		Insensitivity of *dadA* to catabolite repression		36	412, 418
dam		DNA adenine methylase	*dam*	NM	362, 394, 418
dapA	Diaminopimelate	Dihydropicolinate synthase (EC 4.2.1.52)	*dapA*	50	410, 412, 418
dapB	Diaminopimelate	Dihydropicolinate reductase	*dapB*	0	410, 412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
dapC	Diaminopimelate	Tetrahydropicolinate succinylase	*dapC*	5	410, 412, 418
dapD	Diaminopimelate	Succinyl-diaminopimelate aminotransferase	*dapD*	5	410, 412, 418
dapF	Diaminopimelate	Diaminopimelate epimerase (EC 5.1.1.7)		5	410, 412, 418
dcd		dCTP deaminase (EC 3.5.4.13)	*dcd*	43	412, 418
dcm		DNA cytosine methylation	*dcm*	NM	418
dcp		Dipeptidyl carboxypeptidase	*dcp*	28	418
dctA		Transport of dicarboxylic acids	*dctA*	77	412, 418
deoA	Deoxyribose	*tpp*; thymidine phosphorylase (EC 2.4.2.4)	*deoA*	99	410, 412, 418
deoB	Deoxyribose	*drm*; phosphopentomutase (EC 2.7.5.6)	*deoB*	99	410, 412, 418
deoC	Deoxyribose	*dra*; phosphodeoxyriboaldolase (EC 4.1.2.4)	*deoC*	99	410, 412, 418
deoD	Deoxyribose	*pnu*, *pup*; purine nucleoside phosphorylase (EC 2.4.2.1)	*deoD*	99	410, 412, 418
deoK	Deoxyribose	Deoxyribokinase		20	410, 412, 418
deoP	Deoxyribose	Deoxyribose transport		20	410, 412, 418
deoR	Deoxyribose	Constitutive for enzymes of *deoA*, *deoB*, *deoC*, and *deoD*	*deoR*	19	410, 412, 418
dhb		2,3-Dihydroxybenzoic acid requirement		19	412, 418
dhuA	D-Histidine	Utilization; increased activity of histidine-binding protein J		46	8, 281, 283, 410, 412, 418, 428, 455
divA	Division	*wrkA*; septum initiation defect		87	410, 412, 418
divC	Division	*smoA*; septum initiation defect		3	93, 410, 412, 418
divD	Division	Round cell morphology		53	11, 418
dml	D-Malate	Utilization		80	410, 412, 418
dnaA	DNA	DNA initiation	*dnaA*	81	123, 313, 412, 418, 441
dnaB	DNA	DNA synthesis	*dnaB*	89	312–314, 418, 519
dnaC	DNA	DNA synthesis initiation and cell division uncoupling	*dnaC*	99	255, 312, 313, 398, 410, 412, 418
dnaE	DNA	DNA synthesis	*dnaE*	6	312, 313, 418
dnaG	DNA	DNA biosynthesis; DNA primase	*dnaG*	NM	125, 313, 398, 418
dnaJ	DNA	DNA biosynthesis	*dnaJ*	NM	313
dnaK	DNA	DNA biosynthesis	*dnaK*	NM	313
dnaL	DNA	DNA biosynthesis	*dnaL*	NM	313
dnaN	DNA	DNA biosynthesis; DNA polymerase III, beta subunit	*dnaN*	NM	123, 313
dnaQ	DNA	DNA biosynthesis	*dnaQ*	NM	312, 313
dnaX	DNA	DNA biosynthesis	*dnaX*	NM	313
dnaY	DNA	DNA biosynthesis	*dnaY*	NM	313
dnaZ	DNA	DNA biosynthesis	*dnaZ*	NM	123, 313, 398
dor		Deletion of *r*-determinants from plasmids		53	159, 210, 399, 418
dpp		Dipeptide permease		74	AG
dsd		D-Serine sensitivity; D-serine dehydratase (EC 4.2.1.14)	*dsd*	48	410, 412, 418
dum		dUMP synthesis			412, 418
earA		Regulates expression of *aniG*		86	3
eca		Enterobacterial common-antigen synthesis		NM	412, 418
endA		Endonuclease I	*endA*	63	418
eno	Enolase	Enolase (EC 4.2.1.11)	*eno*	60	418
ent	Enterochelin	*asc*, *enb*; enterochelin (dihydroxybenzoyl-serine trimer)	*ent*	13	410, 412
envA	Envelope	Cell division defect, chain formation	*envA*	3	412, 418
envB	Envelope	*bac*; spherical cells, drug sensitivity	*envB*	70	10, 11, 410, 412, 418
envD	Envelope	Autolysis; drug sensitivity; alterations in cell morphology		17	371, 412, 418
envZ	Outer membrane protein	*ompB*, *tppB*; positive regulation of tripeptide permease and outer membrane protein	*envZ*	74	152, 294, X
eutA	Ethanolamine utilization	Required for use of ethanolamine as sole carbon or nitrogen source		50	395
eutB	Ethanolamine utilization	Ethanolamine ammonia lyase, subunit I		50	395
eutC	Ethanolamine utilization	Ethanolamine ammonia lyase, subunit II		50	395
eutD	Ethanolamine utilization	CoA-dependent acetaldehyde dehydrogenase		50	395
eutE	Ethanolamine utilization	Required for use of ethanolamine as sole carbon source		50	395
eutR	Ethanolamine utilization	Positive regulatory gene for *eut* operon		50	395, V

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
fabB	Fatty acid biosynthesis	β-Ketoacyl acyl carrier protein synthetase I (EC 2.3.1.41)		47	98, 410, 412, 418
fdhA		Formate hydrogenlyase complex; formate dehydrogenase	*fdhA*	77	410, 412, 418
fdhB		Formate hydrogenlyase complex; formate dehydrogenase		18	412, 418
fdhC		Formate hydrogenase associated with both hydrogenase and nitrate reductase		80	418
fdhF		*fhl*; formate dehydrogenase	*fdhF*	93	21
fdnA		Formate dehydrogenase associated with nitrate reductase		85	367, 418, B
fdnB		Synthesis or activation of the cytochrome associated with formate dehydrogenase		85	367, 418
fdnC		Synthesis or activation of the cytochrome associated with formate dehydrogenase		85	367
fdp		Fructose-1,6-diphosphatase (EC 3.1.3.11)	*fdp*	99	410, 412, 418
fhlD		Formate dehydrogenase 2 activity		81	21, 412, 418
fim	Pili	*pil*; fimbriae (pili)	*fim*	14	90, 130, 382, 410, 412, 418
flgA	Flagella	*flaFI*; flagellar synthesis; function unknown	*flgA*	23	222, 302, 412, 418
flgB	Flagella	*flaFII*; flagellar synthesis; function unknown	*flgB*	23	222, 302, 412, 418
flgC	Flagella	*flaFIII*; flagellar synthesis; basal-body protein	*flgC*	23	222, 302, 412, 418
flgD	Flagella	*flaFIV*; flagellar synthesis; basal-body rod modification	*flgD*	23	222, 302, 412, 418
flgE	Flagella	*flaFV*; flagellar synthesis; hook protein	*flgE*	23	2, 207, 222, 302, 354, 412, 418
flgF	Flagella	*flaFVI*; flagellar synthesis; basal-body rod protein	*flgF*	23	2, 207, 222, 302, 412, 418
flgG	Flagella	*flaFVII*; flagellar synthesis; basal-body rod protein	*flgG*	23	2, 207, 222, 302, 412, 418
flgH	Flagella	*flaFVIII*; flagellar synthesis; basal-body L-ring protein	*flgH*	23	222, 302, 412, 418
flgI	Flagella	*flaFIX*; flagellar synthesis; basal-body P-ring protein	*flgI*	23	2, 204, 222, 302, 412, 418
flgJ	Flagella	*flaFX*; flagellar synthesis; function unknown	*flgJ*	23	222, 302, 412, 418
flgK	Flagella	*flaW*; flagellar synthesis; hook-associated protein 1	*flgK*	23	198, 200–202, 205, 206, 222, 247, 302, 525
flgL	Flagella	*flaU*; flagellar synthesis; hook-associated protein 3	*flgL*	23	198, 200–202, 205, 206, 222, 247, 302, 418, 525
flhA	Flagella	*flaC*; flagellar synthesis; function unknown	*flhA*	40	222, 302, 410, 412, 418
flhB	Flagella	*flaM*; flagellar synthesis; function unknown	*flhB*	40	222, 268, 302, 410, 412, 418, 460
flhC	Flagella	*flaE*; flagellar synthesis; regulation of gene expression	*flhC*	40	222, 268, 302, 410, 412, 418
flhD	Flagella	*flaK*; flagellar synthesis; regulation of gene expression (flagellum-specific sigma factor?)	*flhD*	40	222, 268, 302, 410, 412, 418
fliA	Flagella	*flaL*; flagellar synthesis; regulation of late gene expression	*fliA*	40	222, 247, 302, 410, 412, 418
fliB	Flagella	*nml*; flagellar synthesis; N-methylation of lysine residues in flagellin		40	222, 302, 410, 412, 418
fliC	Flagella	*H1*; flagellar synthesis; phase 1 flagellin (filament structural protein)	*fliC*	40	143, 199, 205, 222, 243, 247, 302, 410, 412, 418, 507, 508
fliD	Flagella	*flaV*; flagellar synthesis; hook-associated protein 2	*fliD*	40	198, 200–202, 205, 206, 222, 246, 247, 302, 525
fliE	Flagella	*flaAI*; flagellar synthesis; function unknown	*fliE*	40	222, 302, 410, 412, 418
fliF	Flagella	*flaAII.1*; flagellar synthesis; basal-body M-ring protein	*fliF*	40	2, 105, 222, 302, 418, 523
fliG	Flagella	*flaAII.2*, *motC*, *cheV*; flagellar synthesis; motor switching and energizing	*fliG*	40	105, 222, 302, 418, 522, 523
fliH	Flagella	*flaAII.3*; flagellar synthesis; function unknown	*fliH*	40	105, 203, 222, 302, 418, 523
fliI	Flagella	*flaAIII*; flagellar synthesis; function unknown	*fliI*	40	203, 222, 302, 410, 412, 418, 523
fliJ	Flagella	*flaS*; flagellar synthesis; function unknown	*fliJ*	40	203, 222, 302, 412, 418
fliK	Flagella	*flaR*; flagellar synthesis; hook length control	*fliK*	40	203, 222, 302, 412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
fliL	Flagella	*flaQI*; flagellar synthesis; function unknown	*fliL*	40	203, 222, 302, 523
fliM	Flagella	*flaQII*, *cheC*, *cheU*; flagellar synthesis; motor switching and energizing	*fliM*	40	203, 222, 302
fliN	Flagella	*flaN*; flagellar synthesis; motor switching and energizing	*fliN*	40	222, 302, 412, 418, 522, 523
fliO	Flagella	*flaP*; flagellar synthesis; function unknown	*fliO*	40	222, 302, 410, 412, 418
fliP	Flagella	*flaB*; flagellar synthesis; function unknown	*fliP*	40	222, 302, 410, 412, 418
fliQ	Flagella	*flaD*; flagellar synthesis; function unknown	*fliQ*	40	222, 302, 410, 412, 418, 522
fliR	Flagella	*flaX*; flagellar synthesis; function unknown	*fliR*	40	222, 302
fljA	Flagella	*rh1*; flagellar synthesis; repressor of phase 1 flagellin gene (*fliC*)	None	56	143, 222, 302, 410, 412, 418, 524
fljB	Flagella	*H2*; flagellar synthesis; phase 2 flagellin (filament structural protein)	None	56	199, 222, 223, 302, 410, 412, 418, 478, 524
flrB	Fluoroleucine resistance	Leucine or isoleucine regulation or both		14	410, 412, 418
fol	Folate	Trimethoprim resistance; tetrahydrofolate dehydrogenase (folate reductase)	*folA*	2	410, 412, 418
fpk	Fructose	Fructose phosphate kinase	*fpk*	45	148
frd	Fumarate reductase	Fumurate reductase (EC 1.3.99.1)	*frd*	NM	32
fruB	Fructose	Fructose phosphotransferase enzyme IIIA		45	148
fruF	Fructose	Fructose phosphotransferase pseudo-HPr		45	148
fruR	Fructose	Regulation of the fructose regulon, regulation of gluconeogenesis; may be the same as *ppsB*	*fruR*	3	82, 148
fuc	Fucose	L-Fucose utilization	*fuc*	61	412, 418
fur	Ferrichrome	Ferrichrome uptake, regulation of iron uptake; constitutive synthesis of iron-enterochelin		NM	418
galC	Galactose	Constitutive synthesis of specific galactose permease		18	412, 418
galE	Galactose	UDP glucose 4-epimerase (EC 5.1.3.2)	*galE*	18	224, 301, 348, 349, 410, 412, 418, P
galF	Galactose	Modifier of UDP-glucose pyrophosphorylase		42	410, 412, 418
galK	Galactose	Galactokinase (EC 2.7.1.6)	*galK*	18	410, 412, 418, P
galP	Galactose	Specific galactose permease	*galP*	NM	412, 418, P
galR	Galactose	Regulation	*galR*	61	410, 412, 418, P
galT	Galactose	Galactose-1-phosphate uridylyltransferase (EC 2.7.7.10)	*galT*	18	418
galU	Galactose	Glucose-1-phosphate uridylyltransferase (EC 2.7.7.9)	*galU*	34	192, 410, 412, 418
garA	Gamma resistant	Resistant to γ and UV radiation; large cells; high RNA and protein content (may be equivalent to *rodA*)		0	418
garB	Gamma resistant	Resistant to γ and UV radiation; large cells; high RNA and protein content		0	418
gcv	Glycine cleavage	Defective in the glycine cleavage enzyme system	*gcv*	62	AA
gdh	Glutamate	Glutamate dehydrogenase (EC 1.4.1.4)	*gdh*	27	193, 327, 418
gleR		Glycyl-leucyl-resistant regulatory gene for transport of branched-chain amino acids		7	418
glgA	Glycogen	Starch (bacterial glycogen) synthase (EC 2.4.1.21)	*glgA*	74	290, 291, 412, 418
glgC	Glycogen	Glucose-1-phosphate adenylyltransferase (EC 2.7.7.27)	*glgC*	74	290, 291, 412, 418
glnA	Glutamine	Glutamine synthetase (EC 6.3.1.2)	*glnA*	85	8, 173, 231, 258, 259, 267, 315, 412, 418
glnD	Glutamine	PIIA uridyl transferase	*glnD*	5	412, 418
glnE	Glutamine	Covalent modification of glutamine synthetase; glutamine synthetase adenylyl transferase (EC 2.7.2.42)		NM	267, 418
glnH	Glutamine	Periplasmic glutamine-binding protein		NM	418
glnP	Glutamine	Glutamine transport (high-affinity system)	*glnP*	20	412, 418
glnR	Glutamine	Regulation of enzymes for glutamine metabolism		85	418
glpA	Glycerol phosphate	Glycerol-3-phosphate dehydrogenase (anaerobic) (EC 1.1.99.5)	*glpA*	45	410, 412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
glpD	Glycerol phosphate	Glycerol-3-phosphate dehydrogenase (NAD^+) (EC 1.1.1.8)	*glpD*	74	410, 412, 418
glpK	Glycerol phosphate	Glycerol kinase (EC 2.7.1.30)	*glpK*	87	351, 410, 412, 418
glpQ	Glycerol phosphate	Glycerol-3-phosphate diesterase	*glpQ*	45	182
glpR	Glycerol phosphate	Regulatory gene for *glpD*, *glpK*, and *glpT*		74	412, 418
glpT	Glycerol phosphate	*sn*-Glycerol-3-phosphate transport	*glpT*	45	183, 410, 412, 418
gltA	Glutamate	Requirement	*gltA*	16	410, 412, 418
gltB	Glutamate	Glutamate synthetase (EC 2.6.1.53)	*gltB*	69	303, 418
gltC	Glutamate	Growth on glutamate as sole source of carbon		80	412, 418
gltD	Glutamate	Glutamate synthase, small subunit		69	303
gltF	Glutamate	Glutamate-specific transport system		100	5
gltH	Glutamate	Requirement	*gltH*	25	418
gltS	Glutamate	Glutamate permease	*gltS*	80	5
glyA	Glycine	Serine hydroxymethyltransferase (EC 2.1.2.1)	*glyA*	53	410, 412, 418, 488
glyS	Glycine	Glycyl-tRNA synthetase (EC 6.1.1.14)	*glyS*	78	412, 418
gnd		Phosphogluconate dehydrogenase (EC 1.1.1.43)	*gnd*	42	18, 53, 410, 412, 418
gpd		Glucosamine-6-phosphate deaminase		NM	412, 418
gpsA		*sn*-Glycerol-3-phosphate dehydrogenase [$NAD(P)^+$] (EC 1.1.1.94)		NM	412, 418
gpt		*gxu*; guanine-hypoxanthine phosphoribosyltransferase (EC 2.4.2.8)	*gpt*	6	355, 393, 412, 418
gsk		Guanosine kinase	*gsk*	13	412, 418
guaA	Guanine	GMP synthetase (EC 6.3.4.1)	*guaA*	52	131, 410, 412, 418
guaB	Guanine	IMP dehydrogenase (EC 1.1.1.205)	*guaB*	52	131, 410, 412, 418
guaC	Guanine	GMP reductase (EC 1.6.6.8)	*guaC*	3	412, 418
guaP	Guanine	Guanine uptake	*guaC*	3	410, 412, 418
gyrA	Gyrase	*hisW*, *nalA*; resistance or sensitivity to nalidixic acid; DNA gyrase	*gyrA*	46	241, 313, 387, 412, 418, 526
gyrB	Gyrase	*hisU*, DNA gyrase	*gyrB*	81	241
hemA	Heme	5-Aminolevulinate synthase (EC 2.3.1.37)	*hemA*	34	410, 412, 418
hemB	Heme	Heme deficient	*hemB*	8	410, 412, 418
hemC	Heme	Heme deficient; urogen I synthase	*hemC*	83	412, 418
hemD	Heme	Heme deficient; uroporphyrinogen III cosynthase	*hemD*	83	412, 418
hemE	Heme	Accumulation of uroporphyrin III	*hemE*	88	412, 418
hemG	Heme	Defective in heme synthesis	*hemG*	84	J
hemH	Heme	Defective in heme synthesis	*hemH*	11	J
hemL	Heme	*popC*; defective in synthesis of aminolevulinate or heme	*popC*	5	K
hin	H inversion	*vh2*; flagellar synthesis; regulation of flagellin gene expression by site-specific inversion of DNA	None	56	57, 58, 222, 302, 418, 432, 478, 524
hisA	Histidine	*N*-(5′-phospho-L-ribosylformimino)-5-amino-1-(5′-phosphoribosyl)-4-imidazolecarboxamide isomerase (EC 5.3.1.16)	*hisA*	42	71, 220, 410, 412, 418
hisB	Histidine	Imidazoleglycerol-phosphate dehydratase (EC 4.2.1.19) and histidinol phosphatase (EC 3.1.3.15)		0	71, 410, 412, 418
hisC	Histidine	Histidinol-phosphate aminotransferase (EC 2.6.1.9)	*hisC*	42	390, 410, 412, 418
hisD	Histidine	Histidinol dehydrogenase (EC 1.1.1.23)	*hisD*	42	13, 59, 220, 287, 390, 410, 412, 418, 424
hisE	Histidine	Phosphoribosyl-ATP pyrophosphohydrolase	*hisE*	42	59, 71, 72, 81, 410, 412, 418
hisF	Histidine	Cyclase	*hisF*	42	71, 81, 410, 412, 418
hisG	Histidine	ATP phosphoribosyltransferase (EC 2.4.2.17)	*hisG*	42	7, 13, 20, 87, 88, 144, 391, 401, 410, 412, 418
hisH	Histidine	Amido transferase	*hisH*	42	71, 410, 412, 418
hisI	Histidine	Phosphoribosyl-AMP cyclohydrolase (EC 3.5.4.19) (may be bifunctional with *hisE*)	*hisI*	42	59, 71, 81, 410, 412, 418
hisJ	Histidine	Periplasmic histidine-binding protein J for high-affinity histidine transport system	*hisJ*	46	12, 62, 281, 283, 410, 412, 418, 535
hisM	Histidine	Histidine transport; location of protein not known		46	9, 345, 368, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
hisP	Histidine	High-affinity histidine transport; P protein in the inner membrane		46	9, 12, 154, 283, 410, 412, 418
hisQ	Histidine	Histidine transport; Q, a membrane protein		46	9, 12, 283. 418
hisR	Histidine	tRNA structural gene	*hisR*	83	41, 44, 72, 410, 412, 418
hisS	Histidine	Histidyl-tRNA synthetase (EC 6.1.1.21)	*hisS*	53	410, 412, 418
hisT	Histidine	Pseudouridine modification of tRNA	*hisT*	47	340, 410, 412, 418
hpt		Hypoxanthine phosphoribosyltransferase (not EC 2.4.2.8) (see *gpt*)	*hpt*	NM	355, 412, 418
hsdL	Host specificity	*hspLT*; restriction modification system		8	410, 412, 418
hsdSA	Host specificity	*hspS*; restriction modification system	*hsd*	98	262, 410, 412, 418
hsdSB	Host specificity	Restriction modification system	*hsd*	98	61, 262, 412, 418
hutC	Histidine utilization	Utilization; repressor		18	410, 412, 418
hutG	Histidine utilization	Formiminoglutamase (EC 3.5.3.8)		18	410, 412, 418
hutH	Histidine utilization	Histidine ammonia-lyase (EC 4.3.1.3)		18	410, 412, 418
hutI	Histidine utilization	Imidazolonepropionase (EC 3.5.2.7)		18	410, 412, 418
hutM	Histidine utilization	Utilization; promoter for *hutIGC*		18	410, 412, 418
hutP	Histidine utilization	Utilization; promoter for *hutUH*		18	410, 412, 418
hutQ	Histidine utilization	Utilization; promoter for *hutUH*		18	410, 412, 418
hutR	Histidine utilization	Utilization; catabolite insensitivity of *hutUH*		18	410, 412, 418
hutU	Histidine utilization	Utilization; urocanate hydratase (EC 4.2.1.49)		18	410, 412, 418
hyd		*aniA*, *fhlB*; hydrogenase	*hyd*	59	21, 412, 418, 420
hyp	Hydrophobic peptide auxotrophy	Hydrophobic polypeptide requirement		48	418
ilvA	Isoleucine-valine	*ile*; threonine dehydratase (EC 4.2.1.16)	*ilvA*	83	116, 177, 410, 412, 418, 479
ilvB	Isoleucine-valine	Acetolactate synthetase I, large subunit (valine sensitivity) (EC 4.1.3.18)	*ilvB*	80	101, 279, 280, 410, 412, 418, 512
ilvC	Isoleucine-valine	*ilvA*; 2-acetolactate mutase (EC 5.4.99.3)	*ilvC*	83	35, 410, 412, 418
ilvD	Isoleucine-valine	*ilvB*; dihydroxyacid dehydratase (EC 4.2.1.19)	*ilvD*	83	116, 177, 410, 412, 418
ilvE	Isoleucine-valine	*ilvC*; branched-chain aminotransferase (EC 2.6.1.42)	*ilvE*	83	116, 177, 410, 412, 418
ilvG	Isoleucine-valine	Acetolactate synthase II, large subunit (feedback inhibition insensitive)	*ilvG*	83	101, 116, 177, 278, 280, 412, 418, 421
ilvH	Isoleucine-valine	Acetolactate synthase II subunit (normally inactive)	*ilvH*	3	418, 451
ilvI	Isoleucine-valine	Acetolactate synthase II subunit (normally inactive)	*ilvI*	3	418, 451
ilvM	Isoleucine-valine	Acetolactate synthase II, small subunit (feedback inhibition insensitive)		83	101, 421
ilvN	Isoleucine-valine	Acetolactate synthase I, small subunit		80	101
ilvS	Isoleucine-valine	Isoleucyl-tRNA synthetase (EC 6.1.1.5)		1	410, 412, 418
ilvY	Isoleucine	Regulation of *ilvC*	*ilvY*	83	418
inlA	Inositol	Fermentation		92	35, 410, 412, 418
inlB	Inositol	Fermentation		54	410, 412, 418
inm		Sensitivity to mutagenesis by nitrosoguanidine		79	92
katG	Catalase	*cls*; HPI and HPII catalases (EC 1.11.1.6)	*katG*	88	333, 412, 418
kdsA		Ketodeoxyoctonate synthesis		39	70, 156, 157, 172, 174, 384, 388, 412, 418, 465
kdsB		CMP ketodeoxyoctonate synthetase		16	70, 155–157, 418
lamB	Lambda	Determines a protein resembling the lambda receptor	*lamB*	91	418
leuA	Leucine	α-Isopropylmalate synthase (EC 4.1.3.12)	*leuA*	2	73, 74, 149, 150, 178, 410, 412, 418, 433
leuB	Leucine	β-Isopropylmalate dehydrogenase	*leuB*	2	410, 412, 418, 433
leuC	Leucine	α-Isopropylmalate isomerase subunit	*leuC*	2	410, 412, 418
leuD	Leucine	α-Isopropylmalate isomerase subunit	*leuD*	2	142, 396, 410, 412, 418, 464
leuS	Leucine	Leucyl-tRNA synthetase (EC 6.1.1.4)	*leuS*	14	410, 412, 418
leuT	Leucine	Leucine transport		35	410, 412, 418
leuU	Leucine	*leuT*; tRNA structural gene for leucine	*leuT*	83	41, 44
lev		Levomycetin resistance		NM	412, 418
lig	Ligase	DNA ligase	*lig*	NM	313
lip	Lipoic acid	Requirement	*lip*	13	410, 412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
livA	Leucine, isoleucine, valine	High-affinity branched-chain amino acid transport	*liv*	76	310, 311, 418
livS	Leucine, isoleucine, valine	Regulatory gene; high-affinity branched-chain amino acid transport	*livR*	19	337
lkyA	Leaky	Leakage of periplasmic proteins		58	412, 418
lkyB	Leaky	Leakage of periplasmic proteins		58	412, 418
lkyC	Leaky	Leakage of periplasmic proteins		20	412, 418
lkyD	Leaky	Leakage of periplasmic proteins; morphology defect		20	80, 93, 300, 412, 418
lon	Long form	*capR*; filamentous growth; radiation sensitivity; polyamine metabolism; stabilization of abnormal proteins	*lon*	9	115, 412, 418, 504
lpd		Dihydrolipoamide dehydrogenase (EC 1.8.1.4)	*lpd*	3	412, 418
lpp	Lipoprotein	Murein lipoprotein structural gene		NM	418
lys	Lysine	Requirement	*lysA*	62	95, 410, 412, 418
malE	Maltose	*malB*; maltose uptake; periplasmic maltose-binding protein	*malE*	91	410, 412, 418, A
malF	Maltose	Maltose uptake; inner membrane protein	*malF*	91	A
malG	Maltose	Maltose uptake; inner membrane protein	*malG*	91	A
malK	Maltose	Maltose uptake; inner membrane protein	*malK*	91	A
malQ	Maltose	Amylomaltase (EC 1.2.1.25)	*malQ*	74	410, 412, 418
malT	Maltose	Regulation of maltose genes	*malT*	74	418
melA	Melibiose	α-Galactosidase (EC 3.2.1.22)	*mel*	93	418
melB	Melibiose	Permease		93	418
mem	Membrane	Sugar transport and membrane protein defective		78	412, 418
menA	Menaquinone	Menaquinone deficient; defective in trimethylamine oxide reduction; grows on vitamin K_1	*menA*	87	270, 271, 418
menB	Menaquinone	Biosynthesis; grows on vitamins K_1 and K_5		46	89, 270, 271
menC	Menaquinone	Biosynthesis	*menC*	46	271
menD	Menaquinone	Biosynthesis	*menD*	46	271
metA	Methionine	*metI*; homoserine transsuccinylase (EC 2.3.1.46)	*metA*	89	410, 412, 418
metB	Methionine	Cystathionine γ-synthase (EC 4.2.99.9)	*metB*	87	410, 412, 418, 487, 489, 490
metC	Methionine	Cystathionine γ-lyase (EC 4.4.1.1)	*metC*	64	410, 412, 418, AA
metE	Methionine	Tetrahydropteroyltriglutamate methyltransferase (EC 2.1.1.14)	*metE*	84	372, 410, 412, 418, 431, 494
metF	Methionine	5,10-Methylenetetrahydrofolate reductase (EC 1.1.99.15)	*metF*	87	410, 412, 418
metG	Methionine	Methionyl-tRNA synthetase	*metG*	44	410, 412, 418
metH	Methionine	Vitamin B_{12}-dependent homocysteine-N^5-methylenetetrahydrofolate transmethylase	*metH*	89	410, 412, 418, 491, 494
metJ	Methionine	Methionine analog resistant; protein for methionine pathway regulation	*metJ*	87	410, 412, 418, 489, 490, 492
metK	Methionine	Methionine analog resistant; *S*-adenosylmethionine synthetase	*metK*	63	410, 412, 418
metL	Methionine	Aspartokinase II-homoserine dehydrogenase II	*metL*	87	490
metP	Methionine	High-affinity methionine transport	*metD*	5	373, 410, 412, 418
metR	Methionine	*trans*-Acting protein for expression of *metE* and *metH*		84	372, 493, 494
mglA	Methyl galactosidase	Membrane-bound protein for transport	*mglA*	NM	336, F
mglB	Methyl galactoside	Galactose-binding protein	*mglB*	NM	336, 412, 418, F
mglC	Methyl galactosidase	Membrane-bound protein for transport	*mglC*	NM	336, F
mglD	Methyl galactosidase	Repressor for *mgl* operon		NM	F
mglE	Methyl galactosidase	Transport		NM	336, F
mgtA	Magnesium transport	Magnesium transport		98	AF
mgtB	Magnesium transport	Magnesium transport		81	AF
miaA		Deficient in the nucleotide ms^2io^6A adenosine, a modified base present in some tRNAs		96	46, 60, 126
min	Minicells	Cell division	*min*	NM	412, 418
motA	Motility	Nonmotile but flagellate	*mot*	40	268, 410, 412, 418
motA	Motility	Nonmotile but flagellate	*mot*	40	268, 410, 412, 418

Continued on following page

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
mta	*meso*-Tartaric acid	Utilization of and resistance to *meso*-tartaric acid		NM	418
mtlA	Mannitol	D-Mannitol phosphotransferase enzyme IIA	*mtlA*	78	161, 377, 405, 410, 412, 418
mtlD	Mannitol	Mannitol-1-phosphate dehydrogenase (EC 1.1.1.17)	*mtlD*	78	412, 418
musA	Mu sensitivity	Adsorption of phage Mu		42	335, 418
musB	Mu sensitivity	Adsorption of phage Mu		42	418
mutB	Mutator	Increased frequency of mutation with alkylating agents		64	418
mutG	Mutator	Increased frequency of mutation in host chromosome, not in P22		NM	412, 418
mutH	Mutator	Mutator	*mutH*	61	363, 412, 418
mutL	Mutator	Increased frequency of mutation	*mutL*	96	362, 363, 412, 418
mutS	Mutator	Increased frequency of mutation with alkylating agents	*mutS*	59	168, 362, 363, 418
mviA	Mouse virulence	Affects the growth rate of cells in mice		34	G
mviC	Mouse virulence	Affects the growth rate of cells in mice		16	G
nadA	Nicotinamide	*nicA*; requirement; quinolinic acid synthetase	*nadA*	17	19, 197, 410, 412, 418, 448, 482, 533
nadB	Nicotinamide	*nic*; L-aspartate oxidase	*nadB*	55	19, 94, 197, 412, 418, 533
nadC	Nicotinamide	Quinolinic acid PRPP phosphoribosyl transferase	*nadC*	3	19, 197, 412, 418
nadD	Nicotinamide	Requirement; NAMN adenylyl transferase		14	19, 214, 215, 418
nadE	Nicotinamide	Essential biosynthetic gene, unsupplementable; NAD synthetase		25	217, Q
nadI	Nicotinamide	Derepression of *nadA* and *nadB*		99	94, 533, AF
nadR	Nicotinamide	Controls expression of several genes for NAD synthesis		99	137, 197, O
nag	*N*-Acetylglucosamine	Nonutilization	*nag*	15	412, 418
nalB	Nalidixic acid	Resistance or sensitivity	*nalB*	57	412, 418
nap		Deficiency for nonspecific acid phosphatase I		NM	418
ndk		Nucleosidediphosphate kinase (EC 2.7.4.6)		53	412, 418
newD		Substitute gene for *leuD*		7	393, 412, 418, 464
nit	Nitrogen	Nitrogen metabolism		28	412, 418
nol	Norleucine	Norleucine resistance; possible defect in valine uptake or regulation		60	412, 418
nrdA		Ribonucleoside diphosphate reductase (EC 1.17.4.1), subunit B1	*nrdA*	NM	313
nsiA		Nicotinamide starvation inducible; NAD metabolism regulation		77	418
ntrA	Nitrogen regulation	*glnF*; repressor-activator for *glnA* expression and for other nitrogen-controlled genes	*glnF*	69	259, 315, 412, 418
ntrB	Nitrogen regulation	*glnR*; regulation of *glnA* expression and other nitrogen-controlled genes	*glnR*	85	8, 258, 259, 267, 315, 346, 347, 418
ntrC	Nitrogen regulation	*glnR*; regulation of *glnA* expression and other nitrogen-controlled genes	*glnR*	85	8, 258, 259, 315, 347, 361, 418
nuvA		Uridine thiolation factor A activity	*nuvA*	NM	261, R
oafA	O-antigen factor	*O-5*, *ofi*; lipopolysaccharide O-factor 5 (acetyl group)		43	410, 412, 418
oafC	O-antigen factor	Determines factor 1 in lipopolysaccharide of group E *Salmonella* spp.		13	410, 412, 418
oafR	O-antigen factor	Synthesis of lipopolysaccharide O antigen 12^2		12	410, 412, 418
ompA		Outer membrane protein 33K (II* of *E. coli*)	*ompA*	20	140, 412, 418
ompC		Outer membrane protein 36K (Ib of *E. coli*)	*opmC*	45	412, 418, 532
ompD		Outer membrane protein 34K		32	412, 418
ompF		Outer membrane protein 35K (Ia)	*ompF*	NM	418
ompR	Outer membrane protein	*ompB*, *tppA*; positive regulation of tripeptide permease and of outer membrane protein	*ompR*	74	152, 153, 186, 228, 293, 294, 412, 418
oppA	Oligopeptide permease	Oligopeptide-binding protein	*opp*	34	160, 186, 187, 190–192, 418
oppB	Oligopeptide permease	Oligopeptide transport system	*opp*	34	160, 186, 190, 192, 418
oppC	Oligopeptide permease	Oligopeptide transport system	*opp*	34	160, 186, 190, 192, 418
oppD	Oligopeptide permease	Oligopeptide transport system	*opp*	34	160, 186, 190, 192, 418
oppF	Oligopeptide permease	Oligopeptide transport system	*opp*	34	190, 192

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
optA	Oligopeptidase	Oligopeptidase [hydrolyzes *N*-acetyl-(L-alanyl)$_4$]		76	418
oriC	Origin	*poh*; origin of replication of chromosome	*oriC*	81	418
oxdA	Oxygen dependent	Gene activity controlled by *oxrA*		64	467
oxdB	Oxygen dependent	Gene activity controlled by *oxrA*		93	467
oxiA	Oxygen inducible	Induced by anaerobiosis		10	4, 230
oxiB	Oxygen inducible	Induced by anaerobiosis		22	4
oxiC	Oxygen inducible	Induced by anaerobiosis		30	4
oxiE	Oxygen inducible	Induced by anaerobiosis		88	4
oxrA	Oxygen regulation	Prevent oxygen regulation of *pepT*	*nirR*	30	467
oxrF	Oxygen regulation	Regulates expression of *aniH*		NM	3
oxrG	Oxygen regulation	Regulates expression of *aniC, I*		88	3
oxyR	Oxidative stress resistant	Positive regulator		88	85, 286, 333, 463
pabA	P-amino benzoate	Requirement; *p*-aminobenzoate synthase	*pabA*	NM	248
panB	Pantothenic acid	Ketopantohydroxymethyl transferase (EC 4.1.2.12)	*panB*	5	410, 412, 418
panC	Pantothenic acid	Pantothenate synthetase (EC 6.3.2.1)	*panC*	5	410, 412, 418
panD	Pantothenic acid	Ketopantoic acid reductase		5	412, 418, 513
panE	Pantothenic acid	Ketopantoic acid reductase		NM	418
panT	Pantothenic acid	Pantothenate transport		NM	412, 418
pasA		6-Aminonicotinic acid sensitive		92	136, 410, 412, 418
pasB		6-Aminonicotinic acid sensitive		66	136, 418
pasC		6-Aminonicotinic acid sensitive		20	136, 418
pasD		6-Aminonicotinic acid sensitive		18	136
pasE		6-Aminonicotinic acid sensitive		55	136
pck		Phosphoenolpyruvate carboxykinase (ATP) (EC 4.1.1.49)	*pck*	13	410, 412, 418
pclA	Permissive for *cly*	Permissive for lytic growth of P22 *cly*		NM	418
pclB	Permissive for *cly*	Permissive for lytic growth of P22 *cly*		NM	418
pclC	Permissive for *cly*	Permissive for lytic growth of P22 *cly*		NM	418
pde	Phosphodiesterase	2′,3′-Cyclic nucleotide 2′-phosphodiesterase		96	418
pdxB	Pyridoxine	Requirement	*pdxB*	47	410, 412, 418
pepA	Peptidase	Peptidase A (similar to aminopeptidase A of *E. coli*)		97	153, 386, 412, 418
pepB	Peptidase	Peptidase B (aminopeptidase)		53	386, 418
pepD	Peptidase	*ptdD*; Peptidase D (a dipeptidase, carnosinase)	*pepD*	6	386, 410, 412, 418
pepE	Peptidase	Peptidase E (splits Asp-X peptide bonds)		90	75, 418
pepM	Peptidase	Peptidase M; aminopeptidase that removes N-terminal methionine from proteins		3	326
pepN	Peptidase	*ptdN*; peptidase N (an aminopeptidase, naphthylamidase)	*pepN*	20	55, 386, 410, 412, 418
pepP	Peptidase	*ptdP*; peptidase P (splits X-Pro peptide bonds)		63	410, 412, 418
pepQ	Peptidase	Peptidase Q (splits X-Pro peptide bonds)		84	412, 418
pepT	Peptidase	Peptidase T (a tripeptidase)		25	418, 466, 468
pfkA		6-Phosphofructokinase (EC 2.7.1.11)	*pfkA*	85	412, 418
pfl		Pyruvate formate lyase	*pfl*	19	196, 412, 418
pgi	Phosphoglucose isomerase	*oxrC, pasA*; regulation of fermentative or biosynthetic enzymes; glucosephosphate isomerase (EC 5.3.1.9)	*pgi*	92	229, 412, 418
pgtA	Phosphoglycerate	Positive activator of phosphoglycerate transport		49	236, 407, 527, 530
pgtB	Phosphoglycerate	Protein for signal transmission for phosphoglycerate transport		49	236, 407, 527, 530
pgtC	Phosphoglycerate	Protein for signal transmission for phosphoglycerate transport		49	236, 407, 527, 530
pgtP	Phosphoglycerate	Transporter for phosphoglycerate transport		49	158, 236, 407, 527, 530
pheA	Phenylalanine	Chorismate mutase (EC 5.4.99.5)	*pheA*	55	410, 412, 418
pheR	Phenylalanine	Regulator gene for *pheA*		64	410, 412, 418
phoN	Phosphatase	Nonspecific acid phosphatase		25	412, 418
phoP	Phosphatase	Nonspecific acid phosphatase I		95	412, 418
phoS	Phosphatase	Periplasmic phosphate-binding protein		NM	23, 412, 418
phs		Hydrogen sulfide production		41	89, 412, 418
pig	Pigment	Brownish colonies		54	410, 412, 418
ply	Phage lysogeny	*pox*; control of P22 lysogeny		NM	418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
pmi	Mannose	Mannose-6-phosphate isomerase (EC 5.3.1.8)	*manA*	30	164, 410, 412, 418
pmrA		Polymyxin resistance		94	412, 418, Z
pncA	Pyridine nucleotide cycle	Nicotinamide deamidase (EC 3.5.1.19)	*pncA*	27	193, 214, 412, 418
pncB	Pyridine nucleotide cycle	Nicotinic acid phosphoribosyltransferase (EC 2.4.2.11)		20	254, 412, 418
pncH	Pyridine nucleotide cycle	Nicotinamide used as sole nitrogen source		27	193
pncX		6-Aminonicotinamide resistant		27	193, 214
pnuA	Pyridine nucleotide uptake	*pncC*; NMN uptake deficient		99	137, 418, 449, AF
pnuB	Pyridine nucleotide uptake	Growth on lower than normal levels of NMN		99	418, 449
pnuC	Pyridine nucleotide uptake	NMN uptake deficient		17	418, 449, 482
pnuD	Pyridine nucleotide uptake	Reduced NMN uptake in *nad pncA*$^+$ strain		60	449
pnuE	Pyridine nucleotide cycle	Failure to use exogenous NAD; periplasmic NAD pyrophosphorylase		86	366
polA	Polymerase	DNA nucleotidyltransferase (EC 2.7.7.7)		0	167, 410, 412, 418
poxA	Pyruvate oxidase	Hypersensitivity to antimicrobial agents; lower levels of pyruvate oxidase and acetolactate synthase deficiency in α-ketobutyrate metabolism	*poxA*	95	499
ppc		Phosphoenolpyruvate carboxylase (EC 4.1.1.31)	*ppc*	87	404, 410, 412, 418
ppsA		Phosphoenolpyruvate synthase	*ppsA*	37	148
ppsB		Deficiency in phosphoenolpyruvate synthase; may be identical to *fruR*	*ppsA*	3	412, 418
praA		Phage P221 receptor function		94	412, 418
praB		Phase P221 receptor function		60	412, 418
prbA		Phage ES18 receptor function		92	412, 418
prbB		Phase ES18 receptor function		30	412, 418
prdB		Phage PH51 receptor function		31	412, 418
prh		Phage HK009 receptor function		94	412, 418
prk		Phage HK068 receptor function		30	412, 418
proA	Proline	Glutamate to glutamic-γ-semialdehyde	*proA*	7	304, 393, 410, 412, 418
proB	Proline	Glutamate to glutamic-γ-semialdehyde	*proB*	7	304, 410, 412, 418
proC	Proline	Pyrroline-5-carboxylate reductase (EC 1.5.1.2)	*proC*	8	51, 410, 412, 418
proP	Proline	Proline permease II; betaine and proline; low affinity	*proP*	93	67, 68, 120, 242, 412, 418
proT	Proline	tRNA structural gene for proline	*proT*	83	41, 44
proU	Proline	Proline/glycine betaine permease (high-affinity betaine uptake)	*proU*	57	12, 66, 118, 120, 185, 242, 418, 473
proV	Proline	Periplasmic betaine-binding protein	*proV*	57	188
prp	Propionate	Propionate metabolism	*prp*	97	M
prsA		Phosphoribosylpyrophosphate synthetase	*prs*	35	48, 237, 418
prsB		Phosphoribosylpyrophosphate synthetase		44	418
psiA		Phosphate starvation inducible		74	138
psiB		Phosphate starvation inducible		88	138
psiC		Phosphate starvation inducible		10	138
psiD		Phosphate starvation inducible		93	138
psiR		Regulates *psiC* activity		82	138
psuA		Suppressor of polarity		NM	412, 418
pta	Phosphotransacetylase	Acetyl-CoA:orthophosphate acetyltransferase (EC 2.3.1.8)		46	272, 418, 498
ptsF	Phosphotransferase system	*fruA*; fructose phosphotransferase enzyme IIA	*ptsF*	NM	148, 377, 405, 412, 418
ptsG	Phosphotransferase system	*glu*, *gpt*; glucose phosphotransferase enzyme IIB′-factor III (*crr*) system (methyl-β-D-glucoside)	*ptsG*	25	47, 161, 377, 405, 412, 418, 462
ptsH	Phosphotransferase system	*carB*; phosphohistidine protein-hexose phosphotransferase (EC 2.7.1.69	*ptsH*	49	25, 65, 162, 189, 330, 332, 377, 378, 405, 410, 412, 418, 505, 510
ptsI	Phosphotransferase system	*carA*; enzyme I of the phosphotransferase system	*ptsI*	49	65, 161, 265, 330, 332, 377, 405, 410, 412, 418, 509, 511

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
ptsJ	Phosphotransferase system	Enzyme I* of the phosphotransferase system, not expressed in wild type	*ptsJ*	49	83
ptsM	Phosphotransferase system	*manA*; mannose-glucose phosphotransferase enzyme IIA (2-deoxyglucose)	*ptsM*	NM	377, 405, 412, 418, 462
purA	Purine	Adenylosuccinate synthetase (EC 6.3.4.4)	*purA*	96	410, 412, 418
purB	Purine	Adenylosuccinate lyase (EC 4.3.2.2)	*purB*	25	410, 412, 418
purC	Purine	Phosphoribosylaminoimidazole-succinocarboxamide synthetase (EC 6.3.2.6)	*purC*	51	410, 412, 418
purD	Purine	Phosphoribosylglycinamide synthetase (EC 6.3.1.13)	*purD*	89	121, 410, 412, 418
purE	Purine	Phosphoribosylaminoimidazole carboxylase (EC 4.1.1.21)	*purE*	11	355, 410, 412, 418
purF	Purine	Amidophosphoribosyltransferase (EC 2.4.2.14)	*purF*	47	114, 410, 412, 418
purG	Purine	Phosphoribosylglycinamidine synthetase (EC 6.3.5.3)	*purL*	54	410, 412, 418
purH	Purine	Phosphoribosylaminoimidazolecarboxamide formyltransferase (EC 2.1.2.3)	*purH*	89	121, 410, 412, 418
purI	Purine	Phosphoribosylaminoimidazole synthetase (EC 6.3.3.1)	*purM*	51	410, 412, 418
purJ	Purine	IMP cyclohydrolase (EC 3.5.4.10)		89	410, 412, 418
purN	Purine	Cryptic *purF* analog; synthesis of phosphoribosylamine		4	I, AD
purR	Purine	Constitutive high expression of *pur* genes		30	I
putA	Proline	*putB*; utilization; bifunctional enzyme; proline oxidase and pyrroline-5-carboxylate dehydrogenase	*putA*	22	12, 100, 171, 307, 410, 412, 418, T
putP	Proline	Utilization; major L-proline permease		22	12, 68, 108, 120, 307, 410, 412, 418, T
pyrA	Pyrimidine	*argD*, *ars*; arginine + uracil requirement; carbamoyl-phosphate synthase (glutamine) (EC 6.3.5.5)	*car*	1	342, 410, 412, 418
pyrB	Pyrimidine	Aspartate carbamoyltransferase (EC 2.1.3.2)	*pyrB*	98	133, 233, 322, 323, 410, 412, 418
pyrC	Pyrimidine	Dihydro-orotase (EC 3.5.2.3)	*pyrC*	23	250, 343, 410, 412, 418, 486
pyrD	Pyrimidine	Dihydro-orotate oxidase (EC 1.3.3.1)	*pyrD*	20	250, 410, 412, 418, 486
pyrE	Pyrimidine	Orotate phosphoribosyltransferase (EC 2.4.2.10)	*pyrE*	79	233, 250, 344, 410, 412, 418
pyrF	Pyrimidine	Orotidine-5′-phosphate decarboxylase (EC 4.1.1.23)	*pyrF*	33	410, 412, 418, 481
pyrG	Pyrimidine	CTP synthetase	*pyrG*	60	410, 412, 418
pyrH	Pyrimidine	UMP kinase	*pyrH*	5	232, 249, 342, 410, 412, 418
pyrI	Pyrimidine	Regulatory polypeptide for aspartate transcarbamylase (EC 2.1.3.2), regulatory subunit	*pyrI*	98	133, 323
rbsP	Ribose	Ribose-binding protein	*rbsP*	82	412, 418
recA		Recombination deficient; degrades DNA	*recA*	58	17, 117, 159, 165, 216, 357, 359, 410, 412, 418, 425
recB		Recombination deficient; exonuclease V	*recB*	61	117, 216, 412, 418, 443
recC		Recombination deficient; exonuclease V	*recC*	61	117, 216, 412, 418, 443
relA	RNA relaxed	*RC*; regulation of RNA synthesis	*relA*	61	224, 261, 412, 418
rfaB	Rough	UDP-D-galactose:lipopolysaccharide α-1,6-D-galactosyl transferase		79	245, 418
rfaC	Rough	Lipopolysaccharide core defect; proximal heptose deficient	*rfa*	79	49, 54, 356, 412, 418
rfaD	Rough	D-Glycero-D-manno-heptose epimerase	*rfaD*	79	412, 418, 518
rfaE	Rough	Lipopolysaccharide core defect; proximal heptose deficient		76	49, 339, 383, 410, 412, 418, 480
rfaF	Rough	Lipopolysaccharide core defect; distal heptose deficient		79	410, 412, 418
rfaG	Rough	Lipopolysaccharide core defect; glucose I transferase		79	50, 245, 410, 412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
rfaH	Rough	Deficient in lipopolysaccharide core synthesis and in F-factor expression; transcription control factor	*sfrB*	84	99, 410, 412, 418
rfaI	Rough	Lipopolysaccharide core defect; galactose I deficient		78	244, 245, 418
rfaJ	Rough	Lipopolysaccharide core defect; glucose II transferase		79	244, 245, 410, 412, 418
rfaK	Rough	Lipopolysaccharide core defect; *N*-acetylglucosamine transferase deficient		79	410, 412, 418
rfaL	Rough	Lipopolysaccharide core defect; *O*-translocase		79	410, 412, 418
rfaP	Rough	Lipopolysaccharide core defect; phosphorylation of heptose		77	410, 412, 418
rfbA	Rough	TDP-glucose pyrophosphorylase		42	52, 53, 292, 389, 410, 412, 418, 501
rfbB	Rough	TDP-glucose oxidoreductase		42	52, 53, 410, 412, 418
rfbD	Rough	TDP-rhamnose synthetase		42	52, 53, 410, 412, 418
rfbE	Rough	Lipopolysaccharide side chain defect; CDP paratose synthesis in *S. typhi*		42	53, D
rfbF	Rough	Glucose-1-phosphate cytidylyltransferase (EC 2.7.7.33)		42	52, 53, 410, 412, 418
rfbG	Rough	CDP-glucose oxidoreductase		42	52, 53, 410, 412, 418
rfbH	Rough	CDP-abequose synthetase		42	52, 53, 410, 412, 418
rfbJ	Rough	CDP-4-keto-3,6-D-glucose dehydrogenase		42	53, D
rfbK	Rough	Lipopolysaccharide side chain defect	*rfb*	42	52, 53, 410, 412, 418
rfbL	Rough	Phosphomannomutase B		42	52, 53, 410, 412, 418
rfbM	Rough	Mannose-1-phosphate guanylyltransferase (EC 2.7.7.22)		42	52, 53, 410, 412, 418
rfbN	Rough	Galactose-diphosphoglycosyl carrier lipid synthetase		42	52, 53, 412, 418
rfbT	Rough	*O*-Translocase		42	52, 410, 412, 418
rfc	Rough	*rouC*; O-repeat unit not polymerized		31	369, 410, 412, 418
rfe	Rough	Defect in synthesis of enterobacterial common antigen, the T1 antigen, and O-side chains of *Salmonella* groups L and C1	*rfe*	83	292, 410, 412, 418
rff	Rough	Block in synthesis of enterobacterial common antigen	*rff*	84	292, 319, 412, 418
rft	Rough	"Transient" T1 forms		15	410, 412, 418
rfu	Rough	"Transient" T1 forms		NM	418
rhaA		L-Rhamnose isomerase (EC 5.3.1.14)	*rhaA*	86	6, 410, 412, 418
rhaB		L-Rhamnulokinase (EC 2.7.1.5)	*rhaB*	86	6, 410, 412, 418
rhaC		Regulation	*rhaC*	86	6, 410, 412, 418
rhaD		L-Rhamnulose-1-phosphate aldolase (EC 4.1.2.19)	*rhaD*	86	6, 410, 412, 418
rhaT		L-Rhamnose transport		86	6, 410, 412, 418
rho		*psu*; polarity suppressor; transcription terminator factor Rho	*rho*	83	418
rna		*rnsA*; RNase I	*rna*	17	412, 418
rnc		RNase III	*rnc*	NM	412, 418
rnpB	RNase	RNase P, RNA component	*rnpB*	NM	15
rodA	Rod	Round cell morphology; mecillinam resistant	*rodA*	13	11, 418
rplJ	Ribosomal protein, large	Ribsomal protein subunit	*rplJ*	88	475, 483
rplL	Ribosomal protein, large	Ribosomal protein subunit	*rplL*	88	475, 483
rpoB	RNA polymerase	*rif*; RNA polymerase, β subunit (EC 2.7.7.6)	*rpoB*	89	233, 342, 410, 412, 418, 474, 475, 483, 484
rpoC	RNA polymerase	RNA polymerase, β′ subunit (EC 2.7.7.6)	*rpoC*	89	233, 412, 418, 475, 483, 484
rpoD	RNA polymerase	RNA polymerase, σ subunit	*rpoD*	NM	125, 180, 418
rpsE	Ribosomal protein, small	*spcA*; 30S ribosomal subunit protein S5	*rpsE*	71	410, 412, 418
rpsL	Ribosomal protein, small	*strA*; 30S ribosomal subunit protein S12	*rpsL*	71	410, 412, 418
rpsU	Ribosomal protein, small	30S ribosomal subunit protein S21	*rpsU*	NM	125

Continued on following page

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
rrnA	rRNA	rRNA operon	*rrnA*	86	288, 289, 418
rrnB	rRNA	rRNA operon	*rrnB*	89	288, 289, 418, 446
rrnC	rRNA	rRNA operon	*rrnC*	82	288, 289, 418
rrnD	rRNA	rRNA operon	*rrnD*	73	288, 418
rrnE	rRNA	rRNA operon	*rrnE*	89	288, 289, 418
rrnG	rRNA	rRNA operon	*rrnG*	55	288
rrnH	rRNA	rRNA operon	*rrnH*	6	288, 418
selA	Selenium	Selenium incorporation		21	260
serA	Serine	Phosphoglycerate dehydrogenase (EC 1.1.1.95)	*serA*	62	410, 412, 418
serB	Serine	Phosphoserine phosphatase (EC 3.1.3.3)	*serB*	19	137, 410, 412, 418
serC	Serine	Requirement	*serC*	19	196, 418
serD	Serine	Requirement for pyridoxine plus L-serine or glycine		44	412, 418
sidC		Siderochrome utilization; ferrichrome transport; albomycin resistance		4	412, 418
sidF		Siderochrome utilization; ferrichrome transport; albomycin resistance		4	412, 418
sidK		Siderochrome utilization; albomycin resistance; receptor of phage ES18 in *S. typhimurium* and of T5 in *S. paratyphi* B	*tonA*	5	412, 418
smoB		Smooth colony morphology in histidine-constitutive mutants		99	410, 412, 418
spcB	Spectinomycin	Resistance (nonribosomal)		72	410, 412, 418
spoT	Spot	Guanosine 5′-diphosphate, 3′-diphosphate pyrophosphatase	*spoT*	79	400, 418
srlA	Sorbitol	*gut*; D-glucitol-specific enzyme II of the phosphotransferase system	*srlA*	59	419, W
srlB	Sorbitol	*gut*; D-glucitol-specific enzyme III of the phosphotransferase system		59	419, W
srlC	Sorbitol	*gut*; Regulatory gene	*srlC*	59	410, 412, 418, 419, W
srlD	Sorbitol	*gut*; sorbitol-6-phosphate dehydrogenase (EC 1.1.1.140)	*srlD*	59	W
srlM	Sorbitol	*gut*; DNA-binding protein which activates transcription of *srl*		59	W
srlR	Sorbitol	*gut*; regulatory gene	*srlR*	59	W
ssb	Single-strand binding	Single-strand DNA-binding protein	*ssb*	NM	313
stiA	Starvation inducible	*sinA*; starvation for carbon source or other requirements causes induction; repressed in *relA*		32	135, 450
stiB	Starvation inducible	Starvation for carbon source or other requirements causes induction		NM	450
stiC	Starvation inducible	Starvation for carbon source or other requirements causes induction		75	450
stiD	Starvation inducible	Starvation for carbon source or other requirements causes induction		32	450
stiE	Starvation inducible	Starvation for carbon source or other requirements causes induction		41	450
stiF	Starvation inducible	Starvation for carbon source or other requirements causes induction		NM	450
stiG	Starvation inducible	Starvation for carbon source or other requirements causes induction		86	450
stiH	Starvation inducible	Starvation for carbon source or other requirements causes induction		55	450
strB	Streptomycin	Low-level resistance plus auxotrophy; nonribosomal		53	410, 412, 418
strC		Streptomycin resistance, not *strA* or *strB*		NM	412, 418
stx	*Salmonella* toxin	Enterotoxin			84, U
sucA	Succinate	*lys*, *suc*; succinate requirement; α-ketoglutarate dehydrogenase (decarboxylase component)	*sucA*	17	410, 412, 418
sufA		Frameshift suppressor affecting proline tRNA and correcting +1 frame shifts at runs of C in the mRNA		77	256, 263, 410, 412, 418
sufB		Frameshift suppressor affecting proline tRNA and correcting +1 frame shifts at runs of C in the mRNA		45	256, 263, 410, 412, 418

Continued on following page

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
sufC		Recessive suppressor of +1 frameshift mutations at runs of C in the mRNA		15	410, 412, 418
sufD		Frameshift suppressor affecting glycine tRNA and correcting +1 frameshift mutations at runs of G in the mRNA		62	256, 410, 412, 418
sufE		Frameshift suppressor correcting +1 frameshift mutations at runs of G in the mRNA		89	256, 410, 412, 418
sufF		Recessive frameshift suppressor correcting +1 frameshift mutations at runs of G in the mRNA		11	410, 412, 418
sufG		Frameshift suppressor correcting +1 frameshift mutations at runs of A in the mRNA		15	256, 412, 418
sufH		Frameshift suppressor		50	256, 412, 418
sufI		Frameshift suppressor		11	256, 412, 418
sulB	Suppressor	Suppressor of *lon*	*sulB*	NM	96
sumA		Suppressor of missense		96	412, 418
supC	Suppressor	Ochre suppressor	*supC*	34	410, 412, 418
supD	Suppressor	Amber suppressor; serine insertion	*supD*	40	42, 218, 410, 412, 418
supE	Suppressor	*supY*; amber suppressor; glutamine insertion	*supE*	15	27, 42, 218, 410, 412, 418
supF	Suppressor	See *tyrT*			42, 218, 412, 418
supG	Suppressor	Ochre suppressor; lysine insertion	*supG*	NM	412, 418
supI	Suppressor	Nonsense suppressor induced by ICR-191 and allelic to *sufG*		15	412, 418
supJ	Suppressor	*supH*; amber suppressor; leucine insertion	*supJ*	82	42, 412, 418
supK	Suppressor	*supT*; recessive UGA suppressor; also corrects some frameshift mutations		62	410, 412, 418
supM	Suppressor	See *tyrU*			410, 412, 418
supQ	Suppressor	Suppressor of nonsense and deletion mutations of *leuD*		7	410, 412, 418, 464
supR	Suppressor	Amber suppressor; haploid lethal		82	410, 412, 418
supS	Suppressor	UGA suppressor; haploid lethal		82	410, 412, 418
supU	Suppressor	Suppressor of UGA muations; may be due to alteration of ribosome structure		72	418
tar	Taxis-associated receptor	Chemotaxis transduction polypeptide; aspartate receptor	*tar*	40	134, 331, 402, 418
tctA	Tricarboxylate transport	Membrane protein		57	418, 515, 516
tctB	Tricarboxylate transport	Membrane protein		57	515, 516
tctC	Tricarboxylate transport	Tricarboxylate-binding protein		57	412, 418, 447, 476, 515, 516
tctD	Tricarboxylate transport	Regulatory protein		57	515, 516
tctII	Tricarboxylate transport	Transport		15	AE
tctIII	Tricarboxylate transport	Transport		1	AE
tdk		Thymidine kinase (EC 2.7.1.21)	*tdk*	34	410, 412, 418
thiA	Thiamine	*thiG*; thiamine or thiazole moiety	*thiA*	89	410, 412, 418
thiC	Thiamine	*thiA*; thiamine or pyrimidine moiety	*thiC*	89	410, 412, 418
thiD	Thiamine	Thiamine requirement		46	410, 412, 418
thiE	Thiamine	Thiazole type		52	410, 412, 418
thiF	Thiamine	Thiazole type		52	410, 412, 418
thiH	Thiamine	*thiB*; thiamine requirement		54	412, 418
thiI	Thiamine	*thiC*; thiazole type		10	412, 418
thrA	Threonine	*thrC*, *thrD*; aspartokinase (EC 2.7.2.4) and homoserine dehydrogenase I (EC 1.1.1.3)	*thrA*	0	410, 412, 418
thrB	Threonine	*thrA*; and homoserine kinase (EC 2.7.1.39)	*thrB*	0	410, 412, 418
thrC	Threonine	*thrB*; and homoserine synthase (EC 4.2.99.2)	*thrC*	0	410, 412, 418
thrT	Threonine	*sufJ*; threonine tRNA		88	43, 256, 257
thyA	Thymine	Requirement	*thyA*	61	412, 418, 443
tip	Taxis-involved protein	Methyl-accepting chemotaxis protein (aspartate receptor)	*tap*	NM	403
tkt		Transketolase (EC 2.2.1.1)	*tkt*	NM	412, 418
tlp		Loss of protease II		37	412, 418

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TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
tlr		Thiolutin resistance; P22 development at high temperature		NM	418
tonB		*chr*; regulates levels of some outer membrane proteins; resistance to ES18; determines a salmonellocin; affects iron transport	*tonB*	34	111, 192, 410, 412, 418
top	DNA topoisomerase I	*supX*; Topoisomerase	*topA*	33	185, 241, 357, 359, 360, 379–381, 387, 410, 412, 418, 477, 526
tor		Trimethylamine oxide reductase		80	269, 270, 418
tppA	Tripeptide permease	Resistance to alafosfalin, regulator of *tppB*		74	153, 186, 228
tppB	Tripeptide permease	Resistance to alafosfalin; tripeptide permease		27	153, 186, 228, 229
tppR	Tripeptide permease	Regulator of tripeptide permease		3	229
traT	Transfer	Membrane protein cross-reacts immunologically with TraT protein of F; restores permeability mutants to normal		pSLT	469–472, 495, AC
tre	Trehalose	Utilization	*tre*	37	376, 377, 410, 412, 418
triM		Tricarboxylic acid metabolism; see *tctIII*		1	412, 418
triR		Tricarballylic acid transport; see *tctIII*		1	412, 418
trmD		Likely to be defective in the tRNA (guanine-1-)-methyltransferase (EC 2.1.1.31)	*trmD*	55	E
trpA	Tryptophan	*trpC*; tryptophan synthetase, component alpha (EC 4.2.1.20)	*trpA*	34	1, 320, 410, 412, 418, 452, 531
trpB	Tryptophan	*trpD*; tryptophan synthetase, component beta (EC 4.2.1.20)	*trpB*	34	410, 412, 418
trpC	Tryptophan	*trpE*; *N*-(5-phosphoribosyl) anthranilate isomerase and indole-3-glycerol phosphate synthase (EC 4.1.1.48)	*trpC*	34	209, 410, 412, 418
trpD	Tryptophan	*trpB*; anthranilate phosphoribosyltransferase (EC 2.4.2.18)	*trpD*	34	209, 410, 412, 418
trpE	Tryptophan	*trpA*; anthranilate synthase (EC 4.1.3.27)	*trpE*	34	103, 139, 410, 412, 418
trpR	Tryptophan	Resistance to 5-methyltryptophan; derepression of tryptophan enzymes	*trpR*	99	410, 412, 418
tsr		Chemotaxis receptor; serine specificity		NM	
ttr		Tetrathionate reductase		35	410, 412, 418
tufA		Protein chain elongation factor EF-Tu	*tufA*	71	211–213, 418
tufB		Protein chain elongation factor EF-Tu	*tufB*	88	211–213, 418
tyn		Tyramine oxidase		NM	418
tyrA	Tyrosine	Requirement	*tyrA*	55	410, 412, 418
tyrR	Tyrosine	Regulator gene for *aroF* and *tyrA*	*tyrR*	32	410, 412, 418
tyrT	Tyrosine	*supC*; ochre suppressor; tyrosine tRNA1	*tyrT*	34	42, 46, 126, 412, 418
tyrU	Tyrosine	*supM*; ochre suppressor; tyrosine tRNA2	*tyrU*	88	412, 418
ubiF	Ubiquinone	*cad*; deficient in ubiquinone synthesis; accumulates 2-octaprenyl-3-methyl-6-methoxy-1,4-benzoquinone	*ubiF*	14	418
ubiX	Ubiquinone	Growth stimulation by *p*-hydroxybenzoic acid; polyprenyl *p*-hydrobenzoate carboxylase		46	412, 418
udk		Uridine kinase (EC 2.7.1.48)	*udk*	43	410, 412, 418
udp		Uridine phosphorylase (EC 2.4.2.3)	*udp*	84	410, 412, 418
uhpA		Utilization of hexose phosphate		80	412, 418
uhpT		Hexosephosphate transport	*uhpT*		412, 418
umuC		Induction of mutations by UV; sensitivity to UV	*umuC*		184, 418, 440
uncA	Uncoupling	Membrane-bound (Mg^{2+}, Ca^{2+})ATPase	*unc*	81	418, 454
upp		Uracil phosphoribosyltransferase (EC 2.4.2.9)	*upp*	51	410, 412, 418
urs	Uracil	Uracil catabolism defect		30	513
use	Uracil sensitivity	Altered expression of genes *pyrA*, *pyrC*, *pyrD*, and *argI*		84	418
ushA	UDP sugar hydrolase	UDP-sugar hydrolase (5′-nucleotidase) (silent gene in *Salmonella* spp.)	*ushA*	11	62, 63, 412, 418, C
ushB	UDP sugar hydrolase	UDP-sugar hydrolase (membrane associated)		90	62, 63, 412, 418, C
usp	Ureidosuccinate	Permeability to ureidosuccinate (i.e., carbamyl asparatate)		NM	410, 412, 418
uvrA	UV	Repair of UV damage to DNA; UV endonuclease, component B	*uvrA*	91	410, 412, 418
uvrB	UV	Repair of UV damage to DNA; UV endonuclease component B	*uvrB*	18	410, 412, 418

Continued on following page

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternative symbol; enzyme deficiency or other phenotype[a]	Gene in *E. coli*[b]	Map (min)[c]	References[d]
uvrC	UV	Repair of UV damage to DNA	*uvrC*	40	412, 418
uvrD	UV	Repair of UV damage to DNA; increased sensitivity to mutagenesis by alkylating agents	*uvrD*	84	363–365, 412, 418
valS		Valyl-tRNA synthetase (EC 6.1.1.9)	*valS*		412, 418
viaA		*ViA*; Vi antigen		46	410, 412, 418
viaB		*ViB*; Vi antigen (in *S. typhosa*)		94	410, 412, 418
xylA	D-Xylose	Xylose isomerase (EC 5.3.1.5)	*xyl*	78	151, 412, 418
xylB	D-Xylose	Xylulokinase (EC 2.7.1.17)		78	151, 412, 418
xylR	D-Xylose	Regulation		78	151, 412, 418
xylT	D-Xylose	Transport		78	151, 412, 418

[a] Abbreviations: AMP, adenosine monophosphate; ATP, adenosine triphosphate; ATPase, adenosine triphosphatase; cAMP, cyclic AMP; CDP, cytidine diphosphate; CMP, cytidine monophosphate; CoA, coenzyme A; CTP, cytidine triphosphate; DAHP, 3-deoxy-D-arabinoheptulsonic acid 7-phosphate; dCTP, deoxycytidine triphosphate; dUMP deoxyuridine monophosphate; DMB, dimethylbenzimidazole; GMP, guanosine monophosphate; HP, hydrogen peroxide; IMP, inosine monophosphate; NAD(P), nicotinamide adenine dinucleotide (phosphate); PRPP, phosphoribosyl pyrophosphate; NAMN, nicotinic acid mononucleotide; NMN, nicotinamide mononucleotide; RNase, ribonuclease; TDP, thymidine diphosphate; UDP, uridine diphosphate; UMP, uridine monophosphate; UV, ultraviolet.

[b] The homologous gene in *E. coli* is described by Bachmann (14).

[c] Map positions in minutes are shown in Fig. 1, from 9 to 99 min. NM indicates that the gene is not mapped. The symbol pSLT indicates that the gene is on the plasmid of LT2.

[d] The numbers refer to references in Literature Cited. References 410, 412, and 418 refer to earlier editions of the linkage map in which other references to the indicated gene are given. Sanderson and Hurley (413) list all major references up to 1983 in a single source. There are many papers in the summary of the cellular and molecular biology of *E. coli* and *S. typhimurium* (346) which have important information on the genes of *S. typhimurium*. Letters A through AG refer to personal communications from the following sources: A, G. F.-L. Ames; B, E. Barrett; C, I. Beacham, D, P. R. Reeves; E, G. Björk; F, W. Boos; G, J. R. Curtiss and W. H. Benjamin, Jr.; H, T. Doak and J. R. Roth; I, D. Downs and J. R. Roth; J, T. Elliott, J. Delling, and J. R. Roth; K, T. Elliott and J. R. Roth; L, J. Escalante-Semerena and J. R. Roth; M, A. Fernandez-Briera and A. Garrido-Pertierra; N, P. Fields, E. Groisman, and F. Heffron; O, J. W. Foster; P, H.-S. Houng, D. J. Kopecko, and L. S. Baron; Q, K. T. Hughes, B. M. Olivera, and J. R. Roth; R, G. Kramer and B. N. Ames; S, N. D. Kredich; T, S. Maloy; U, J. W. Peterson; V, D. Roof and J. R. Roth; W, M. Saier; X, M. Schmid; Y, S. A. Simms and J. Stock; Z, J. K. Spitznagel; AA, G. V. Stauffer; AB, G. Storz and B. N. Ames; AC, S. Sukupolvi; AD, G.-M. Tang and J. R. Roth; AE, K. Widenhorn, J. Somers, and W. W. Kay; AF, N. Zhu and J. R. Roth; AG, C. Higgins.

and *E. coli* to see whether a name has been published, and they are encouraged to contact the *Salmonella* Genetic Stock Centre (SGSC) to see whether an unpublished name is on record. Allele number assignment should also be obtained from the SGSC. It is important that all mutations be identified by a unique allele number, but it is especially important for transposon insertions in which the gene name, based on the *z* . . system proposed by Hong and Ames (208) and used in the strains in Table 3, will change as the map location of the insertion is determined more exactly. It is also vital that strains be identified by a unique strain designation, which includes two or three capital letters (assigned by the SGSC to the laboratory) plus a number. The expanding use of the computer to keep records demands correct strain designations; suffixes and phenotypic designations after the strain designation to refer to derivatives of a strain are usually not accepted by the computer.

When possible, the same name should be used for homologous genes in related species such as *E. coli* and *Salmonella* spp. Many changes have been made in naming of genes in these organisms to bring them into correspondence, and each edition of the maps of both *S. typhimurium* and *E. coli* has seen such changes. A new system of naming the genes for flagellar synthesis and function, which recognizes the homology of these two genera, was proposed by Iino et al. (222) and is adopted in this edition (Table 1). The gene for control of P22 lysogeny, named *pox* in edition VI, is changed to *ply* (control of phage lysogeny); *poxA* in edition VII, as in *E. coli* K-12 (14), indicates the gene for pyruvate oxidase. The chromosomal gene for fimbriae, or pili, is renamed *fim*. As in *E. coli* K-12, the F-pili will continue to be called pili.

Modifications of the Transduction Mapping Function

In the previous editions of the map, we discussed the Kemper (251) and Wu (520) functions for using transductional linkages to estimate physical distances; this is also discussed by Low (299). The Wu method is clearly superior, since the method of Kemper ignores transduced fragments that end between the two markers used; this can lead to large errors when markers are widely separated. Both of these methods were developed with the assumption that the mutations used are point mutations and that the donor and recipient alleles do not differ significantly in size. This assumption is not valid when the donor marker is larger than the recipient marker, as would be the case when an insertion mutation is used as the donor marker or a deletion is used as recipient marker. These situations are now much more frequent with the use of transposons in genetic analysis. The excess size of the donor allele introduces material into the transduced fragment which (owing to lack of recipient homology) is not subject to recombination with the recipient chromosome; since transducing-fragment size is dictated by P22 packaging, inclusion of this nonparticipating donor material displaces sequences that could be used for recombination. Thus, use of donor markers of excess size necessitates modification of the Wu function. Since insertion elements of various sizes are used frequently in genetic analysis, we have modified the Wu function to accommodate such markers.

The Wu function, modified for use with donor markers larger than recipient alleles, is presented in Fig. 2. In this function, m represents the excess size of the selected donor marker and n represents the excess size of the unselected donor marker. (For example, if the donor allele is a Tn*10* insertion and the recipient allele is a wild-type gene, m = 10 kb.) If the donor and recipient marker are the same size (differing by a point mutation) or if the donor marker is smaller than the recipient allele, no correction is necessary (m = 0, n = 0); the expression then reduces to the original function suggested by Wu. As is apparent in Fig. 2, the

TABLE 2. Alternative gene symbols[a]

Former or alternative symbol	Current symbol
aniA	*hyd*
apeD	*apeR*
argA	*argE*
argB	*argA*
argC	*argB*
argD	*pyrA*
argE	*argG*
argF	*argH*
argG	*argD*
argH	*argC*
argT	*argU*
aroC	*aroE*
aroD	*aroC*
aroE	*aroD*
ars	*pyrA*
asc	*ent*
attP14	*atdA*
attP22 I	*ataA*
attP22I	*atcA*
attP27 I	*atbA*
attP27II	*atbB*
bac	*envB*
bfe	*btuB*
cad	*ubiF*
capR	*lon*
carA	*ptsI*
carB	*ptsH*
cheP	*cheA*
cheQ	*cheY*
cheT	*cheZ*
cheX	*cheB*
chr	*tonB*
cls	*katG*
dad	*dadA*
dra	*deoC*
drm	*deoB*
enb	*ent*
fhl	*fdhF*
fhlB	*hyd*
flaAI	*fliE*
flaAII.1	*fliF*
flaAII.2	*fliG*
flaAII.3	*fliH*
flaAIII	*fliI*
flaB	*fliP*
flaC	*flhA*
flaD	*fliQ*
flaE	*flhC*
flaFI	*flgA*
flaFII	*flgB*
flaFIII	*flgC*
flaFIV	*flgD*
flaFIX	*flgI*
flaFV	*flgE*
flaFVI	*flgF*
flaFVII	*flgG*
flaFVIII	*flgH*
flaFX	*flgJ*
flaH	*flgH*
flaK	*flhD*
flaL	*fliA*
flaM	*flhB*
flaN	*fliN*
flaP	*fliO*
flaQI	*fliL*
flaQII	*fliM*
flaR	*fliK*
flaS	*fliJ*
flaU	*flgL*
flaV	*fliD*
flaW	*flgK*
flaX	*flgF*
flaX	*fliR*
fruA	*ptsF*
galE	*galF*
glnF	*ntrA*
glnR	*ntrB*
glnR	*ntrC*
glu	*ptsG*
gpt	*ptsG*
gut	*srlC*
gut	*srlD*
gut	*srlA*
gut	*srlB*
gut	*srlM*
gut	*srlR*
gxu	*gpt*
H1	*fliC*
H2	*fljB*
hisU	*gyrB*
hisW	*gyrA*
hspLT	*hsdL*
hspS	*hsdSA*
ile	*ilvA*
ilvA	*ilvC*
ilvB	*ilvD*
ilvC	*ilvE*
ilvT	*brnQ*
leuT	*leuU*
lys	*sucA*
malB	*malE*
manA	*ptsM*
metI	*metA*
nalA	*gyrA*
nic	*nadB*
nicA	*nadA*
nml	*fliB*
O-5	*oafA*
ofi	*oafA*
ompB	*envZ*
ompB	*ompR*
oxrC	*pgi*
pasA	*pgi*
pil	*fim*
pncC	*pnuA*
pnu	*deoD*
poh	*oriC*
popC	*hemL*
pox	*ply*
psu	*rho*
ptdD	*pepD*
ptdN	*pepN*
ptdP	*pepP*
pup	*deoD*
putB	*putA*
rhl	*fljA*
rif	*rpoB*
rnsA	*rna*
rouC	*rfc*
sinA	*stiA*
smoA	*divC*
spcA	*rpsE*
strA	*rpsL*
suc	*sucA*
sufJ	*thrT*
supC	*tyrT*
supH	*supJ*
supM	*tyrU*
supT	*supK*
supX	*top*
supY	*supE*
thiA	*thiC*
thiB	*thiH*
thiC	*thiI*
thiG	*thiA*
thrA	*thrB*
thrB	*thrC*
thrC	*thrA*
thrD	*thrA*
tpp	*deoA*
tppA	*ompR*
tppB	*envZ*
trpA	*trpE*
trpB	*trpD*
trpC	*trpA*
trpD	*trpB*
trpE	*trpC*
trz	*cysK*
vh2	*hin*
ViA	*viaA*
ViB	*viaB*
wrkA	*divA*

[a] The alternative symbols have been used in past publications. It is recommended that their use be abandoned and that the current symbols, listed and described in Table 1 and in the references referred to there, be used in the future.

TABLE 3. Strains with transposable element-induced mutations in *S. typhimurium* LT2[a]

Map region (min)	Strains with insertions in region[b] [gene designation and allele no.::element inserted[c,d] (strain designation) (genetic linkage measured by P22-mediated joint transduction to linked genes)[e]]
0	*thr-447*::Tn*5* (TT2384[f]), *thr-557*::Tn*10* (TT191[f]), *thr-469*::Mu dI*1734* (TL886[g]), *thr-485*::Mu dI*1734* (TL888[g]); *zaa-1004*::Tn*10* (TT6736[f]) (*pnuA* 66%, *thr* 15%, *serB* 50%)
1	*pyrA234*::Tn*10* (TT1198[f]), *pyrA685*::Tn*10* (TT136[f]); *zab-2011*::Tn*10* (KS1169[h]) (*tctIII* 44%), *zab-2012*::Tn*10* (KS1170[h]) (*tetIII* 58%, *pyrA* 49%)
2	*leu-1173*::Tn*5* (TT2383[f]), *leu-1151*::Tn*10* (TT206[f])
3	*nadC1004*::Mu d1(Ap) (JF467[i]); *zad-804*::Tn*10* (TN1004[j]) (*leu* 75%), *zad-803*::Tn*10* (TN745[j]) (*leu* 30%), *zad-3131*::Tn*10*[d] (AK3131[j]) (*leuBCD* 24%), *zad-3137*::Tn*10*[d] (AK3137[j]) (*leuBCD* 9%), *zad-1022*::Tn*10* (TT7170[f]) (*nadC* 67%)
4	*zae-3149*::Tn*10*[d] (AK3149[j]) (*pepM* 42%), *zae-1633*::Tn*10* (Cm) (TN2852[j]) (*pepM* 75%), *zae-1614*::Tn*10* (Km) (TN2500[j]) (*pepM* 68%)
5	*pan-540*::Tn*10* (TT421[f]) *zaf-1351*::Tn*10* (HU521[k]) (*metP* 38%) (P1)[e]
6	*zag-208*::Tn*10* (SK2314[l]) (*glnD* 90%), *zag-3262*::Tn*10*[d] (AK3262[j]) (*dnaE* 54%), *zag-1254*::Tn*10* (RM268[m]) (*dnaE* 50%), *zag-305*::Tn*10* (DB9069[n]) (*dnaE* 50%)
7	*proA1656*::Tn*10* (JL2690[o]), *proA692*::Mu d1–8(Ap) (TT7882[f]); *proBA662*::Tn*10* (TT184[f]); *proB1661*::Tn*5* (JL3804[o]), *proB1657*::Tn*10* (JL2520[o]); *zah-3139*::Tn*10*[d] (AK3139[j]) (*proAB* 6%, *pepD* ND[e]), *zah-3150*::Tn*10*[d] (AK3150[j]) (*proAB* 82%, *pepD* ND), *zah-807*::Tn*10* (TN986[j]) (*proAB* 40%), *zah-3214*::Tn*10*[d] (AK3214[j]) (*proAB* 5%, *pepD* 4%), *zah-3215*::Tn*10*[d] (AK3215[j]) (*proAB* 7%, *pepD* 2%), *zah-806*::Tn*10* (TN801[j]) (*pepD* 75%)
8	*proC691*::Mu d1–8(Ap) (TT9670[f]), *proC693*::Mu d1–8(Ap) (TT9667[f]); *zai-808*::Tn*10* (TN789[j]) (*proC* 30%, on *proB* side), *zai-3029*::Tn*10*[d] (AK3029[j]) (*proC* 12%), *zai-3030*::Tn*10*[d] (AK3030[j]) (*proC* 28%), *zai-3170*::Tn*10*[d] (AK3170[j]) (*proC* 26%), *zai-3059*::Tn*10*[d] (AK3059[j]) (*clmB* 35%)
9	*zaj-1034*::Tn*10* (TT8024[f]) (*lon* 50%)
10	*psiC17*::Mu d1(Ap) (JF515[i]); *zba-883*::Tn*10* (TN1785[j]) (*apeB21* 50%), *zba-284*::Tn*10* (TN924[j]) (*thiI* 90%), *zba-6034*::Tn*10* (JF562[i]) (*psiC* 94%)
11	*oxiA1049*::Mu dA(Ap) (JF897[i]); *zbb-876*::Tn*10* (TN1781[j]) (*apeA* 75%), *zbb-121*::Tn*10* (TA4326[p]), *zbb-2351*::Tn*10* (SE7079[q]), *zbb-3089*::Tn*10*[d] (AK3089[j]) (*clmG* 19%), *zbb-3217*::Tn*10*[d] (AK3217[j]) (*purE* 14%), *zbb-3260*::Tn*10*[d] (AK3260[j]) (*purE* 7%), *zbb-3296*::Tn*10*[d] (AK3296[j]) (*clmG* 10%)
12	*purE884*::Tn*10* (TT289[f]) (*clmG* 68%), *apeE1*::Tn*5* (TN964[j]); *apeE2*::Tn*10* (TN966[j]); *zbc-809*::Tn*10* (TN780[j]) (*purE* 40%), *zbc-854*::Tn*10* (TN1338[j]) (*apeE* 25%), *zbc-873*::Tn*10* (TN1744[j]) (*apeE* 60%)
13	*ahp-11*::Tn*10* (TA4190[f])
14	*zbe-1023*::Tn*10* (TT7247[f]) (*nadD* 55%, *lip* 90%)
15	*zbf-99*::Tn*10* (TT2342[f]) (*supE* 74%), *zbf-904*::Tn*10* (SL2439[r]), *zbf-57*::Tn*10* (DB4289[n]) (*sufG* 75%), *zbf-903*::Tn*10* (SL2444[r])
17	*nadA213*::Tn*10* (TT398[f]), *nadA216*::Mu dI*1734*(Km) (TL1182[g]), *nadA219*::Mu dI*1734*(Km) (TL1184[g]), *nadA1011*::Mu dJ(Km) (JF1522[i]); *zbh-1009*::Tn*10* (TT6577[f]) (*nadA* ND)
18	*bio-102*::Tn*10* (TT403[f]), *bio-203*::Tn*10* (JL2688[o]); *zbi-812*::Tn*10* (TN1117[j]) (*galE* 90%), *zbi-3020*::Tn*10*[d] (AK3020[j]) (*oxd-8* 6%, *galE* 10%)
19	*aroA554*::Tn*10* (SL1346[r]); *zbj-3142*::Tn*10*[d] (AK3142[j]) (*oxd-12* 23%, *aroA* 2%)
20	*aspC409*::Tn*10* (SMS409[s]); *asp-544*::Tn*10* (TT176[f]); *pepN88*::Tn*10* (TN770[j]), *pepN103*::Mu dJ(Km) (TN2557[j]); *pncB150*::Tn*10* (TT6197[f]), *pncB213*::Mu d(Ap) (TT7233[f]); *zca-6001*::Tn*10* (JF188[i]) (*pncB* 80%), *zca-6008*::Tn*10* (JF330[i]) (*pncB* 65%), *zca-6009*::Tn*10* (JF331[i]) (*pncB* 65%), *zca-6010*::Tn*10* (JF332[i]) (*pncB* 96%), *zca-6011*::Tn*10* (JF333[i]) (*pncB* 70%), *zca-1048*::Tn*10* (TT7445[f]) (*pncB* 94%), *zca-843*::Tn*10* (TN799[j]) (*pepN* 30%)
21	*pyrD2286*::Tn*5* (TT2289[f]), *pyrD2266*::Tn*10* (TT468[f]); *zcb-3232*::Tn*10*[d] (AK3232[j]) (*pyrD* 32%, *pepN* 20%)
22	*oxiB1056*::Mu dA(Ap) (JF928[i]); *put-834*::Tn*5* (TT2292[f]); *putA810*::Tn*10* (TT946[f]); *putP214*::Tn*5* (CH378[t]), *putP201*::Mu d1(Ap) (CH321[t]), *putP1669*::Mu d1(Ap) (CH496[t])

Continued on following page

TABLE 3—*Continued*

Map region (min)	Strains with insertions in region[b] [gene designation and allele no.::element inserted[c,d] (strain designation) (genetic linkage measured by P22-mediated joint transduction to linked genes)[e]]
23	*pyrC691*::Tn*10* (TT459[f]), *pyrC1655*::Mu d2–8(Ap) (KP1581[u]); *zcd-1429*::Tn*10* (DB4672[n]) (*pyrC* 50%), *zcd-3176*::Tn*10*[d] (AK3176[j]) (*pyrC* 30%)
25	*purB877*::Tn*10* (TT282[f]); *pts4152*::Tn*10* (PP1139[v]); *zcf-850*::Tn*10* (TN1358[j]) (*purB* 50%), *zcf-3032*::Tn*10*[d] (AK3032[j]) (*oxd-6* 75%), *zcf-3041*::Tn*10*[d] (AK3041[j]) (*pepT* 41%, *oxd-18* 46%, *oxd-6* not linked), *zcf-3140*::Tn*10*[d] (AK3140[j]) (*oxd-6* 85%, *oxd-18* 2%, *pepT* 1%), *zcf-3233*::Tn*10*[d] (AK3233[j]) (*pepT* 20%, *oxd-6* 17%, *oxd-18* 23%)
27	*pncA148*::Tn*10* (TT6195[f]), *pncA212*::Mu d1–8(Ap) (TT7674[f]); *tppB9*::Tn*5* (CH345[r]), *tppB16*::Tn*10* (CH356[r]); *zch-1436*::Tn*10* (SK741[l]) (*gdh* 95%, *nit* 26%), *zch-1004*::Tn*10* (TT6736[f]) (*pncA* 66%)
28	*zci-3314*::Tn*10*[d] (AK3314[j]) (*dcp* 6%), *zci-847*::Tn*5* (TN1006[j]) (*dcp* 40%, *zci-3314* 16%)
30	*oxiC1048*::Mu dJ(Km) (JF1423[i]) (*zda-888* 98%); *zda-3127*::Tn*10*[d] (AK3127[j]) (*oxrA* 2%), *zda-3261*::Tn*10*[d] (AK3261[j]) (*oxrA* 2%), *zda-3258*::Tn*10*[d] (AK3258[j]) (*oxrA* 20%)
32	*ompD159*::Tn*10* (SH7235[w]); *stiA1*::Mu dJ (JF1222[i]); *zdc-6025*::Tn*10* (JF428[i]) (*stiA* 83%)
33	*pyrF696*::Tn*10* (TT464[f])
34	*oppA305*::Mu d1(Ap) (CH272[r]); *oppB255*::Tn*10* (CH50[r]), *oppB303*::Mu d1(Ap) (CH270[r]); *oppC304*::Mu d1(Ap) (CH271[r]); *oppD302*::Mu d2 (CH269[r]); *oppE326*::*cat* (Cm) (*CH1460*[r]); *trp-2475*::Tn*5* (TT4700[f]), *trp-2451*::Tn*10* (TT1333[f]), *trp-3477*::Mu dI*1734* (TT10270[f]); *zde-94*::Tn*10* (TT2337[f]) (*supF* 50%), *zde-815*::Tn*10* (TN817[f]) (*trp* 50%), *zde-605*::Tn*10* (TT2345[f]) (*supC* 54%), *zde-3026*::Tn*10*[d] (AK3026[j]) (chlC::Mu d1–8 41%), *zde-3211*::Tn*10*[d] (AK3211[j]) (*chlC*::Mu d1–8 48%), *zde-3218*::Tn*10*[d] (AK3218[j]) (*chlC*::Mu d1–8 11%), *zde-3219*::Tn*10*[d] (AK3219[j]) (*chlC*::Mu d1–8 6%)
36	*dadB5*::Tn*10* (DB7818[n]), *dadB6*::Tn*10* (DB7819[n]), *dadB7*::Tn*10* (DB7820[n]); *tre-57*::Tn*10* (TT1518[f]); *zdg-1201*::Tn*10* (DB7913[n]) (close to *dadA/B*), *zdg-3063*::Tn*10*[d] (AK3063[j]) (*dadB* 90%), *zdg-3234*::Tn*10*[d] (AK3234[j]) (*dadB* 40%), *zdg-3037*::Tn*10*[d] (AK3037[j]) (*dadB* 90%)
37	*tlp-71*::Tn*5* (TN921[j])
40	*cheA501*::Tn*10* (KK2051[x]); *cheB502*::Tn*10* (KK2078[x]); *cheY503*::Tn*10* (KK2014[x]); *zea-81*::Tn*10* (TT1952[f]) (*H1* 75%), *zea-1437*::Tn*10* (ST314[y]) (*cheR* 66%, *flaC* 84%), *zea-4*::Tn*10* (ST316[y]) (*H1* 1%, *flaK* 44%, *cheA* 44%, *cheW* 40%, *cheR* 33%, *cheB* 27%, *cheY* 33%), *zea-1434*::Tn*10* (ST322[y]) (*flaR* 33%, *cheR* 46%), *zea-618*::Tn*10* TT2070[f] (*supD* 90%), *zea-609*::Tn*10* (TT8388[f]) (*supD* ND), *zea-609*::Tn*10* (TT7610[f]) (*supD* ND)
41	*phs-2101*::Mu d1(Ap) (EB222[z]) (*zec-2*::Tn*10* 20%, *hisD*::Tn*10* 76%)
42	*hisA8676*::Tn*10* (NK1255[aa]); *hisB9442*::Tn*10* (TT7242[f]); *hisC8667*::Tn*10* (TT1127[f]); *hisD5408*::Tn*10* (TT34[f]), *hisD9953*::Mu dJ(Km) (TT10286[f]), *hisD9950*::Mu dI*1734*(Km) (TL1153[s]); *hisE9446*::Tn*10* (NK1146[aa]); *hisF8672*::Tn*10* (NK1256[aa]); *hisG9424*::Tn*10* (NK1158[aa]); *hisH9430*::Tn*10* (NK1220[aa]); *gnd-161*::Tn*10* (NK114[aa]); *zec-1*::Tn*10* (NK397[aa]) (*his* 50%, at *hisE* end), *zec-2*::Tn*10* (TT513[f]) (*his* 50%, at *hisO* end), *zec-3255*::Tn*10*[d] (AK3255[j]) (*his* 59%), *zec-2*::Tn*10* (NR5293[bb]) (*dam-1* 25%)
44	*zee-1*::Tn*10* (TT781[f]) (*his* ND), *zee-78*::Tn*10* (TT2242[f]) (*metG* 80%), *zee-3061*::Tn*10*[d] (AK3061[j]) (*metG* 6%)
45	*zef-754*::Tn*10* (TT5371[f]) (*hisW* 90%), *zef-4*::Tn*10* (DB9031[n]) (*gyrA* 95%)
46	*ack-408*::Tn*10* (SMS408[s]); *hisJ8908*::Tn*10* (TA3178[p]); *hisP5049*::Tn*10* (TA3090[p]), *hisP6641*::Tn*10* (TA3193[p]); *hisM6643*::Tn*10* (TA3195[p]); *hisQ6642*::Tn*10* (TA3194[p]); *ompC396*::Tn*10* (SH7241[w]); *pta-406*::Tn*10* (SMS406[s]); *zeg-102*::Tn*10* (TA3088[p]) (*dhuA* 80%), *zeg-3118*::Tn*10*[d] (AK3118[j]) (*oxd-3* 100%, *oxd-9* 100%, *oxd-10* 100%, *oxd-13* 100%, *oxd-14* 100%, *oxd-16* 100%), *zeg-3198*::Tn*10*[d] (AK3198[j]) (*oxd-3* 50%, *oxd-9* 48%, *oxd-10* 41%, *oxd-13* 37%, *oxd-14* 40%, *oxd-16* 24%), *zeg-3291*::Tn*10*[d] (AK3291[j]) (*oxd-3* 68%, *oxd-9* 67%, *oxd-10* 65%, *oxd-13* 57%, *oxd-14* 64%, *oxd-16* 60%)
47	*menB101*::Mu d1(Ap) (EB139[z]); *purF1714*::Tn*10* (TT317[f]); *hsiT290*::Tn*5* (TT5866[f]); *zeh-608*::Tn*10* (TA3092[p]) (*aroC* 40%, *hisT* 27%), *zeh-3138*::Tn*10*[d] (AK3138[j]) (*purF* 14%)
48	*crr-307*::Tn*10* (PP994[v]); *zei-636*::Tn*5* (TT4279[f])
49	*cysA1545*::Tn*5* (TT2373[f]), *cysA1367*::Tn*10* (NK186[aa]); *ptsI421*::Tn*10* (PP1228[v]); *zej-1031*::Tn*10* (TT7293[f]) (*ptsI* 95%, between *cysA* and *ptsI*), *zej-3271*::Tn*10*[d] (AK3271[j]) (*cysA* 99%)
51	*purC882*::Tn*10* (TT287[f]); *purI1757*::Tn*10* (TT11[f])
52	*guaA554*::Tn*10* (TT278[f]); *guaB544*::Tn*10* (TT275[f]); *pepB22*::Mu dJ(Km) (TN2727[j])

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TABLE 3—*Continued*

Map region (min)	Strains with insertions in region[b] [gene designation and allele no.::element inserted[c,d] (strain designation) (genetic linkage measured by P22-mediated joint transduction to linked genes)[e]]
53	*zfd-801*::Tn*10* (TN858[f]) (*strB* 33%, *pepB* 65%, *glyA* 10%), *zfd-1617*::Tn*10* (Km) (TN2560[f]) (*pepB* 99%)
54	*glyA971*::Tn*5* (SK2292[t]), *glyA540*::Tn*10* (TT418[f]); *purG1739*::Tn*10* (TT315[f])
55	*nadB214*::Tn*10* (TT399[f]), *nadB1017*::Mu dJ(Km) (JF1521[i]), *nadB901*::Mu d1(Ap) (JF153[i]), *nadB499*::Mu d*1734*(Km) (TT1114[f]); *tyrA555*::Tn*10* (TT126[f]); *pheA534*::Tn*10* (TT1339[f]); *zff-3028*::Tn*10*[d] (AK3028[j]) (*tryA* 6%), *zff-789*::Tn*10* (TT6581[f]) (*nadB* 55%), *zff-3181*::Tn*10*[d] (AK3181[j]) (*pheA* 38%, *tryA* 42%), *zff-3055*::Tn*10*[d] (AK3055[j]) (*tyrA* 2%, *phe* 0%), *zff-3222*::Tn*10*[d] (AK3222[j]) (*tyrA* 44%, *pheA* 54%), *zff-6029*::Tn*10* (JF509[j]) (*nadB* 30%), *zff-6030*::Tn*10* (JF516[i]) (*nadB* 60%)
56	*zfg-82*::Tn*10* (TT1896[f]) (*H2* 95%)
57	*proU1655*::Tn*10* (TL188[g]), *proU1697*::Tn*10* (CH710[r]), *proU1702*::Mu d1–8(Ap) (CH946[r]), *proU1705*::Mu dJ(Km) (CH1301[r]), *proU1873*::Mu d1(Ap) (TL335[g]), *proU1873*::Mu d*1734*(Km) (TL1150[g]), *proU1873*::Mu d1–8(Ap) (TL1310[g]), *proU1844*::Mu d1(Ap) (TL346[g]), *proU1844*::Mu d*1734*(Km) (TL671[g]), *proU1844*::Mu d1–8(Ap) (TL1311[g]); *tctI1511*::Tn*10* (KS204[h]), *tctI1512*::Tn*10* (KS205[h]), *tctI1513*::Tn*10* (KS202[h]), *tctI1514*::Tn*10* (KS203[h]); *zfi-2001*::Tn*10* (KS177[h]) (*H2* 90%, *tct* 22%, *zfi-2002* 2%), *zfi-2002*::Tn*10* (KS182[h]) (*H2* 2%, *tct* 16%, *zfi-2001* 2%), *zfi-2005*::Tn*10* (KS178[h]) (*tct* 12%, *srl* 0.1%, *nalB* 60%), *zfi-2006*::Tn*10* (KS179[h]) (*tct* 9%, *srl* 0.2%, *nalB* 60%), *zfi-2007*::Tn*10* (KS184[h]) (*tct* ND, *srl* ND, *nalB* ND), *zfi-2009*::Tn*10* (KS180[h]) (*tct* 3%, *srl* 0.6%, *nalB* 74%)
58	*zfj-1623*::Tn*10* (Cm) (TN2700[f]) (*recA* 88%)
59	*hyd-101*::Mu d1(Ap) (EB138[z]) (*srl*::Tn*10* 20%); *aniA1088*::Mu dJ(Km) (JF1534[t]) (*srl*::Tn*10* 45%); *mutS121*::Tn*10* (GW1702[cc]), *mutS121*::Tn*10* (GW1704[cc]); *srl-211*::Tn*5* (TT2979[f]) (*recA* ND), *srl-202*::Tn*10* (TT520[f]) (*recA* ND)
60	*cysC1511*::Tn*10* (TT173[f])
61	*argA1832*::Tn*10* (TT146[f]); *mutH101*::Tn*5* (GW1810[cc]), *mutH101*::Tn*5* (GW1824[cc]); *recBC531*::Tn*10* (DB4659[n]); *relA21*::Tn*10* (TT7542[f]); *zgb-1041*::Tn*5* (TT3680[f]) (*relA* 70%), *zgb-12*::Tn*10* (TT1710[f]) (*recBC* ND, *thy* ND), *zgb-18*::Tn*10* (TT1711[f]) (*recBC* 67%, *thy* 85%, *argA* 3%), *zgb-18*::Tn*10* (TT1712[f]) (*thyA* 84%, *recBC* 67%, *argA* 3%), *zgb-607*::Tn*10* (TA2437[p]) (*relA* 13%)
62	*lysA577*::Tn*5* (TT2376[f]), *lysA565*::Tn*10* (TT215[f]); *zgc-3121*::Tn*10*[d] (AK3121[j]) (*thyA* 12%), *zgc-3122*::Tn*10*[d] (AK3122[j]) (*thyA* 15%), *zgc-732*::Tn*10* (TT3680[f]) (*sufD* 90%, *recBC* 67%, *argA* 3%), *zgc-3132*::Tn*10*[d] (AK3132[j]) (*lysA* 20%), *zgc-3143*::Tn*10*[d] (AK3143[j]) (*lysA* 3%), *zgc-3146*::Tn*10*[d] (AK3146[j]) (*thyA* 18%), *zgc-3179*::Tn*10*[d] (AK3179[j]) (*lysA* 6%), *zgc-3231*::Tn*10*[d] (AK3231[j]) (*lysA* 12%)
63	*aniF1068*::Mu dA(Ap) (JF1101[i]) (*metC*::Tn*10* 20%); *aniG1072*::Mu dJ(Km) (JF1295[i]) (*metC*::Tn*10* 5%); *pepP6*::Tn*10* (TN853[j]); *serA977*::Tn*10* (TT169[f]); *zgd-866*::Tn*5* (TN1655[j]) (*serA* 60%), *zgd-3085*::Tn*10*[d] (AK3085[j]) (*serA* 36%, *pepP* 78%), *zgd-3209*::Tn*10*[d] (AK3209[j]) (*serA* 16%, *pepP* 10%), *zgd-3159*::Tn*10*[d] (AK3159[j]) (*serA* 64%)
64	*mutB131*::Tn*5* (GW1809[cc]); *zge-3076*::Tn*10*[d] (AK3076[j]) (*oxd-5* 10%, *metC* 0%, *clmF* 0%), *zge-3134*::Tn*10*[d] (AK3134[j]) (*oxd-5* 7%, *metC* 0%), *zge-3189*::Tn*10*[d] (AK3189[j]) (*oxd-5* 20%, *metC* 0%, *clmF* 0%), *zge-3012*::Tn*10*[d] (AK3012[j]) (*oxd-2* 59%), *zge-3084*::Tn*10* (AK3084[j]) (*oxd-2* 71%)
65	*metC1975*::Tn*10* (TT14[f]); *zgf-2010*::Tn*10* (KS1086[h]) (*metC* 30%, *cpd* 30%), *zgf-3017*::Tn*10*[d] (AK3017[j]) (*metC* 60%, *oxd-5* 2%, *clmF* 50%), *zgf-3213*::Tn*10*[d] (AK3213[j]) (*metC* 82%, *oxd-5* 30%), *zgf-3246*::Tn*10*[d] (AK3246[j]) (*metC* 30%, *oxd-5* 2%, *clmF* 25%)
68	*argG1822*::Tn*10* (TT142[f]); *zgi-3136*::Tn*10*[d] (AK3163[j]) (*argG* 49%, *dna-610* 85%), *zgi-3177*::Tn*10*[d] (AK3177[j]) (*argG* 34%, *dna-610* 88%)
69	*ntrA209*::Tn*10* (SK284[t]); *zgj-3265*::Tn*10*[d] (AK3265[j]) (*clmC* 6%), *zgj-201*::Tn*10* (SK195[t]) (*ntrA* 90%, *gltB* 7%)
70	*argR372*::Tn*10* (KR1400[dd])
71	*zhb-3195*::Tn*10*[d] (AK3195[j]) (*aroE* 84%, *oxrB* 5%, *clmE* 30%), *rpsL* 1%), *zhb-3124*::Tn*10*[d] (AK3124[j]) (*aroE* 69%, *oxrB* 2%), *zhb-3301*::Tn*10*[d] (AK3301[j]) (*aroE* 20%, *oxrB* 2%)
72	*crp-773*::Tn*10* (PP1037[v]); *zhc-1431*::Tn*10* (DU8802[ee]) (*crp* 50%)
73	*cysG1542*::Tn*5* (TT2290[f]), *cysG1510*::Tn*10* (TT172[f]); *zhd-117*::Tn*10* (TA3947[p]) (*malA* 46%), *zhd-3081*::Tn*10*[d] (AK3081[j]) (*aroB* 70%), *zhd-3173*::Tn*10*[d] (AK3173[j]) (*aroB* 86%)

Continued on following page

TABLE 3—*Continued*

Map region (min)	Strains with insertions in region[b] [gene designation and allele no.::element inserted[c,d] (strain designation) (genetic linkage measured by P22-mediated joint transduction to linked genes)[e]]
74	*dpp-101*::Tn*10* (CH736[t]); *envZ1005*::Mu dJ(Km) (CH1118[t]); *malQ210*::Mu dII(Ap) (TA3999[p]); *ompR1001*::Tn*5* (CH511[t]), *ompR1009*::Tn*10* (CH1351[t])
77	*opt-10*::Mu dJ(Km) (TN3101[j]); *zhh-3108*::Tn*10*^{d} (AK3108[j]) (*opt-10*::Mu dJ 50%), *zhh-3109*::Tn*10*^{d} (AK3109[j]) (*opt-10*::Mu dJ 78%), *zhh-3228*::Tn*10*^{d} (AK3228[j]) (*opt-10*::Mu dJ 20%), *zhh-3287*::Tn*10*^{d} (AK3287[j]) (*opt-10*::Mu dJ 60%), *zhh-3073*::Tn*10*^{d} (AK3073[j]) (*opt-10*::Mu dJ 40%), *zhh-3082*::Tn*10*^{d} (AK3082[j]) (*opt-10*::Mu dJ 40%), *zhh-3147*::Tn*10*^{d} (AK3147[j]) (*opt-10*::Mu dJ 55%), *zhh-1635*::Tn*10* (Cm) (TN3060[j]) (*optA1* 40%)
78	*xyl-183*::Tn*10* (SA1982[ff]); *zhi-3040*::Tn*10*^{d} (AK3040[j]) (*xyl* 4%), *zhi-1426*::Tn*10* (SA1980[ff]) (*xyl* 50%)
79	*zhj-1024*::Tn*5* (TT7244[f]) (*pyrE* 75%), *zhj-118*::Tn*10* (TA4217[p]) (*pyrE* 64%, *rfa* 93%), *zhj-901*::Tn*10* (SL1432[r]) (*cysE* ND), *zhj-1025*::Tn*10* (TT7245[f]) (*pyrE* 70%), *zhj-1404*::Tn*10* (SA2703[ff]) (*pyrE* 67%, *cysE* 2%), *zhj-1405*::Tn*10* (SA2704[ff]) (*pyrE* 78%, *cysE* 9%), *zhj-1416*::Tn*10* (SA2715[ff]) (*pyrE* 4%, *cysE* 87%), *zhj-3312*::Tn*10*^{d} (AK3312[j]) (*oxrE* 40%, *pyrE* 0%, *apeR* 0%)
80	*avtA1*::Tn*5* (CBS514[gg]); *mgtB10*::Mu dJ (Km) (MM196[hh]); *zia-1026*::Tn*5* (TT7246[f]) (*gltC* 40%), *zia-1438*::Tn*10* (DU2603[ee]) (*ilvB* 50%), *zia-3205*::Tn*10*^{d} (AK3205[j]) (*mgtB* 36%, *gltC* 0%, *dnaA* 0%, *pyrE* 0%, *apeR* 0%), *zia-3294*::Tn*10*^{d} (AK3294[j]) (*gltC* 5%, *pyrE* 0%, *mgtB* 0%, *ilvC* %), *zia-3295*::Tn*10*^{d} (AK3295[j]) (*mgtB* 4%, *ilvC* 0%, *pyrE* 0%, *apeR* 0%), *zia-1630*::Tn*10* (Cm) (MM223[hh]) (*mgtB* 85%, *gltC* 29%), *zia-3048*::Tn*10*^{d} (AK3048[j]) (*mgtB* 95%, *gltC* 2%), *zia-3104*::Tn*10*^{d} (AK3104[j]) (*mgtB* 11%, *pyrE* 0%), *zia-3123*::Tn*10*^{d} (AK3123[j]) (*mgtB* 95%, *pyrE* 0%, *apeR* 0%), *zia-3125*::Tn*10*^{d} (AK3125[j]) (*mgtB* 95%), *zia-3306*::Tn*10*^{d} (AK3306[j]) (*mgtB* 10%)
81	*zib-1040*::Tn*5* (TT8038[f]) (*hisU* 30%), *zib-748*::Tn*10* (TT3920[f]) (*hisU* 30%), *zib-6*::Tn*10* (DB9048[n]) (*dnaA* 95%), *zib-3119*::Tn*10*^{d} (AK3119[j]) (*apeR49*::Tn*5* 94%, *dnaA* 56%, *apeR* 22%), *zib-3120*::Tn*10*^{d} (AK3120[j]) (*apeR49*::Tn*5* 100%, *dnaA* 68%, *apeR* 24%), *zib-3130*::Tn*10*^{d} (AK3130[j]) (*apeR49*::Tn*5* 36%, *dnaA* 90%, *apeR* 7%), *zib-3241*::Tn*10*^{d} (AK3241[j]) (*apeR49*::Tn*5* 85%)
82	*apeR47*::Tn*5* (TN901[j]); *psiR1*::Tn*10* (JF753[i]); *unc-102*::Tn*10* (TT1042[f]); *zic-3068*::Tn*10*^{d} (AK3068[j]) (*apeR49*::Tn*5* 49%, *apeR* 50%), *zic-870*::Tn*10* (TN1741[j]) (*apeR* 66%), *zic-851*::Tn*10* (TN1239[j]) (*apeR* 73%)
83	*cya-1091*::Tn*10* (PP1002[v]), *cya-1092*::Tn*10* (PP1038[v]); *ilvA595*::Tn*10* (TT58[f]), *ilvA2173*::Tn*10* (TT4[f]); *ilvD2103*::Tn*10* (TT81[f]); *ilvE1005*::Tn*10* (TT48[f]), *ilvE201*::Tn*10* (CBS514[gg]), *ilvE2903*::Tn*10* (TT71[f]); *ilvG1006*::Tn*10* (TT61[f]), *ilvG1007*::Tn*10* (TT66[f]); *zid-62*::Tn*10* (TT2104[f]) (*cya* 95%, *hisR* 40%), *zid-64*::Tn*10* (TT2010[f]) (*ilv* 40%), *zid-3265*::Tn*10*^{d} (AK3265[j]) (*ilvC* 9%, *corA* 0%, *clmD* 70%)
84	*metE2092*::Tn*5* (TT2370[f]), *metE862*::Tn*10* (TT218[f]); *uvrD421*::Tn*5* (GW1808[cc]); *corA45*::Mu dJ(Km) (MM199[hh]); *pepQ8*::Mu dJ(Km) (TN2712[j]); *zie-822*::Tn*5* (TN1064[j]) (*metE* 70%), *zie-1634*::Tn*10* (Cm) (TN3005[j]) (*polA* 3%), *zie-3145*::Tn*10*^{d} (AK3145[j]) (*pepQ* 89%, *ilvC* 33%, *metE* 0–1%), *zie-3024*::Tn*10*^{d} (AK3024[j]) (*pepQ* 65%, *metE* 2%, *polA* 6%), *zie-3161*::Tn*10*^{d} (AK3161[j]) (*pepQ* 4%, *metE* 30%, *corA* 8%, *ilvC* 0%), *zie-3162*::Tn*10*^{d} (AK3162[j]) (*metE* 75%, *corA* 25%, *ilvC* 0%, *pepQ* 0%), *zie-3299*::Tn*10*^{d} (AK3299[j]) (*clmA* 70%), *zie-3305*::Tn*10*^{d} (AK3305[j]) (*clmA* 45%), *zie-3228*::Tn*10*^{d} (AK3228[j]) (*clmA* 45%), *zie-3229*::Tn*10*^{d} (AK3229[j]) (*clmA* 50%), *zie-3235*::Tn*10*^{d} (AK3235[j]) (*metE* 7%, *corA* 37%, *pepQ* 0%)
85	*glnA392*::Tn*5* (SK1239[l]), *glnA120*::Tn*10* (SK389[l]); *ntrB137*::Tn*10* (SK398[l]); *ntrC352*::Tn*10* (SK835[l]); *zif-205*::Tn*10* (SK273[l]) (*glnA* 40%, *rha* proximal), *zif-214*::Tn*10* (SK811[l]) (*ntrC* 75%, *polA* proximal)
86	*zig-1935*::Tn*10* (JF1493[i])
87	*metB879*::Tn*10* (TT225[f]); *metF2094*::Tn*5* (TT2381[f]), *metF877*::Tn*10* (TT233[f]); *ppc-2*::Tn*10* (KS77[h])
88	*argH1823*::Tn*10* (TT137[f]); *oxiE4*::Mu dJ(Km) (JF1420[i]); *psiB12*::Mu d1(Ap) (JF512[i]); *zii-166*::Tn*5* (TA4101[p]), *zii-614*::Tn*10* (TT2385[f]) (*argH* 30%, *sufJ* 40%)
89	*aceA1*::Tn*10* (TT8027[f]), *aceA101*::Tn*10* (MS226[ii]), *aceA112*::Mu dJ(Km) (MS1309[ii]); *aceB102*::Tn*10* (MS229[ii]), *aceB113*::Mu dJ(Km) (MS1311[ii]); *met-900*::Tn*10* (TT256[f]); *purD1735*::Tn*10* (TT311[f]); *purH1829*::Tn*5* (TT2792[f]), *purH887*::Tn*10* (TT292[f]); *thiA541*::Tn*10* (TT501[f])
90	*zja-1230*::Tn*10* (TA5053[p]) (*malB* 28%, *malG* ND)
91	*malE776*::Tn*10* (TS616[ii]); *malL212*::Tn*10* (TA5051[p]); *pepE8*::Mu dJ(Km) (TN2719[j]); *zjb-861*::Tn*5* (TN1425[r]) (*malB* 5%, *pepE* 3%)
93	*aniB1054*::Mu dA(Ap) (JF1437[i]) (*zid-27*::Tn*10* 15%); *aniC1052*::Mu dJ(Km) (JF1325[i]) (*zid-27*::Tn*10* 98%); *fhl-101*::Mu d1(Ap) (EB137[z]) (*melA*::Tn*10* 21%, *zdj-27*::Tn*10* 46%); *mel-351*::Tn*10* (TT1662[f]); *proP1667*::Tn*5* (CH638[t]), *proP1673*::Mu d1(Ap) (CH500[t]), *proP1681*::Mu d1(Ap) (TL357[g]), *proP1696*::Mu d1(Ap) (TL372[g]), *proP1681*::Mu dI*1734*(Km) (TL1278[g]), *proP1696*::Mu dI*1734*(Km) (TL1280[g]); *psiD19*::Mu dA(Ap) (JF663[i]) (*zid-27*::Tn*10* 74%); *zjd-27*::Tn*10* (TT1800[f]) (*proP* 60%, *mel* 30%)

Continued on following page

TABLE 3—*Continued*

Map region (min)	Strains with insertions in region[b] [gene designation and allele no.::element inserted[c,d] (strain designation) (genetic linkage measured by P22-mediated joint transduction to linked genes)[e]]
94	*mutL111*::Tn*10* (GW1714[cc])
95	*poxA401*::Tn*10* (SMS401[s])
96	*mutL111*::Tn*10* (GW1716[cc]); *purA874*::Tn*10* (TT273[f]); *zjg-3290*::Tn*10* (AK3290[j]) (*purA* 88%)
97	*pepA201*::Tn*10* (CH351[r]); *zjh-1628*::Tn*10* (Cm) (MM116[hh]) (*pyrB* 20%, *mgtA* 40%)
98	*pyrB692*::Tn*10* (TT460[f]); *argI1850*::Tn*5* (TT2374[f]), *argI1833*::Tn*10* (TT147[f]); *zji-842*::Tn*5* (TN1040[j]) (*argI* 88%, *pyrB* 44%, *pepA* 28%), *zji-1072*::Tn*5* (TT8082[f]) (*hsaSA* 20%, *argI* 7%), *zji-3196*::Tn*10*[d] (AK3196[j]) (*pepA* 38%, *argI* 72%, *pyrB* 22%), *zji-3200*::Tn*10*[d] (AK3200[j]) (*pepA* 15%, *argI* 30%, *pyrB* 21%), *zji-33*::Tn*10* (TT563[f]) (*pyrB* 83%), *zji-841*::Tn*10* (TN797[j]) (*pepA* 45%), *zji-3103*::Tn*10*[d] (AK3103[j]) (*pepA* 46%, *argI* 82%, *pyrB* 22%), *zji-3160*::Tn*10*[d] (AK3160[j]) (*pepA* 46%, *argI* 88%, *pyrB* 8%), *zji-1073*::Tn*10* (TT8088[f]) (*argI* 15%), *zji-3252*::Tn*10*[d] (AK3252[j]) (*pepA* 70%, *argI* 52%, *pyrB* 12%), *zji-3253*::Tn*10*[d] (AK3253[j]) (*pepA* ND, *argI* ND, *pyrB* ND)
99	*serB965*::Tn*10* (TT21[f]); *zjj-1433*::Tn*10* (DB9159[n]) (*dnaC* 95%), *zjj-3042*::Tn*10*[d] (AK3042[j]) (*serB* 99%, *trpR* 44%, *thrB* 0%), *zjj-3112*::Tn*10*[d] (AK3112[j]) (*serB* 78%, *trpR* 46%, *thrB* 0%), *zjj-3116*::Tn*10*[d] (AK3116[j]) (*thrB* 8%, *serB* 25%, *trpR* 76%), *zjj-3240*::Tn*10*[d] (AK3240[j]) (*serB* 86%, *trpR* 27%, *thrB* 0%)

[a] All strains are available from SGSC. Strains with transposon insertions were obtained from many different laboratories; these sources are indicated in footnotes *f* to *jj*.

[b] Insertions into a gene cause loss of gene function; e.g., insertions in *thr* result in a Thr$^-$ (threonine-requiring) phenotype. Transposon insertions which cause no detectable change in phenotype are designated *zxx* with an allele number. If these insertions are found to be linked to specific genes and hence at known map locations, this gene designation is changed according to the system of Hong and Ames (208), so insertions at 0 min are *zaa*, insertions at 1 min are *zab*, insertions at min 2 are *zac*, etc. The allele number of the mutation produced by the insertion does not change if the map position must be changed as data are refined, but the second two letters in the gene designation may be changed. Full genotypes of the strains are not described in the table.

[c] The transposable element is usually the normal form, with Tn*5* determining Km^r and Tn*10* determining Tc^r, and with transposition properties which are normal for the element. In some cases a recombinant with altered antibiotic resistance has been constructed; this is shown in parentheses. In some cases transposition properties have been modified; this is indicated by a footnote.

[d] The Tn*10* element used in strains from C. G. Miller (footnote *j*) (264) is a mutant called Δ16Δ17; it is defective in transposition and thus behaves as a very stable insertion.

[e] ND, Linkage was detected, but the exact percentage of linkage was not determined. P1, P1 phage is used rather than P22.

[f–jj] The laboratory in which the strain carrying the transposon insertion originated is indicated as follows: [f] J. R. Roth; [g] L. Csonka; [h] J. M. Somers and W. W. Kay; [i] J. W. Foster; [j] C. G. Miller; [k] P. D. Ayling; [l] S. Kustu; [m] R. Maurer; [n] D. Botstein; [o] J. L. Ingraham; [p] B. N. Ames; [q] M. B. Schmid; [r] B. A. D. Stocker; [s] T. Van Dyk; [t] C. F. Higgins; [u] J. Neuhard; [v] P. W. Postma; [w] P. H. Mäkelä; [x] K. Kutsukake; [y] D. E. Koshland, Jr.; [z] E. L. Barrett; [aa] N. Kleckner; [bb] D. M. Podger; [cc] G. C. Walker; [dd] R. A. Kelln; [ee] R. O. Burns; [ff] K. E. Sanderson; [gg] C. M. Berg; [hh] M. Maguire; [ii] S. Maloy; [jj] E. T. Palva.

correction becomes largest when both donor and recipient markers exceed the size of normal alleles. The graphs of this function in Fig. 2 can be used to directly estimate the physical distance from cotransduction percentages obtained in several common situations.

Comparison of the Linkage Maps of *S. typhimurium* and *E. coli* K-12

Earlier comparisons of the linkage maps of *S. triphimurium* and *E. coli* K-12 (412, 418), updated and extended recently (392), continue to show striking overall similarity in the map order of genes. However, some differences have been found. A large inversion covering up to 15% of the chromosome, in the region of the *trp* gene, has occurred between the two genera. In addition, Riley and Krawiec (392) have noted regions of the *E. coli* and *S. typhimurium* genomes which seem, on the basis of comparisons of the linkage maps, to have either excess genetic segments or deletions with respect to one another. Their analysis indicated that the *E. coli* map has 14 excess segments (loops) not found in *S. typhimurium* and that *S. typhimurium* has 15 segments not found in *E. coli*. The hypothesis that these segments represent excess DNA in one genus or the other has not generally been confirmed by physical data, except in the region of the *lac* gene, which is present in *E. coli* but absent in *S. typhimurium*. In this case, loops of DNA, which carry *argF*, *cod*, *lac*, or *phoA*, are present in *E. coli* but missing from *S. typhimurium*, whereas *newD* and *supQ* are on a loop of *S. typhimurium* DNA (64, 277).

In addition to the above, where genes are apparently present in one genus but not the other, in some cases genes having similar phenotypes are given the same name but are shown at very different locations in the two genera. These include the following: *cod* (cytosine deaminase), 69 min on the *S. typhimurium* map, 94 min in *E. coli*; *pck* (phosphoenolpyruvate carboxylase), 13 and 75 min, respectively; *pheR* (regulatory gene for *pheA*) 64 and 94 min, respectively; and *fim* (type I somatic fimbriae), 14 and 98 min, respectively. These examples may represent homologous genes at different map locations, nonhomologous genes with related and compensating functions, or genes that have been incorrectly mapped in one of the two genera. An apparent conflict of this type was resolved by Burns and Beacham (63). They discovered that the *ushA* gene (for uridine diphosphate-sugar hydrolase) at 11 min in *E. coli* has a silent homolog at the same location in *S. typhimurium*, designated *ushA*°; *S. typhimurium* does not make the homologous enzyme. However, *Salmonella* spp. possess a functionally similar but substantially different, nonhomologous uridine diphosphate-sugar hydrolase encoded by the *ushB* gene, which maps at 90 min in *S. typhimurium*. The *ushB* enzyme is membrane associated, has broader specificity, and is genetically and immunologically distinct from the *ushA* gene product of *E. coli*. It is not clear whether *E. coli* contains a gene which is homologous to *ushB*.

The genes for the pathway of isoleucine-valine biosynthesis provide another example of differences between the two genera. Valine inhibits isoleucine biosynthesis in *E. coli*

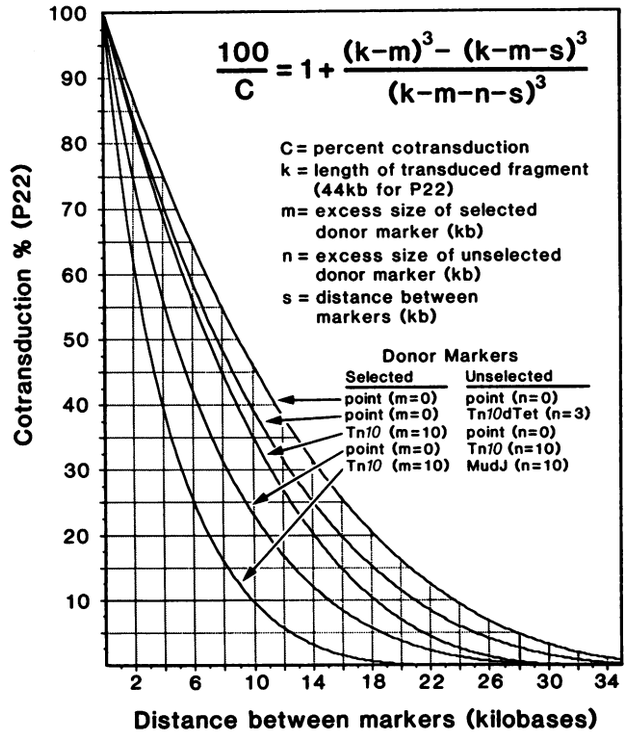

FIG. 2. Relationship between P22 cotransduction frequency and distance between markers (in kilobases) based on the formula of Wu (520) with modification to accommodate the use of donor markers with increased dimensions.

K-12 but not in *S. typhimurium* LT2. This results from the presence of different sets of acetohydroxy acid synthetase isozymes in the two organisms. *S. typhimurium* has two active acetohydroxy acid synthetase isozymes, the valine-sensitive form (encoded by *ilvBN*) and the valine-resistant form (encoded by *ilvGM*). Isoleucine synthesis in *E. coli* is valine sensitive because only the two valine-sensitive isozymes, determined by the *ilvBN* and *ilvHI* operons, are expressed. Although wild-type strains of *S. typhimurium* LT2 and *E. coli* K-12 each fail to express an acetohydroxy acid synthetase enzyme found in the other genus, mutants which express the cryptic enzyme can be isolated (101, 451, 512).

IS Elements in the Two Genera and Their Role in Spontaneous Mutation

The existence of insertion (IS) elements was initially detected by the occurrence of absolutely polar insertion mutations among spontaneous mutations of the *gal* operon of *E. coli* (239, 435). A surprisingly large fraction (ca. 15%) of spontaneous mutations were due to transposition of elements already present in the genome of *E. coli*. Other selection systems also reveal that IS elements are an important contributor to spontaneous mutation in *E. coli* (434). In *Salmonella* spp. the situation seems to be distinctly different. Insertion mutations have not been identified as a frequent subset of spontaneous mutants. In fact, to our knowledge, only one mutation caused by an endogenous insertion sequence (IS*200*) has been described in *Salmonella* spp., although transposition of this element to new sites has been observed by Southern hybridization (274–276).

An extensive (but unsuccessful) hunt for spontaneous insertion mutations in the *his* operon was recently completed (J. Casadesus and J. R. Roth, unpublished data). The selection method (salt tolerance of certain *his* constitutive mutants which are salt sensitive) detects insertions of Tn*5* and Tn*10* in the *his* operon, and it allows survival of strains carrying the one known IS insertion mutant, *hisD984*::IS*200*. No spontaneous insertions were recovered among several thousand spontaneous auxotrophs analyzed. The hunt included the use of strains in which IS*200* was known to be present in 13 copies (LT2 has only 6 copies) and in which extensive transposition had been observed. We tried several growth conditions including anaerobic liquid cultures and long-term storage of strains in standard stab vials.

The differences between *Salmonella* spp. and *E. coli* may be important in understanding the natural distribution of IS elements and the role of these sequences in chromosome evolution. The standard *E. coli* IS elements (IS*1* to IS*5*), generally thought to be ubiquitous in enteric bacteria, are not present in *Salmonella* spp. (352, 429). Conversely, IS*200* is found in most *Salmonella* species but not in *E. coli* or several related enteric bacteria (274–276).

Plasmid pSLT in *S. typhimurium* LT2

Because the plasmid content of strains of bacteria is often extremely variable, the plasmids are not normally considered part of the linkage map of the organism; usually only the bacterial chromosome is considered in discussions of genetic content. Wild-type *S. typhimurium* strains may carry plasmids of one or more of the Inc groups; these plasmids are not considered to be part of the genome. However, the original line of *S. typhimurium* LT2 contained a specific plasmid which could be considered part of the normal genotype. This plasmid has been called the "cryptic plasmid," the "virulence plasmid," MP10, pLT2, the 100-kb plasmid, or the 60-megadalton plasmid; we refer to it in this publication (and in reference 415) as pSLT, standing for Salmonella LT (having originally cleared the designation with the Plasmid Reference Center, Stanford University, Stanford Calif.).

The 60-megadalton plasmid pSLT was first recognized in *S. typhimurium* LT2 by Dowman and Meynell (113). Investigators working with *S. typhimurium* LT2 must be aware of this plasmid. For two reasons, we are treating it as part of the genome. First, the plasmid is an almost invariable part of the genotype. It is carried by all the lines of LT2 which we have tested, except for the few strains from which it has been intentionally eliminated. This is true even though LT2 has been in culture for many years and has been subjected to innumerable single-colony isolations. In addition to stable maintenance in LT2, pSLT or a closely related plasmid is commonly found in independent *S. typhimurium* isolates. pSLT from *S. typhimurium* LT2 has a characteristic fingerprint when digested with restriction enzymes *Pst*I and *Sma*I; a plasmid with this fingerprint was present in 67% of a set of wild-type *S. typhimurium* isolates representing most of the different bacteriophage types, but was found more commonly in veterinary than in human isolates (56). It was not found in any of the 96 strains representative of other *Salmonella* serotypes, and thus it is serotype specific (56). Among wild-type strains of *S. typhimurium*, plasmids with molecular masses of 60 megadaltons (90 kb) were reported as common by Jones et al. (238), who also suggested that they were similar to the plasmid of *S. typhimurium* LT2. Related plasmids were found in *S. typhimurium* and strains of other serotypes (374).

The second argument for considering pSLT to be part of the normal genotype is that it influences the phenotype in several ways, and thus mutations in the plasmid may be confused with chromosomal mutations. The influence of pSLT on the phenotype of LT2 was first recognized by Smith et al. (444), who noted that it encoded Fin^+ (fertility inhibition) properties. This Fin^+ property reduces the fertility of F^+ and Hfr strains, but the fertility of *S. typhimurium* LT2 in F-mediated conjugation can be restored by methods described by Sanderson et al. (415). In addition, defects affecting the membrane have been traced to mutations in pSLT. Sukupolvi et al. (472) isolated mutants of *S. typhimurium* LT2 with altered outer membrane permeability; these were placed in three classes called SS-A, SS-B, and SS-C (for supersensitive A, B, and C, respectively). The phenotype of the SS-A mutants was restored to normal by the *traT* gene product of the F factor or of R factor R6-5 (471). The SS-A gene has been cloned, and it produces a protein recognized by monoclonal anti-TraT antibodies (M. Rhen, S. Sukupolvi, J. Hackett, and D. O'Conner, personal communication). The gene producing the protein has been mapped on the plasmid of *S. typhimurium*; we are following the proposal of these authors in calling the gene *traT* by analogy with the *traT* gene of the F factor, and for convenience this gene is shown in Table 1.

It has been shown that a related plasmid in wild-type strains of *S. typhimurium* is associated with virulence, adhesion, invasiveness, and resistance to serum (238, 496). In a worldwide survey of *Salmonella* isolates, Helmuth et al. (181) found that a correlation between the presence of large plasmids and resistance to serum exists not only in *S. typhimurium* but also in *S. enteriditis*, *S. dublin*, and *S. heidelberg*. Restoration of resistance to serum can be mediated by a 1.0-kb cloned segment of the plasmid (16, 496; G. W. Jones, personal communication). A gene from the plasmid of a virulent strain of *S. typhimurium* was cloned on a 2.1-kb fragment and shown to express an 11-kilodalton protein that mediates serum resistance in both *E. coli* K-12 and a virulence-plasmid-free (serum-sensitive) strain of *S. typhimurium* (169, 170). Gulig and Curtis (166) have confirmed a role for the plasmid in virulence, primarily in invasion of mesenteric lymph nodes and spleen after oral inoculation of mice, but they could not confirm an influence of the plasmid on phagocytosis and killing by macrophages or on sensitivity to serum. A plasmid of similar size in *S. gallinarum* also contributes to virulence (22).

A detailed restriction map derived from the plasmid from *S. typhimurium* C5 has been published (324). It indicates a physical map of 90 kb and restriction sites for *Hin*dIII, *Bam*HI, and *Bgl*II. Restriction maps of a related 80-kb plasmid from *S. dublin* have also been presented (26, 179). In addition, numerous Tn*10* and Tn*5* insertions into the plasmid have been isolated, and some of the genes from the plasmid have been cloned and analyzed. We hope that this component of the genotype can be reported in detail in the next edition of the linkage map of *S. typhimurium*.

We hope that correspondence among researchers with these plasmids will lead to the use of a standardized nomenclature. This nomenclature must take account of the fact that although the plasmids in lines of LT2 are for the most part identical, the apparently related plasmids in other wild-type, non-LT2 strains of *S. typhimurium* will not be identical to pSLT.

MATERIALS AND METHODS FOR GENETIC ANALYSIS

Below is a series of genetic methods and information relevant to genetic analysis of *Salmonella* spp. Most of these have appeared since the last edition of this map (418); several were mentioned previously but are now described in more detail. They indicate the range of materials and methods available for genetic analysis of this organism.

Advantages of Transposition-Defective Transposons

Although transposons encoding drug resistance have been extremely valuable for genetic analysis and study of chromosomal rearrangements (422–424), several problems with their use suggest that it is preferable to use derivatives that are defective for transposition. The chief problem derives from the fact that most of the elements in current use (e.g., Tn*5* and Tn*10*) include IS sequences that are independently transposable (30, 436). Thus, strains carrying a transposon accumulate secondary transpositions of the entire element and, more frequently, of the component IS sequences. The IS sequences are particularly troublesome; they transpose more frequently than the entire element and are most probably not noticed phenotypically, since they do not encode drug resistance. An additional problem with the complete transposons is the high frequency of adjacent deletion formation. Depending on the selection used, these can contribute significantly to the adjacent mutations isolated in local mutagenesis experiments. Disadvantages in deletion generation are described below.

To avoid the problems associated with active transposition, a series of defective transposons have been developed. These elements can transpose only if transposase function is provided by a plasmid or another element; once removed from this source of transposition function, the elements are stable and are not subject to further acts of transposition. Included among these defective elements are derivatives of Tn*10* that encode tetracycline resistance, Tn*10* dTc (505); kanamycin resistance, Tn*10* dKm (505); and chloramphenicol resistance, Tn*10* dCm (122a). Transposition-defective derivatives of Tn*5* have also been constructed (122, 397).

Transposition-defective derivatives of Tn*10* (Tn*10* dTc) have several advantages over the original Tn*10* element as a means of generating deletions affecting the chromosomal region adjacent to the insertion site. This method has been used extensively, since one can select for Tc^s derivatives of the parent Tn*10* insertion mutant and then identify deletions among the survivors (38, 306). Two disadvantages of using the original Tn*10* are as follows: (i) deletion endpoints are not randomly distributed, but tend to fall at hot spots that may coincide with preferential insertion sites for Tn*10* (350), and (ii) many of the selected Tc^s derivatives are due to imprecise excision of Tn*10*, which removes the Tc^r determinant but does not delete chromosomal material adjacent to the insertion site. Results vary from one insertion to another, but for many insertions, deletions are found to be rare among Tc^s derivatives. These problems are solved by using Tn*10* dTc as the parental insertion. Selection for Tc^s derivatives yields about 100-fold fewer survivors, and a higher proportion are the desired deletions. Presumably, these deletions occur by mechanisms independent of Tn*10* transposition functions and therefore show a more nearly random endpoint distribution. It has been found (K. Hughes, personal communication) that the selection for Tc^s survivors is more effective if performed at high temperature (40 to 42°C).

A Standard Set of Tn*10* dTc Insertions for Mapping

It has now become standard practice to place a Tn*10* element near a gene of interest to facilitate the analysis. Such linked insertions are useful in local mutagenesis of the region, in determining its chromosomal map location, and perhaps in cloning the gene by using Tc^r as a selective marker. Generally, insertions have been found by selecting or screening for an appropriate linked insertion from a pool of random Tn*10* insertions; typically, between 0.1 and 1% of the insertions in a large pool prove to be cotransducible by P22 with a particular chromosomal marker. The identified insertion must then be made phage free, transferred to a new genetic background to ensure purity, and mapped.

An improvement on this methodology has been devised by Kukral et al. (264). They constructed a collection of 279 strains, each carrying one randomly placed insertion of Tn*10* dTc (3 kb). The random distribution of these insertions was ensured by their isolation method. First, a library of λ clones of *Salmonella* DNA was constructed. For each of 279 λ clones, one derivative was isolated that acquired a Tn*10* dTc transposition in the cloned fragment. Each of these insertions was then transferred by transductional recombination into the *Salmonella* chromosome, generating a series of 279 strains, each with one insertion. The random chromosomal distribution of these inserts is governed by the randomness of the λ cloning procedure and is not disturbed by the tendency of Tn*10* to insert at hot spots.

This collection of strains has many applications. One can screen these strains, rather than a large pool of Tn*10* inserts, in search of insertions linked to a gene of interest; the vast majority of mutations tested are linked to at least one of the Tn*10* dTc insertions in the collection. The chromosomal map locations of many of these insertions are known (264), and more data are accumulating. Therefore, there is a substantial and growing probability that by demonstrating cotransduction of a new mutation with one of these inserts, one can immediately learn the chromosomal map position of the new mutation.

Furthermore, the method by which these insertions were isolated provides a λ clone corresponding to the chromosomal region of each of the Tn*10* dTc insertions. Thus, by demonstrating genetic linkage to an insertion in the collection, one identifies an available clone that carries the gene of interest or sequences very close to it.

The value of this collection increases in proportion to the number of people who use it and communicate their observations to the authors of the method. Therefore, we urge researchers to use this set of strains; they will be immediately useful, and data obtained with them will increase the value of this method. These strains can be obtained from the SGSC, University of Calgary, Calgary, Alberta, Canada.

A Collection of Transposon Insertions in the Chromosome

Summarizing the work of numerous investigators, Sanderson and Roth (418) and Berg and Berg (28) showed the map locations of many transposon insertions into the chromosome of *S. typhimurium*. Many of these strains, plus the strains in the Tn*10* set of Kukral et al. (264), have been assembled at the SGSC. They are described in Table 3 and are available on request. They are valuable for testing linkages, for transferring mutant alleles of genes, and for making Hfr strains. Researchers with strains which have insertions in genes or map locations presently not represented or which provide insertions with different transposons are requested to contact the SGSC. These additional strains would greatly improve the utility of the collection.

Outward Promoters of Transposons Tn*5* and Tn*10*

Several observations have suggested that transposons Tn*5* and Tn*10* include promoters whose transcripts cross into bacterial sequences adjacent to the transposon insertion site (31, 33, 88, 439). Usually these transcripts do not reach the next distal gene in the operon because they are terminated at rho-dependent termination sites (87); therefore, most insertions are absolutely polar. The outward promoters become apparent in *rho* mutant strains or when no transcription termination site is present between the insertion site and the next translation start site. The outward promoters of Tn*5* may be weakly expressed in *E. coli*, since attempts to demonstrate Tn*5* activation of a silent *lacZ* gene in *E. coli* have shown only activation by insertion of IS*50*L; all insertions conferring a Lac^+ phenotype were oriented such that the *neo* promoter of IS*50*L was directed toward the expressed *lacZ* gene (252).

Transferring Large, Genetically Specified Regions of the Chromosome to Plasmids by Transposition

It is frequently useful to clone genetically defined regions of the chromosome. In the absence of sequence data or for very large chromosomal regions, it may be useful to clone by transposition. This has been done for three distinct regions by the general method outlined below.

The basic idea is to flank the region of interest with copies of Tn*10*, thereby constructing a composite transposon that includes the region of interest and can transpose to a plasmid. For a composite with flanking Tn*10* elements, several alternative pairs of IS*10* endpoints can act to permit transposition. The smallest transposable unit includes the chromosomal segment and one IS element from each of the flanking copies of Tn*10*; this unit can transpose to a plasmid without including the Tc^r determinant. The gene of interest is then added to the plasmid between inverse copies of IS*10* and is therefore not subject to recombinational removal (D. Roof, D. Andersson, and J. R. Roth, unpublished results). This method has been used for placing genes on F-prime plasmids and for identifying the desired plasmids by complementation following conjugational transfer into a new genetic background. Defective Tn*10* derivatives can serve as one of the flanking elements, but in this case no simple IS*10* sequences are present on that side of the gene of interest, and one drug resistance marker must invariably be added to the plasmid (G.-M. Tang and J. R. Roth, unpublished results).

The frequency of transposition of large elements is lower than that of the parent Tn*10*, as has been clearly shown by Morisato et al. (334); therefore, use of this method to clone large regions is aided (even for nondefective Tn*10*) by inclusion of a plasmid that overproduces Tn*10* transposase (24).

Use of Mu d-*lac* Transposons in *Salmonella* spp.

Basic methods for use of the Casadaban Mu d-*lac* constructions in *Salmonella* spp. were discussed previously (418). In general, these methods consist of using the Mu d prophages as transposons, moving them into new backgrounds by P22-mediated transduction. The following Mu d derivatives are in regular use in *Salmonella* spp. (i) Mu

d1(Ap *lac*) is the original construction of Casadaban, a full-sized prophage (ca. 39 kb) which forms Lac$^+$ operon fusions; it is capable of independent transposition but is defective for virion formation (77). (ii) Mu d2(Ap *lac*) is a full-sized prophage which forms Lac$^+$ gene (protein) fusions; it is capable of independent transposition but is defective for virion formation (76). (iii) Mini-Mu d(Km *lac*) are smaller elements (ca. 10 kb), such as Mu dJ (derived from Mu d1) and Mu dK (from Mu d2), which are defective for transposition and virion formation but form the same types of fusions as their parent phages (78, 220); The mini-Mu d systems have been used to clone genes in *S. typhimurium* (163). (iv) Mu dF(Km *lacI*$^+$*Z*$^+$ is a recombinant between a Lac$^+$ Mu d phage constructed by Chaconas et al. (79) and the mini-Mu d elements described above. Mu dF (ca. 10 kb) carries a complete, regulatable *lac* operon and a Km^r determinant and is defective for both transposition and virion formation. This phage allows one to introduce a functional *lac* operon at any point in the *Salmonella* chromosome. Mu dF is useful in monitoring Mu d transposition, since all insertions are Lac$^+$ and can be scored on indicator media (R. Sonti, unpublished results). (v) Mu dA and Mu dB are conditionally transposition-defective derivatives of the original Mu d1 (operon fusions) and Mu d2 (gene fusions) of Casadaban (77). The mutant elements transpose normally in strains carrying an amber suppressor, but in the absence of a suppressor, they show a very low frequency of both initial conservative transposition and secondary replicative transposition. Owing to their large size (ca. 39 kb), these transposition-defective Mu d prophages can be used to construct duplications and deletions and to orient transcripts in the *Salmonella* chromosome (219).

A related method (C. G. Miller, personal communication) allows easy isolation of mini-Mu d (Mu dJ and Mu dK) insertions into cloned sequences carried on plasmids such as pBR322. In this procedure, the target plasmid is introduced into a strain carrying both a mini-Mu d element and a transposition-proficient, temperature-inducible (*c*ts) Mu d1. The resulting strain is shifted to 42°C (to induce transposition) and simultaneously infected with P22 HT (high-frequency transducing) phage. Some of the transducing particles in the lysate formed carry plasmids which have received a mini-Mu d element by transposition. These plasmids can be identified by using the lysate to transduce a recipient selecting for both the antibiotic resistance encoded by the plasmid and the antibiotic resistance conferred by the mini-Mu d. These transductants can then be screened to identify those which have lost a phenotype associated with a gene in the cloned sequence. The mini-Mu d insertion mutation can frequently be introduced into the chromosome by transduction with the insertion-bearing plasmid as donor, using selection for only the mini-Mu d-encoded antibiotic resistance and screening the transductants for those which have lost the plasmid-encoded resistance.

A convenient method has been used to achieve transposition of the defective mini-Mu elements (described above) in *Salmonella* spp. (270). The method is based on the use of a strain which carries a defective mini-Mu element Km^r and a transposition-proficient Mu d1 element at closely linked sites in the *his* operon; in this strain the Mu d1 prophage is oriented with its transposition function nearest to the mini-Mu prophage. When this strain is used as the donor in the transductions, selecting Km^r transduced fragments that include the Mini-Mu (Km^r) element frequently include the portion of the transposition functions of the adjacent Mu d1 prophage. In such fragments, the transposase can act in *cis* to permit transposition of the defective mini-Mu element; the transposition functions are lost, since the fragment of the helper prophage can neither transpose nor recombine into the recipient chromosome. As a result, one lysate may be used as a reagent to mutagenize any recipient strain with mini-Mu insertions; each Km^r transductant inherits by transposition only one Mu d element and is left with no residual transposition functions. (As described below, this method should not be used in *rec*-deficient recipients, since a very high frequency of deletions will result.)

Deletion Generation by Mu d-*lac* Transposition

It is known that when phage Mu inserts, about 10% of the resulting lysogens carry deletions of target material (M. M. Howe and D. Zipser, Abstr. Annu. Meet. Am. Soc. Microbiol. 1974, V208, p. 235). This phenomenon is also true for the Mu d-*lac* phages, and in this situation the formation of deletions can (in principle) be misleading to those studying the behavior of the resulting operon fusions. For example, imagine that you isolate a Trp-Mu d insertion and (unbeknownst to you) an associated deletion forms that removes the *trp* promoter, fusing the *lacZ* gene to an unrelated promoter. The regulatory behavior of the fusion might be incorrectly attributed to the *trp* control region, since the fusion was isolated as a simple Trp$^-$ auxotroph. Although this is only a hypothetical problem, we have found conditions under which the frequency of deletions is extremely high. The possibility of deletions should be kept in mind in interpreting the behavior of Mu d-generated fusions. This is particularly true under the conditions described below; here the phenomenon provides a potentially useful means of generating chromosomal deletions.

When a Mu d prophage of any type is introduced into a new genetic background by P22 transduction, the frequency of lysogens arising by transposition from the transduced fragment to the chromosome is greatly reduced in recipients that are *recA*, *recB*, or *recC* (216). This is true whether the transposition functions are provided by the Mu d element itself, by a helper Mu genome, or by a plasmid present in the recipient cell. The lysogens that are found have a very high frequency of deletions associated with the inserted element. The reduction in lysogen frequency appears to be due to secondary transposition events (including deletions) that cause cell death. Apparently, these conditions delay repression, and the introduced element frequently transposes several times before lysogeny is established. This problem also applies to mini-Mu d elements introduced by Mu virions, with helper Mu particles providing transposase. The delaying effect of *rec* mutations on lysogeny is not seen for a full sized Mu d1 element or for a plaque-forming Mu Ap^r phage when they are introduced by a Mu virion. This phenomenon may be due to a protein that is normally injected by the Mu virion in association with particular sequences of the Mu genome; P22 virions cannot provide this protein, and mini-Mu genomes are not able to associate with or be helped by the protein (R. Sonti and D. Keating, personal communication). According to this model, prompt establishment of a lysogen requires either *recABC* functions or the hypothetical protein. Regardless of the explanation, this phenomenon can be a serious problem if one tries to isolate Mu d insertions in a *rec*-deficient strain. This phenomenon can be exploited to isolate deletions; the deletions encountered under these conditions are frequently very large (R. Sonti, D. Keating, and J. R. Roth, unpublished results).

Use of Mu d Elements to Construct Deletions and Duplications and To Determine Transcript Orientation

Methods of constructing deletions and duplications and determining transcript orientation involve the use of Mu dA or Mu dB (described above) under conditions where no transposition functions are present; thus, inheritance must occur by standard recombination events. The methods depend on the fact that Mu dA or Mu dB elements are so large (ca. 39 kb) that a single P22-transduced fragment (44 kb) rarely includes an entire Mu d element; two transduced fragments are required which must recombine with each other as well as with the bacterial chromosome to generate a transductant that inherits (by standard recombination) the entire prophage. To construct duplications and deletions by recombination, P22 phage is prepared on each of two full-sized, transposition-defective Mu dA or Mu dB insertions, and a mixture of the two lysates is used as the donor in a transduction cross selecting for inheritance of the ampicillin resistance encoded in the Mu d elements. Several different pairwise combinations of transduced fragments are able to recombine to generate a complete prophage. (For example, a fragment with the left half of one parental Mu d element might recombine with a fragment carrying the right half of the other parental Mu d element.) If the parental Mu d insertions are in the same orientation in the chromosome, the hybrid fragments will recombine with the chromosome to generate either a duplication or a deletion of the chromosomal material between the two insertion sites. Thus, a duplication and a deletion (if viable) can be constructed by recombination between any pair of direct-order Mu d insertions. If the parental Mu d insertions are in inverse orientation, the hybrid fragments cannot be inherited.

These mixed-lysate transductions can be used to determine the orientation of transcription of any gene for which a Mu d-*lac* fusion is available. By using the unknown insertion in combination with a Mu d insertion whose orientation is known, one can determine whether the unknown fusion is in the same orientation as that of the known insertion (duplications form) or the opposite orientation to that of the known insertion (no duplications form). This permits one to infer the direction of transcription of the unknown gene.

A Locked-In P22 Prophage That Preferentially Donates Fragments from Any Specified Region of the Chromosome

A transposable, locked-in P22 prophage has been constructed that promises to have wide and varied applications to the genetics and molecular biology of *Salmonella* spp. The construction, made by Youderian et al. (529), is a transposon with the ends derived from phage Mu; between these ends was placed a chloramphenicol resistance determinant and a P22 prophage which lacks the attachment sites (*att* sequences) and thus cannot excise upon P22 induction. When the prophage is induced, phage functions are expressed, and DNA replication is initiated at the P22 replication origin. Replication proceeds primarily in one direction from that site, out of the transposon and into adjacent bacterial sequences. DNA is packaged processively into P22 heads, primarily from the P22 *pac* site within the transposon. The first headful includes some prophage sequences and some adjacent bacterial sequences; the second and third include bacterial sequences from one side of the transposon insertion site. The resulting lysate is greatly enriched for virions including about 100 kb (about three headsful) of bacterial sequences (3 min or more of the genetic map).

The uses of Mu d-P22 are many and are likely to increase. The DNA sequences adjacent to the transposon site are sufficiently enriched in the lysate that DNA from such a lysate can be used directly for double-strand dideoxynucleotide sequencing. Furthermore, the transduction frequency for markers in this region is high enough that unselected transductions are possible. In essence, these lysates provide a DNA source approaching that expected of a λ clone (if a λ clone could include a 100-kb insert). A set of 300 random Mu d-P22 insertions has been constructed by David Hillyard and used to locate previously unmapped sequences cloned from the *Salmonella* chromosome (D. Hillyard and K. Nielsen, personal communication). A lysate of each Mu d-P22 lysogen was probed with the sequence in question; the prophage of the lysogens that hybridized strongly were then mapped. As a wider set of mapped Mu d-P22 lysogens is assembled, this may become the method of choice for chromosome mapping both by transduction (with the Mu d-P22 lysates as donors) and by hybridization (with the Mu d-P22 lysates as DNA sources for hybridization). An additional use of these phages may be in the study of transductional recombination, since these lysates provide a high concentration of a fairly uniform set of transduced fragments capable of repairing a particular recipient mutation. A further application of Mu d-P22 is presented below (Tn*phoA*).

The Mu d-P22 transposon can transpose at random to virtually any site on the chromosome (when Mu transposition functions are provided), or it can recombine with previously characterized Mu d insertions to convert them to Mu d-P22 insertions. Two forms of Mu d-P22 have been constructed, Mu dP and Mu dQ, with P22 sequences placed in opposite orientation within the Mu d-derived prophage. Therefore, any previously characterized Mu d prophage can be converted by recombination to a Mu d-P22 that will package deoxyribonucleic acid (DNA) from either side of the insertion site.

Use of Tn*phoA* in *Salmonella* spp.

An extremely useful probe for genetic identification of proteins with signal sequences has been devised by Manoil and Beckwith (308, 309). This probe is a derivative of transposon Tn*5* that carries the gene for alkaline phosphatase cloned near the outside end of IS*5*L; the cloned *phoA* gene lacks a translation initiation site and the signal sequence that is essential for its export and therefore for enzymatic activity. When Tn*phoA* inserts, alkaline phosphatase activity is produced only if the insertion forms a protein fusion to a target gene which can provide signals for export of the *phoA* sequences. The transposon, Tn*phoA*, has been used to determine which portions of a membrane protein are inside and which are outside the inner membrane of the cell.

In using Tn*phoA* in *Salmonella* spp., there is some good news and some bad news. The good news is that *Salmonella* spp. lack alkaline phosphatase, and therefore one need not remove this activity before using Tn*phoA*; the bad news is that the acid phosphatase of *Salmonella* spp., which is controlled by the *phoN* and *phoP* genes, is sufficiently active to make all strains score positive for the chromogenic phosphatase substrate, 5-bromo-4-chloro-3-indolyl phosphate, toluidine salt (X-P). Therefore, either a *phoN* or a *phoP* mutation must be introduced into *Salmonella* spp. before the effects of Tn*phoA* can be scored (N. Zhu, unpublished results).

The frequency of PhoA$^+$ insertions of Tn*phoA* is expected to be low, since the element must insert into a gene which

can provide transport signals and the insertion must be in the correct orientation and translational reading frame. In practice, this is approximately 1 PhoA$^+$ fusion per 1,000 Tn*phoA* transpositions. Therefore, it is important to be able to screen or select the inserts of interest from a large pool of transposition mutations. The Mu d-P22 (described above) developed by Youderian et al. (529) can be used to achieve this. By placing Tn*phoA* near a properly oriented Mu d-P22, one can induce the P22 prophage and generate a lysate with a very high frequency of fragments that include Tn*phoA*. These lysates are suitable for isolation of PhoA$^+$ insertions of Tn*phoA* in *Salmonella* spp. (H. Ardehali, N. Zhu, and D. Roof, unpublished results).

The following technical points are old news to investigators working with phosphatase, but they may be helpful for new users of Tn*phoA* in *Salmonella* spp. The calcium chelator ethylene glycol-bis(β-aminoethyl ether)-*N*,*N*,*N*′,*N*′-tetraacetic acid (EGTA; often used by *Salmonella* geneticists to prevent growth of P22 on transduction plates) can chelate metal essential to alkaline phosphatase and thereby prevent detection of activity. Second, to detect Tn*phoA* fusions with low activity, one can put plates containing X-P indicator in the cold room for several days to allow colonies to develop more color before scoring for fusions (S. Maloy, personal communication).

Gene Replacement Methods

Several methods are available for transferring mutant bacterial sequences into and out of the bacterial chromosome (104, 167, 253). A new method, developed by Blum (P. Blum, personal communication), seems particularly attractive, especially since it has been extensively applied to analysis of *Salmonella* spp.

The method involves the M13mp vector system (321, 528). Some of the M13mp phages carry amber mutations in gene II of the viral genome; since these phages cannot replicate in hosts lacking an amber suppressor, they do not kill the host. Therefore, when one selects for inheritance (by a suppressor-free recipient) of a phenotype encoded in these phages, one demands recombination between the cloned insert and the chromosome, which integrates the phage genome into the chromosome and provides for its replication.

The developers of the new method have identified phenotypes of the resulting M13mp lysogens that allow selection against the lysogen; these selections demand excision of the prophage vector, which has a high probability of leaving behind in the chromosome information originally present in the cloned insert in the infecting M13mp. The phenotypes noted for lysogens are as follows: resistance to killing by phage R17, ability to rescue an M13 gene V amber mutant, sensitivity to bile salts, and resistance to colicin E1. Thus, one can select for M13 vector integration by using resistance to R17 or colicin and for excision of the M13 prophage by using resistance to bile salts (deoxycholate). The same set of selections can be used to transfer chromosomal mutations to a wild-type clone of the corresponding chromosomal region (P. Blum, personal communication).

Conjugal Transfer of pBR322 and pBR325

Conjugal transfer of pBR322 and pBR325 can be mediated by pRK2013 (ColE1 with *tra*$^+$ of RP4 as a mobilizer) (91). This transfer requires the Mob protein from ColE1, *tra*$^+$ gene products of RP4 (109), and a *cis*-acting site, *bom*, on the plasmids to be mobilized (132); this site is deleted from pUC plasmids and pBR328, which cannot be mobilized.

A triparental mating involving a strain carrying pRK2013, a strain carrying pBR322 or pBR325, and a recipient strain results in mobilization of pBR322 or pBR325 onto the recipient strain, with no evidence of recombination with pRK2013 (L. Csonka, personal communication). Even transfer from *E. coli* to *Salmonella* spp. across the restriction barrier proved feasible.

Use of Coliphages in *Salmonella* spp.

Phage lambda will infect and grow in *Salmonella* spp. once two problems are solved. First, λ requires the *lamB* gene product of *E. coli*, which serves as the λ receptor protein; second, the *nusA* protein of *E. coli* is required for the antitermination control of λ. Solutions to these problems have been developed and improved (176).

The most recent development is a plasmid constructed by Harkki et al. (175). This plasmid includes a highly expressed *lamB* gene and the *nusA* gene of *E. coli*. The authors have demonstrated the efficacy of this plasmid by using lambda-based genetic methods to construct and clone an *ompC*::*lac* fusion in *Salmonella* spp.

Phage T4 can grow in *Salmonella* strains carrying a *galE* mutation (R. Meyers and S. Maloy, personal communication). This opens to *Salmonella* spp. the use of bacterial selection methods that are based on sensitivity or resistance to T4 mutants.

Generalized transduction by phage P1 can be done in *S. typhimurium* by using *galE* (galactose-epimerase defective) mutants; the wild-type strains with normal lipopolysaccharide do not adsorb the phage, but the *galE* mutants will do so (124). *galE* mutants can be constructed by using P22-mediated transduction with Tn*10* transposons inserted close to *galE* or by selecting for resistance to Felix-0 (F0) phage which adsorbs to normal lipopolysaccharide. P1 transduces a fragment of DNA considered equivalent to 2 min of chromosomal length, giving it an advantage over P22, which transduces a fragment equivalent to 1 min.

Initiation signals for packaging analogous to the *pac* sequences of the generalized transducing phage P22 have been recognized on the chromosome of *S. typhimurium*. These sites are involved in packaging chromosomal DNA into transducing particles. Chromosomal *pac* sites have been cloned and analyzed (425–427, 502).

Transformation Methods

Transformation of *S. typhimurium* with plasmid DNA by using $CaCl_2$-treated cells was first described by Lederberg and Cohen (282). MacLachlan and Sanderson (301) reported that by using modifications of the $CaCl_2$ method they could obtain ca. 10^4 pBR322 transformants per μg of DNA with smooth strains, but up to 10^5 to 10^6 transformants with *galE* mutants and some *rfa* mutants.

Electrotransformation methods, developed originally for use with animal and plant cells, have recently been extended to bacteria. High-voltage exponential-decay discharge systems which produced voltage in the range of 5 to 13 kV/cm applied for a very brief period (2 to 30 ms) gave plasmid transformation of 10^6 per μg of DNA in *Campylobacter jejuni* (328) and 10^9 to 10^{10} per μg of DNA in *E. coli* K-12 (112). Calvin and Hanawalt (69) report electrotransformation of *E. coli* in the same range of efficiency, with different equipment producing about 14 kV/cm. Using the same equipment and techniques as those used by Dower et al. (112) for *E. coli*, we obtained 10^8 to 10^9 transformants per μg

TABLE 4. Strains of *S. typhimurium* with F-prime factors which carry chromosomal genes of *E. coli* K-12, as well as transposon Tn*5* or Tn*10*[a,b]

Strain	F′ factor	Approx position of factor on *E. coli* map (min)[c]	Genes confirmed on F′[d]	Chromosomal markers	Phenotype of strain	Mutations complemented in this strain by F′ genes	Temp sensitivity[d]
TL851	F′104	97–7	*leu*$^+$ *proB*$^+$*A*$^+$ *zzf-20*::Tn*10*	*pyrB655* Δ(*proBA*)*47*	PyrB$^-$ (Ura) Tc^r	Pro$^+$	R
TL852	F′104	97–7	*leu*$^+$ *proB*$^+$*A*$^+$ *zzf-20*::Tn*10*	*pyrB655* Δ(*proBA*)*47*	PyrB$^-$ (Ura) Tc^r	Pro$^+$	S
TL873	F′128	6–8	*proB*$^+$*A*$^+$ *zzf-20*::Tn*10 argF*$^+$ *lacI4000* Φ(*lacI-Z*) *Y*$^+$	*argI537 ara-9 fol-1*	Ara$^-$ Fol$^-$ Tc^r	Arg$^+$, Lac$^+$	R
TL874	F′128	6–8	*proB*$^+$*A*$^+$ *zzf-20*::Tn*10 argF*$^+$ *lacI4000* Φ(*lacI-Z*) *Y*$^+$	*argI537 ara-9 fol-1*	Ara$^-$ Fol$^-$ Tc^r	Arg$^+$, Lac$^+$	S
TL275	F′128	6–8	*proB*$^+$*A*$^+$ *argF*$^+$ *lacI400* Φ(*lacI-Z*) *Y*$^+$, Tn*5* in unknown location on F′128	Δ(*proBA*)*21 pyrA8 argR5 fol-181 rpsL201*	PyrA$^-$ (Arg + Ura) Fol$^-$ Sm^r Km^r	Pro$^+$	R
TT1948	F′152	12–17	*nadA*$^+$ *zzf-20*::Tn*10*	*hisO124 hisB2142 nad-506 hut*$^+$ *galE542*	His$^-$ Tc^r	Nad$^+$, Gal$^+$	R
TL853	F′148	32–34, 42–44	*his*$^+$ *zzf-20*::Tn*10*	*trpA49 pncA15 hisD9953*::Mu dI*1734*	Trp$^-$ PncA$^-$ Km^r Tc^r	His$^+$	R
TL854	F′148	32–34, 42–44	*his*$^+$ *zzf-20*::Tn*10*	*trpA49 pncA15 hisD9953*::Mu dI*1734 zcc-628*::Tn*5*	Trp$^-$ PncA$^-$ Km^r Tc^r	His$^+$	S
TL1600	F′129	44–51	*his*$^+$ *zzf-20*::Tn*10*	*his-2236 proC1909* Mu d1–8 *rpsL1*	His$^+$ Pro$^-$ Sm^r	His$^+$	S
TL1178	F′198	50–56	*cysA*$^+$ *zzf-20*::Tn*10*	*thr-469*::Mu d1–8	Thr$^-$ Ap^r Tc^r		R
TL1179	F′198	50–56	*cysA*$^+$ *zzf-20*::Tn*10*	*thr-469*::Mu d1–8	Thr$^-$ Ap^r Tc^r		S
TL870	F′143	56–62	*cysC*$^+$ *zzf-20*::Tn*10*	*cysC519 rpsL1*	Sm^r Tc^r	Cys$^+$	R
TL871	F′143	56–62	*cysC*$^+$ *zzf-20*::Tn*10*	*cysC519 rpsL1*	Sm^r Tc^r	Cys$^+$	S
TL863	F′116	59–66	*lysA*$^+$ *serA*$^+$ *zzf-20*::Tn*10*	*lys554 serA790 his644*	His$^-$ Tc^r	Lys$^+$, Ser$^+$	R
TL864	F′116	59–66	*lysA*$^+$ *serA*$^+$ *zzf-20*::Tn*10*	*lys554 serA790 his644*	His$^-$ Tc^r	Lys$^+$, Ser$^+$	S
TL860	F′140	67–81	*cysG*$^+$ *argD*$^+$ *zzf-20*::Tn*10*	*argD455* Δ(*proBA*) *rpsL1*	Pro$^-$ Sm^r Tc^r	Arg$^+$	S
TL865	F′117	94–97	*pyrB*$^+$ *zzf-20*::Tn*10*	*pyrB64 hisD6414 mel*	His$^-$ Tc^r	PyrB$^+$	R
TL866	F′117	94–97	*pyrB*$^+$ *zzf-20*::Tn*10*	*pyrB64 hisD6414 mel*	His$^-$ Tc^r	PyrB$^+$	S

[a] F-prime factors carrying *E. coli* genes were obtained from B. Bachmann, Coli Genetic Stock Center, and had been previously transferred in several different laboratories to *S. typhimurium*; most of these strains were described by Sanderson and Hartman (412). D. Sheaks, M. Haskell, and L. Csonka obtained these strains from K. Hughes and J. Roth and constructed most of the strains in the table by transducing Tn*10* or Tn*5* from strains with these transposons at known insertion sites in the F factor in the the F-prime factors; they then confirmed that the antibiotic resistance (Tc^r for Tn*10*, Km^r for Tn*5*) is transferred by conjugation together with the chromosomal genes on the F′ factor. The strain TT1948 was constructed in the laboratory of J. R. Roth and obtained from B. Ames.

[b] These strains are all available from SGSC.

[c] The F-prime factors of *E. coli* were originally described by Low (298). However, the location of the genes carried is given according to edition VII of the *E. coli* map (14) in which the minutes have been modified.

[d] The temperature-sensitive mutation in F′ *ts*114 *lac* (225), which makes the plasmid unable to replicate at high temperature, is linked by P22 transduction to the transposon insertion *zzf-20*::Tn*10* (constructed by Chumley et al. [86]). The new F factors were made by transducing from an F-prime factor carrying both *ts*114 and *zzf-20*::Tn*10* into the *E. coli* F factors. All isolates selected carried the transposon; some are temperature sensitive (carry the *ts* allele), whereas others are temperature resistant. Those which are temperature sensitive must be grown at 30°C to prevent loss of the plasmid or insertion of the plasmid into the chromosome.

of DNA with pBR322 in *S. typhimurium* (J. Binotto, P. R. MacLachlan, and K. E. Sanderson, unpublished data). Unlike $CaCl_2$-dependent transformation, the lipopolysaccharide composition of the strain did not affect electrotransformation frequencies, since smooth and rough strains all gave approximately the same frequencies. The restriction barrier between *E. coli* and *S. typhimurium* dramatically reduces the frequency of $CaCl_2$ transformation of pBR322 between the genera unless a restriction-deficient recipient strain is used (P. R. Machachlan and K. E. Sanderson, unpublished data), but electrotransformation of pBR322 DNA from *E. coli* to *S. typhimurium* was only slightly affected by the restriction barrier, although transformation of other plasmids was reduced 2 to 3 orders of magnitude by the barrier.

Even large plasmids such as cosmids in the 40- to 50-kb range isolated from *E. coli* can be electrotransformed into an *S. typhimurium* line. A cosmid library of ca. 500 clones derived by ligating *S. typhimurium* DNA into pHC79 has been constructed by E. Vimr and is maintained in *E. coli* K-12 cells (E. Vimr, unpublished data). This library is available from the SGSC. DNA from these clones can be transformed directly into *S. typhimurium* to detect plasmids able to complement *Salmonella* mutations.

F-Factor-Mediated Conjugation Methods

The F factor of *E. coli* K-12 has been transmitted into *S. typhimurium* (534) and *S. abony* (305). Hfr strains have been isolated and described (416, 417). These Hfr strains, which have been used to construct the basic linkage maps and to map new genes, are available from the SGSC. Chumley et al. (86) have developed a system which permits the construction of an Hfr strain with an origin at any site of the chromosome at which a Tn*10* insertion has been isolated. This method requires the directed insertion of an F-prime *ts*114 *lac*$^+$ plasmid into the chromosome by homologous recombination between a Tn*10* sequence carried on the plasmid and a second Tn*10* sequence located on the chromosome. Methods for its use have been described (86, 416). A list of *S. typhimurium* strains with transposon insertions, including Tn*10*, has been published (418), and an expanded list is given in Table 3; these can be obtained from the original investigator or from the SGSC.

Methods for F-factor-mediated conjugation were described earlier (412, 416). The frequency of formation of transconjugants in F-factor-mediated conjugation between *S. typhimurium* donor and recipient strains is the same as in crosses within *E. coli* K-12 if two barriers to mating are overcome. The first barrier to conjugation is due to repression of F-factor expression by the plasmid pSLT which is normally resident in all LT2 lines; this barrier reduces the number of cells with F pili and the frequency of transconjugants per donor cell by 100- to 1,000-fold. It is possible to overcome this barrier by using Hfr or F-prime plasmid-containing strains which have lost pSLT or in which the F factor has mutations in the *traO* or *finP* gene, making them insensitive to pSLT repression (415). The second barrier to conjugation is due to the properties of the recipient strain. The O somatic side chains on the lipopolysaccharide of the wild-type (smooth) strains of *S. typhimurium* reduce the frequency of mating aggregation and of transconjugant formation, especially when mating is carried out in broth rather than on a solid surface such as agar or a membrane filter (119, 414). The yield of transconjugants in broth mating is increased 20-fold for rough mutants which have lost the side chains of the lipopolysaccharide. Thus, F-prime factors which are derepressed for F-factor function will be transferred from *S. typhimurium* donor cells to rough recipient cells of *S. typhimurium* at a frequency of 1.0 transconjugant per donor cell (414).

Some F-prime factors carrying *Salmonella* genetic material have been reported (34, 417); these strains are available from the SGSC. However, F-prime factors carrying *S. typhimurium* genes have not yet been isolated for all regions of the map of *S. typhimurium*, and so *S. typhimurium* strains carrying F-prime factors with *E. coli* genes have been used in a variety of studies (412). Low-efficiency transfer of some *E. coli* F-prime factors is due mainly to restriction (*hsd*) barriers. This restriction can be reduced through the use of a recipient strain which is defective in host restriction; strains which are $r^- m^+$ for all three restriction systems, *hsdL*, *hsdSA*, and *hsdSB* (61), can be used as an intermediate recipient in this transfer. These strains, LB5000 and LB5010, can be obtained from the SGSC. Because of the lack of homology between the *E. coli* genes on the F-prime factor and the *S. typhimurium* chromosome, *E. coli* K-12 F-prime factors in *S. typhimurium* undergo relatively little chromosomal insertion and can be maintained as stable heterogenotes in Rec^+ genetic backgrounds. A set of these F-prime factors carrying most of the chromosome of *E. coli* K-12 was reported by Sanderson and Hartman (412), and most of these strains are maintained at the SGSC. More recently, Sheaks et al. (D. Sheaks, M. Haskell, and L. Csonka, personal communication) have modified some F-prime factors carrying *E. coli* genes by transducing transposons (Tn*10* or Tn*5*), which were previously transposed into the F factor, into a series of these F-prime factors in *Salmonella* spp.; this permits the maintenance and transfer of these plasmids by selection for Tc^r or Km^r. These plasmids (Table 4), which cover a large part of the *E. coli* chromosome, were provided by L. Csonka and are available from the SGSC. In addition, R-prime formation through the use of RP4::mini-Mu (500) has been used to isolate plasmids carrying genes of *S. typhimurium* (245).

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LITERATURE CITED

1. **Ahmed, S. A., H. Kawasaki, R. Bauerle, H. Morita, and E. W. Miles.** 1988. Site-directed mutagenesis of the α subunit of tryptophan synthase from *Salmonella typhimurium*. Biochem. Biophys. Res. Commun. **151**:672–678.
2. **Aizawa, S.-I., G. E. Dean, C. J. Jones, R. M. Macnab, and S. Yamaguchi.** 1985. Purification and characterization of the flagellar hook-basal body complex of *Salmonella typhimurium*. J. Bacteriol. **161**:836–849.
3. **Aliabadi, Z., Y. K. Park, J. L. Slonczewski, and J. W. Foster.** 1988. Novel regulatory loci controlling oxygen- and pH-regulated gene expression in *Salmonella typhimurium*. J. Bacteriol. **170**:842–851.
4. **Aliabadi, Z., F. Warren, S. Mya, and J. W. Foster.** 1986. Oxygen-regulated stimulons of *Salmonella typhimurium* identified by Mu d(Ap*lac*) operon fusions. J. Bacteriol. **165**:780–786.
5. **Alvarez-Jacobs, J., M. de la Garza, and M. V. Ortega.** 1986. Biochemical and genetic characterization of L-glutamate transport and utilization in *Salmonella typhimurium* LT-2 mutants. Biochem. Genet. **24**:195–205.
6. **Al-Zarban, S., L. Heffernan, J. Nishitani, L. Ransome, and G. Wilcox.** 1984. Positive control of the L-rhamnose genetic system in *Salmonella typhimurium* LT2. J. Bacteriol. **158**:603–608.
7. **Ames, B. N., T. H. Tsang, M. Buck, and M. F. Christman.** 1983. The leader mRNA of the histidine attenuator region resembles $tRNA^{His}$: possible general regulatory implications. Proc. Natl. Acad. Sci. USA **80**:5240–5242.
8. **Ames, G. F.-L., and K. Nikaido.** 1985. Nitrogen regulation in *Salmonella typhimurium*. Identification of an *ntrC* protein-binding site and definition of a consensus binding sequence. EMBO J. **4**:539–547.
9. **Ames, G. F.-L., K. Nikaido, A. Hobson, and B. Malcolm.** 1985. Overproduction of the membrane-bound components on the histidine permease from *Salmonella typhimurium*: identification of the M protein. Biochimie **67**:149–154.
10. **Antón, D. N.** 1987. Conditional transduction of *Salmonella typhimurium envB* mutations. J. Bacteriol. **169**:1767–1771.
11. **Antón, D. N., A. T. de Micheli, and A. M. Palermo.** 1983. Isolation of round-cell mutants of *Salmonella typhimurium*. Can. J. Microbiol. **29**:170–173.
12. **Antonucci, T. K., and D. L. Oxender.** 1986. The molecular biology of amino-acid transport in bacteria. Adv. Microb. Physiol. **28**:145–180.
13. **Artz, S., D. Holzschu, P. Blum, and R. Shand.** 1983. Use of M13mp phages to study gene regulation, structure and function: cloning and recombinational analysis of genes of the *Salmonella typhimurium* histidine operon. Gene **26**:147–158.
14. **Bachmann, B. J.** 1983. Linkage map of *Escherichia coli* K-12, edition 7. Microbiol. Rev. **47**:180–230.
15. **Baer, M., and S. Altman.** 1985. A catalytic RNA and its gene from *Salmonella typhimurium*. Science **228**:999–1002.
16. **Baird, G. D., E. J. Manning, and P. W. Jones.** 1985. Evidence for related virulence sequences in plasmids of *Salmonella dublin* and *Salmonella typhimurium*. J. Gen. Microbiol. **131**:

1815–1823.
17. **Barbé, J., A. Villaverde, and R. Guerrero.** 1983. Indirect induction of SOS functions in *Salmonella typhimurium*. Antonie van Leeuwenhoek J. Microbiol. Serol. **49**:471–484.
18. **Barcak, G. J., and R. E. Wolf, Jr.** 1988. Comparative nucleotide sequence analysis of growth-rate-regulated *gnd* alleles from natural isolates of *Escherichia coli* and from *Salmonella typhimurium* LT-2. J. Bacteriol. **170**:372–379.
19. **Barker, R. M., and A. A. Yousuf.** 1985. Clonal relationships among naturally occurring nicotinamide-requiring *Salmonella typhimurium*. Genet. Res. **46**:241–250.
20. **Barnes, W. M., and E. Tuley.** 1983. DNA sequence changes of mutations in the histidine operon control region that decrease attenuation. J. Mol. Biol. **165**:443–459.
21. **Barrett, E. L., H. S. Kwan, and J. Macy.** 1984. Anaerobiosis, formate, nitrate, and *pyrA* are involved in the regulation of formate hydrogenlyase in *Salmonella typhimurium*. J. Bacteriol. **158**:972–977.
22. **Barrow, P. A., J. M. Simpson, M. A. Lovell, and M. M. Binns.** 1987. Contribution of *Salmonella gallinarum* large plasmid toward virulence in fowl typhoid. Infect. Immun. **55**:388–392.
23. **Bauer, K., R. Benz, J. Brass, and W. Boos.** 1985. *Salmonella typhimurium* contains an anion-selective outer membrane porin induced by phosphate starvation. J. Bacteriol. **161**:813–816.
24. **Bender, J., and N. Kleckner.** 1986. Genetic evidence that Tn*10* transposes by a non replicative mechanism. Cell **45**:801–815.
25. **Beneski, D. A., A. Nakazawa, N. Weigel, P. E. Hartman, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Isolation and characterization of a phosphocarrier protein HPr from wild type and mutants of *Salmonella typhimurium*. J. Biol. Chem. **257**:14492–14498.
26. **Beninger, P. R., G. Chikami, K. Tanabe, C. Roudier, J. Fierer, and D. G. Guiney.** 1988. Physical and genetic mapping of the *Salmonella dublin* virulence plasmid pSDL2. Relationship to plasmids from other *Salmonella* strains. J. Clin. Invest. **81**: 1341–1347.
27. **Benson, N., P. Sugiono, S. Bass, L. V. Mendelman, and P. Youderian.** 1986. General selection for specific DNA-binding activities. Genetics **114**:1–14.
28. **Berg, C. M., and D. E. Berg.** 1987. Uses of transposable elements and maps of known insertions, p. 1071–1109. *In* F. C. Neidhardt, J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
29. **Berg, C. M., W. A. Whalen, and L. B. Archambault.** 1983. Role of alanine-valine transaminase in *Salmonella typhimurium* and analysis of an *avtA*::Tn*5* mutant. J. Bacteriol. **155**:1009–1014.
30. **Berg, D. E., L. Johnsrud, L. McDivitt, R. Ramabhadran, and B. J. Hirschel.** 1985. Inverted repeats of Tn*5* are transposable elements. Proc. Natl. Acad. Sci. USA **79**:2632–2635.
31. **Berg, D. E., A. Weiss, and L. Crossland.** 1980. Polarity of Tn*5* insertion mutations in *Escherichia coli*. J. Bacteriol. **142**:439–446.
32. **Bergstrom, S., F. P. Lindberg, O. Olsson, and S. Normark.** 1983. Comparison of the overlapping *frd* and *ampC* operons of *Escherichia coli* with the corresponding DNA sequences in other gram-negative bacteria. J. Bacteriol. **155**:1297–1305.
33. **Blazey, D. L., and R. O. Burns.** 1982. Transcriptional activity of the transposable element Tn*10* in the *Salmonella typhimurium ilvGEDA* operon. Proc. Natl. Acad. Sci. USA **79**:5011–5015.
34. **Blazey, D. L., and R. O. Burns.** 1983. *recA*-dependent recombination between rRNA operons generates type II F′ plasmids. J. Bacteriol. **156**:1344–1348.
35. **Blazey, D. L., and R. O. Burns.** 1984. Regulation of *Salmonella typhimurium ilvYC* genes. J. Bacteriol. **159**:951–957.
36. **Bloch, M.-A., and C. Desaymard.** 1985. Antigenic polymorphism of the LamB protein among members of the family *Enterobacteriaceae*. J. Bacteriol. **163**:106–110.
37. **Blum, P., L. Blaha, and S. Artz.** 1986. Reversion and immobilization of phage Mud1 cts (Ap^r *lac*) insertion mutations in *Salmonella typhimurium*. Mol. Gen. Genet. **202**:327–330.
38. **Bochner, B. R., H.-C. Huang, G. L. Schieven, and B. N. Ames.** 1980. Positive selection for loss of tetracycline resistance. J. Bacteriol. **143**:926–933.
39. **Borczuk, A., A. Staub, and J. Stock.** 1986. Demethylation of bacterial chemoreceptors is inhibited by attractant stimuli in the complete absence of the regulatory domain of the demethylating enzyme. Biochem. Biophys. Res. Commun. **141**:918–923.
40. **Borczuk, A., A. Stock, and J. Stock.** 1987. *S*-Adenosylmethionine may not be essential for signal transduction during bacterial chemotaxis. J. Bacteriol. **169**:3295–3300.
41. **Bossi, L.** 1983. The *hisR* locus of *Salmonella*: nucleotide sequence and expression. Mol. Gen. Genet. **192**:163–170.
42. **Bossi, L.** 1983. Context effects: translation of UAG codon by suppressor tRNA is affected by the sequence following UAG in the message. J. Mol. Biol. **164**:73–87.
43. **Bossi, L., and D. M. Smith.** 1984. Suppressor *sufJ*: a novel type of tRNA mutant that induces translational frameshifting. Proc. Natl. Acad. Sci. USA **81**:6105–6109.
44. **Bossi, L., and D. M. Smith.** 1984. Conformational change in the DNA associated with an unusual promoter mutation in a tRNA operon of *Salmonella*. Cell **39**:643–652.
45. **Botsford, J. L.** 1984. Cyclic AMP phosphodiesterase in *Salmonella typhimurium*: characteristics and physiological function. J. Bacteriol. **160**:826–830.
46. **Bouadloun, F., T. Srichaiyo, L. A. Isaksson, and G. R. Björk.** 1986. Influence of modification next to the anticodon in tRNA on codon context sensitivity of translational suppression and accuracy. J. Bacteriol. **166**:1022–1027.
47. **Bouma, C. L., N. D. Meadow, E. W. Stover, and S. Roseman.** 1987. II-B^{Glc}, a glucose receptor of the bacterial phosphotransferase system: molecular cloning of *ptsG* and purification of the receptor from an overproducing strain of *Escherichia coli*. Proc. Natl. Acad. Sci. USA **84**:930–934.
48. **Bower, S. G., B. Hove-Jensen, and R. L. Switzer.** 1988. Structure of the gene encoding phosphoribosylpyrophosphate synthetase (*prsA*) in *Salmonella typhimurium*. J. Bacteriol. **170**:3243–3248.
49. **Brade, H., H. Moll, and E. T. Rietschel.** 1985. Structural investigations on the inner core region of lipopolysaccharides from *Salmonella minnesota* rough mutants. Biomed. Mass. Spectrom. **12**:602–609.
50. **Brade, L., F. E. Nano, S. Schlecht, S. Schramek, and H. Brade.** 1987. Antigenic and immunogenic properties of recombinants from *Salmonella typhimurium* and *Salmonella minnesota* rough mutants expressing in their lipopolysaccharide a genus-specific chlamydial epitope. Infect. Immun. **55**:482–486.
51. **Brady, R. A., and L. N. Csonka.** 1988. Transcriptional regulation of the *proC* gene of *Salmonella typhimurium*. J. Bacteriol. **170**:2379–2382.
52. **Brahmbhatt, H. N., N. B. Quigley, and P. R. Reeves.** 1986. Cloning part of the region encoding biosynthetic enzymes for surface antigen (O-antigen) of *Salmonella typhimurium*. Mol. Gen. Genet. **203**:172–176.
53. **Brahmbhatt, H. N., P. Wyk, N. B. Quigley, and P. R. Reeves.** 1988. Complete physical map of the *rfb* gene cluster encoding biosynthetic enzymes for the O antigen of *Salmonella typhimurium* LT2. J. Bacteriol. **170**:98–102.
54. **Brañes, L. V., and W. W. Kay.** 1983. Lipopolysaccharide core mutants of *Salmonella typhimurium* containing D-*glycero*-D-*manno*-heptose. J. Bacteriol. **154**:1462–1466.
55. **Braun, V., K. Günthner, K. Hantke, and L. Zimmermann.** 1983. Intracellular activation of albomycin in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **156**:308–315.
56. **Brown, D. J., D. S. Munro, and D. J. Platt.** 1986. Recognition of the cryptic plasmid, pSLT, by restriction fingerprinting and a study of its incidence in Scottish salmonella isolates. J. Hyg. **97**:193–197.
57. **Bruist, M. F., A. C. Glasgow, R. C. Johnson, and M. I. Simon.** 1987. Fis binding to the recombinational enhancer of the Hin DNA inversion system. Genes Dev. **1**:762–772.
58. **Bruist, M. F., and M. I. Simon.** 1984. Phase variation and the Hin protein: in vivo activity measurements, protein overpro-

duction, and purification. J. Bacteriol. **159**:71–79.
59. **Bruni, C. B., M. S. Carlomagno, S. Formisano, and G. Paolella.** 1986. Primary and secondary structural homologies between the HIS4 gene product of *Saccharomyces cerevisiae* and the *hisIE* and *hisD* gene products of *Escherichia coli* and *Salmonella typhimurium*. Mol. Gen. Genet. **203**:389–396.
60. **Buck, M., and B. N. Ames.** 1984. A modified nucleotide in tRNA as a possible regulator of aerobiosis: synthesis of *cis*-2-methyl-thioribosylzeatin in the tRNA of *Salmonella*. Cell **36**:523–531.
61. **Bullas, L. R., and J.-I. Ryu.** 1983. *Salmonella typhimurium* LT2 strains which are r^- m^+ for all three chromosomally located systems of DNA restriction and modification. J. Bacteriol. **156**:471–474.
62. **Burns, D. M., and I. R. Beacham.** 1985. Rare codons in *E. coli* and *S. typhimurium* signal sequences. FEBS Lett. **189**:318–324.
63. **Burns, D. M., and I. R. Beacham.** 1986. Identification and sequence analysis of a silent gene (*ush*A^0) in *Salmonella typhimurium*. J. Mol. Biol. **192**:163–175.
64. **Buvinger, W. E., K. A. Lampel, R. J. Bojanowski, and M. Riley.** 1984. Location and analysis of nucleotide sequences at one end of a putative *lac* transposon in the *Escherichia coli* chromosome. J. Bacteriol. **159**:618–623.
65. **Byrne, C. R., R. S. Monroe, K. A. Ward, and N. M. Kredich.** 1988. DNA sequences of the *cysK* regions of *Salmonella typhimurium* and *Escherichia coli* and linkage of the *cysK* regions to *ptsH*. J. Bacteriol. **170**:3150–3157.
66. **Cairney, J., I. R. Booth, and C. F. Higgins.** 1985. Osmoregulation of gene expression in *Salmonella typhimurium*: *proU* encodes an osmotically induced betaine transport system. J. Bacteriol. **164**:1224–1232.
67. **Cairney, J., I. R. Booth, and C. F. Higgins.** 1985. *Salmonella typhimurium proP* gene encodes a transport system for the osmoprotectant betaine. J. Bacteriol. **164**:1218–1223.
68. **Cairney, J., C. F. Higgins, and I. R. Booth.** 1984. Proline uptake through the major transport system of *Salmonella typhimurium* is coupled to sodium ions. J. Bacteriol. **160**:22–27.
69. **Calvin, N. M., and P. C. Hanawalt.** 1988. High-efficiency transformation of bacterial cells by electroporation. J. Bacteriol. **170**:2796–2801.
70. **Capobianco, J. O., R. P. Darveau, R. C. Goldman, P. A. Lartey, and A. G. Pernet.** 1987. Inhibition of exogenous 3-deoxy-D-*manno*-octulosonate incorporation into lipid A precursor of toluene-treated *Salmonella typhimurium* cells. J. Bacteriol. **169**:4030–4035.
71. **Carlomagno, M. S., F. Blasi, and C. B. Bruni.** 1983. Gene organization in the distal part of the *Salmonella typhimurium* histidine operon and determination and sequence of the operon transcription terminator. Mol. Gen. Genet. **191**:413–420.
72. **Carlomagno, M. S., A. Riccio, and C. B. Bruni.** 1985. Convergently functional, rho-independent terminator in *Salmonella typhimurium*. J. Bacteriol. **163**:362–368.
73. **Carter, P. W., J. M. Bartkus, and J. M. Calvo.** 1986. Transcription attenuation in *Salmonella typhimurium*: the significance of rare leucine codons in the *leu* leader. Proc. Natl. Acad. Sci. USA **83**:8127–8131.
74. **Carter, P. W., D. L. Weiss, H. L. Weith, and J. M. Calvo.** 1985. Mutations that convert the four leucine codons of the *Salmonella typhimurium leu* leader to four threonine codons. J. Bacteriol. **162**:943–949.
75. **Carter, T. H., and C. G. Miller.** 1984. Aspartate-specific peptidases in *Salmonella typhimurium*: mutants deficient in peptidase E. J. Bacteriol. **159**:453–459.
76. **Casadaban, M. J., and J. Chou.** 1984. *In vivo* formation of gene fusions encoding hybrid β-galactosidase proteins in one step with a transposable Mu-*lac* transducing phage. Proc. Natl. Acad. Sci. USA **81**:535–539.
77. **Casadaban, M. J., and S. N. Cohen.** 1979. Lactose genes fused to exogenous promoters in one step using a Mu-*lac* bacteriophage: *in vivo* probe for transcriptional control sequences. Proc. Natl. Acad. Sci. USA **76**:4530–4533.
78. **Castilho, B. A., P. Olfson, and M. J. Casadaban.** 1984. Plasmid insertion mutagenesis and *lac* gene fusion with mini-Mu bacteriophage transposons. J. Bacteriol. **158**:488–495.
79. **Chaconas, G., F. J. deBruijn, M. J. Casadaban, J. R. Lupski, T. J. Kwoh, R. M. Harshey, M. S. DuBow, and A. I. Bukhari.** 1981. *In vitro* and *in vivo* manipulations of bacteriophage Mu DNA: cloning of Mu ends and construction of mini-Mu's carrying selectable markers. Gene **13**:37–46.
80. **Chakraborti, A. S., K. Ishidate, W. R. Cook, J. Zrike, and L. I. Rothfield.** 1986. Accumulation of a murein-membrane attachment site fraction when cell division is blocked in *lkyD* and *cha* mutants of *Salmonella typhimurium* and *Escherichia coli*. J. Bacteriol. **168**:1422–1429.
81. **Chiariotti, L., P. Alifano, M. S. Carlomagno, and C. B. Bruni.** 1986. Nucleotide sequence of the *Escherichia coli hisD* gene of the *Escherichia coli* and *Salmonella typhimurium hisIE* region. Mol. Gen. Genet. **203**:382–388.
82. **Chin, A. M., B. U. Feucht, and M. H. Saier, Jr.** 1987. Evidence for regulation of gluconeogenesis by the fructose phosphotransferase system in *Salmonella typhimurium*. J. Bacteriol. **169**:897–899.
83. **Chin, A. M., S. Sutrina, D. A. Feldheim, and M. H. Saier, Jr.** 1987. Genetic expression of enzyme I* activity of the phosphoenolpyruvate:sugar phosphotransferase system in *ptsHI* deletion strains of *Salmonella typhimurium*. J. Bacteriol. **169**:894–896.
84. **Chopra, A. K., C. W. Houston, J. W. Peterson, R. Prasad, and J. J. Mekalanos.** 1987. Cloning and expression of the *Salmonella* enterotoxin gene. J. Bacteriol. **169**:5095–5100.
85. **Christman, M. F., R. W. Morgan, F. S. Jacobson, and B. N. Ames.** 1985. Positive control of a regulon for defenses against oxidative stress and some heat-shock proteins in *Salmonella typhimurium*. Cell **41**:753–762.
86. **Chumley, F. G., R. Menzel, and J. R. Roth.** 1979. Hfr formation directed by Tn*10*. Genetics **91**:639–655.
87. **Ciampi, M. S., and J. R. Roth.** 1988. Polarity effects in the *hisG* gene of *Salmonella* require a site within the coding sequence. Genetics **118**:193–202.
88. **Ciampi, M. S., M. B. Schmid, and J. R. Roth.** 1982. Transposon Tn*10* provides a promoter for transcription of adjacent sequences. Proc. Natl. Acad. Sci. USA **79**:5016–5020.
89. **Clark, M. A., and E. L. Barrett.** 1987. The *phs* gene and hydrogen sulfide production by *Salmonella typhimurium*. J. Bacteriol. **169**:2391–2397.
90. **Clegg, S., B. K. Purcell, and J. Pruckler.** 1987. Characterization of genes encoding type 1 fimbriae of *Klebsiella pneumoniae*, *Salmonella typhimurium*, and *Serratia marcescens*. Infect. Immun. **55**:281–287.
91. **Comai, L., C. Schilling-Cordaro, A. Mergia, and C. M. Houck.** 1983. A new technique for genetic engineering of *Agrobacterium* Ti plasmid. Plasmid **10**:21–30.
92. **Contreras, A., and J. Casadesus.** 1983. Transposition of Tn*10* to the *xyl-mtl* region (minute 78) of the *Salmonella typhimurium* chromosome. Microbiol. Espan. **36**:23–33. (In Spanish; translated by an abstracting service.)
93. **Cook, W. R., T. J. MacAlister, and L. I. Rothfield.** 1986. Compartmentalization of the periplasmic space at division sites in gram-negative bacteria. J. Bacteriol. **168**:1430–1438.
94. **Cookson, B. T., B. M. Olivera, and J. R. Roth.** 1987. Genetic characterization and regulation of the *nadB* locus of *Salmonella typhimurium*. J. Bacteriol. **169**:4285–4293.
95. **Cooper, S.** 1988. Rate and topography of cell wall synthesis during the division cycle of *Salmonella typhimurium*. J. Bacteriol. **170**:422–430.
96. **Corton, J. C., J. E. Ward, Jr., and J. Lutkenhaus.** 1987. Analysis of cell division gene *ftsZ* (*sulB*) from gram-negative and gram-positive bacteria. J. Bacteriol. **169**:1–7.
97. **Cossart, P., E. A. Groisman, M.-C. Serre, M. J. Casadaban, and B. Gicquel-Sanzey.** 1986. *crp* genes of *Shigella flexneri*, *Salmonella typhimurium*, and *Escherichia coli*. J. Bacteriol. **167**:639–646.
98. **Cottam, P. F., N.-B. He, S. W. Hui, and C. Ho.** 1986. Biochemical and morphological properties of membranes of

unsaturated fatty acid auxotrophs of *Salmonella typhimurium*: effects of fluorinated myristic acids. Biochim. Biophys. Acta **862**:413–428.

99. **Creeger, E. S., T. Schulte, and L. I. Rothfield.** 1984. Regulation of membrane glycosyltransferases by the *sfrB* and *rfaH* genes of *Escherichia coli* and *Salmonella typhimurium*. J. Biol. Chem. **259**:3064–3069.
100. **Csonka, L. N.** 1988. Regulation of cytoplasmic proline levels in *Salmonella typhimurium*: effect of osmotic stress on synthesis, degradation, and cellular retention of proline. J. Bacteriol. **170**: 2374–2378.
101. **Dailey, F. E., J. E. Cronan, Jr., and S. R. Maloy.** 1987. Acetohydroxy acid synthase I is required for isoleucine and valine biosynthesis by *Salmonella typhimurium* LT2 during growth on acetate or long-chain fatty acids. J. Bacteriol. **169**: 917–919.
102. **Dang, C. V., M. Niwano, J. Ryu, and B. L. Taylor.** 1986. Inversion of aerotactic response in *Escherichia coli* deficient in *cheB* protein methylesterase. J. Bacteriol. **166**:275–280.
103. **Das, A., J. Urbanowski, H. Weissbach, J. Nestor, and C. Yanofsky.** 1983. In vitro synthesis of the tryptophan operon leader peptides of *Escherichia coli*, *Serratia marcescens*, and *Salmonella typhimurium*. Proc. Natl. Acad. Sci. USA **80**:2879–2883.
104. **Date, T.** 1983. Demonstration by a novel genetic technique that leader peptidase is an essential enzyme of *Escherichia coli*. J. Bacteriol. **154**:76–83.
105. **Dean, G. E., S.-I. Aizawa, and R. M. Macnab.** 1983. *flaAII* (*motC*, *cheV*) of *Salmonella typhimurium* is a structural gene involved in energization and switching of the flagellar motor. J. Bacteriol. **154**:84–91.
106. **Demerec, M., E. A. Adelberg, A. J. Clark, and P. E. Hartman.** 1966. A proposal for a uniform nomenclature in bacterial genetics. Genetics **54**:61–76.
107. **den Blaauwen, J. L., and P. W. Postma.** 1985. Regulation of cyclic AMP synthesis by enzyme III^{Glc} of the phosphoenolpyruvate:sugar phosphotransferase system in *crp* strains of *Salmonella typhimurium*. J. Bacteriol. **164**:477–478.
108. **Dila, D. K., and S. R. Maloy.** 1986. Proline transport in *Salmonella typhimurium*: *putP* permease mutants with altered substrate specificity. J. Bacteriol. **168**:590–594.
109. **Ditta, G., S. Stanfield, D. Corbin, and D. R. Helinski.** 1980. Broad host range DNA cloning system for gram-negative bacteria: construction of a gene bank of *Rhizobium meliloti*. Proc. Natl. Acad. Sci. USA **77**:7347–7351.
110. **Dobrogosz, W. J., G. W. Hall, D. K. Sherba, D. O. Silva, J. G. Harman, and T. Melton.** 1983. Regulatory interactions among the *cya*, *crp* and *pts* gene products in *Salmonella typhimurium*. Mol. Gen. Genet. **192**:477–486.
111. **Dorman, C. J., G. C. Barr, N. Ni Bhriain, and C. F. Higgins.** 1988. DNA supercoiling and the anaerobic and growth phase regulation of *tonB* gene expression. J. Bacteriol. **170**:2816–2826.
112. **Dower, W. J., J. F. Miller, and C. W. Ragsdale.** 1988. High efficiency transformation of *E. coli* by high voltage electroporation. Nucleic Acids Res. **16**:6127–6145.
113. **Dowman, J. E., and G. G. Meynell.** 1970. Pleiotropic effects of derepressed bacterial sex factors on colicinogeny and cell wall structure. Mol. Gen. Genet. **109**:57–68.
114. **Downs, D. M., and J. R. Roth.** 1987. A novel P22 prophage in *Salmonella typhimurium*. Genetics **117**:367–380.
115. **Downs, D., L. Waxman, A. L. Goldberg, and J. Roth.** 1986. Isolation and characterization of *lon* mutants in *Salmonella typhimurium*. J. Bacteriol. **165**:193–197.
116. **Driver, R. P., and R. P. Lawther.** 1985. Restriction endonuclease analysis of the *ilvGEDA* operon of members of the family *Enterobacteriaceae*. J. Bacteriol. **162**:1317–1319.
117. **Droffner, M. L., and N. Yamamoto.** 1983. Anaerobic cultures of *Salmonella typhimurium* do not exhibit inducible proteolytic function of the *recA* gene and *recBC* function. J. Bacteriol. **156**:962–965.
118. **Druger-Liotta, J., V. J. Prange, D. G. Overdier, and L. N. Csonka.** 1986. Selection of mutations that alter the osmotic control of transcription of the *Salmonella typhimurium proU* operon. J. Bacteriol. **169**:2449–2459.
119. **Duke, J., and D. G. Guiney, Jr.** 1983. The role of lipopolysaccharide structure in the recipient cell during plasmid-mediated bacterial conjugation. Plasmid **9**:222–226.
120. **Dunlap, V. J., and L. N. Csonka.** 1985. Osmotic regulation of L-proline transport in *Salmonella typhimurium*. J. Bacteriol. **163**:296–304.
121. **Edwards, M. F., and B. A. D. Stocker.** 1988. Construction of Δ*aroA his* Δ*pur* strains of *Salmonella typhi*. J. Bacteriol. **170**: 3991–3995.
122. **Egner, C., and D. E. Berg.** 1981. Excision of transposon Tn*5* is dependent on the inverted repeats but not on the transposase function of Tn*5*. Proc. Natl. Acad. Sci. USA **78**:459–463.

122a. **Elliott, T., and J. R. Roth.** 1988. Characterization of Tn*10*d CAM: a transposition-defective Tn*10* specifying chloramphenicol resistance. Mol. Gen. Genet. **213**:332–338.

123. **Engstrom, J., A. Wong, and R. Maurer.** 1986. Interaction of DNA polymerase III γ and β subunits *in vivo* in *Salmonella typhimurium*. Genetics **113**:499–515.
124. **Enomoto, M., and B. A. D. Stocker.** 1974. Transduction by phage P1*kc* in *Salmonella typhimurium*. Virology. **60**:503–514.
125. **Erickson, B. D., Z. F. Burton, K. K. Watanabe, and R. R. Burgess.** 1985. Nucleotide sequence of the *rpsU-dnaG-rpoD* operon from *Salmonella typhimurium* and a comparison of this sequence with the homologous operon of *Escherichia coli*. Gene **40**:67–78.
126. **Erickson, J. U., and G. R. Björk.** 1986. Pleiotropic effects induced by modification deficiency next to the anticodon of tRNA from *Salmonella typhimurium* LT2. J. Bacteriol. **166**: 1013–1021.
127. **Esaki, N., and C. T. Walsh.** 1986. Biosynthetic alanine racemase of *Salmonella typhimurium*: purification and characterization of the enzyme encoded by the *alr* gene. Biochemistry **25**:3261–3267.
128. **Escalante-Semerena, J. C., and J. R. Roth.** 1987. Regulation of cobalamin biosynthetic operons in *Salmonella typhimurium*. J. Bacteriol. **169**:2251–2258.
129. **Faraci, W. S., and C. T. Walsh.** 1988. Racemization of alanine by the alanine racemases from *Salmonella typhimurium* and *Bacillus stearothermophilus*: energetic reaction profiles. Biochemistry **27**:3267–3276.
130. **Feutrier, J., W. W. Kay, and T. J. Trust.** 1986. Purification and characterization of fimbriae from *Salmonella enteritidis*. J. Bacteriol. **168**:221–227.
131. **Fields, P. I., R. V. Swanson, C. G. Haidaris, and F. Heffron.** 1986. Mutants of *Salmonella typhimurium* that cannot survive within the macrophage are avirulent. Proc. Natl. Acad. Sci. USA **83**:5189–5193.
132. **Finnegan, J., and D. Sherratt.** 1982. Plasmid ColE1 conjugal mobility: the nature of *bom*, a region required in *cis* for transfer. Mol. Gen. Genet. **185**:344–351.
133. **Foltermann, K. F., D. A. Beck, and J. R. Wild.** 1986. In vivo formation of hybrid aspartate transcarbamoylases from native subunits of divergent members of the family *Enterobacteriaceae*. J. Bacteriol. **167**:285–290.
134. **Foster, D. L., S. L. Mowbray, B. K. Jap, and D. E. Koshland, Jr.** 1985. Purification and characterization of the aspartate chemoreceptor. J. Biol. Chem. **260**:11706–11710.
135. **Foster, J. W.** 1983. Identification and characterization of a *relA*-dependent starvation-inducible locus (*sin*) in *Salmonella typhimurium*. J. Bacteriol. **156**:424–428.
136. **Foster, J. W., E. A. Holley, and S. Mya.** 1984. NAD metabolism in *Salmonella typhimurium*: isolation of pyridine analogue supersensitive (*pas*) and *pas* suppressor mutants. J. Gen. Microbiol. **130**:2873–2881.
137. **Foster, J. W., E. A. Holley-Guthrie, and F. Warren.** 1987. Regulation of NAD metabolism in *Salmonella typhimurium*: genetic analysis and cloning of the *nadR* repressor locus. Mol. Gen. Genet. **208**:279–287.
138. **Foster, J. W., and M. P. Spector.** 1986. Phosphate starvation regulon of *Salmonella typhimurium*. J. Bacteriol. **166**:666–669.
139. **French, S., K. Martin, T. Patterson, R. Bauerle, and O. L.**

Miller, Jr. 1985. Electron microscopic visualization of *trp* operon expression in *Salmonella typhimurium*. Proc. Natl. Acad. Sci. USA **82**:4638–4642.
140. **Freudl, R., and S. T. Cole.** 1983. Cloning and molecular characterization of the *ompA* gene from *Salmonella typhimurium*. Eur. J. Biochem. **134**:497–502.
141. **Frey, B., J. McCloskey, W. Kersten, and H. Kersten.** 1988. New function of vitamin B_{12}: cobamide-dependent reduction of epoxyqueuosine to queuosine in tRNAs of *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **170**:2078–2082.
142. **Friedberg, D., E. R. Rosenthal, J. W. Jones, and J. M. Calvo.** 1985. Characterization of the 3′ end of the leucine operon of *Salmonella typhimurium*. Mol. Gen. Genet. **199**:486–494.
143. **Fujita, H., S. Yamaguchi, T. Taira, T. Hirano, and T. Iino.** 1987. Isolation and genetic analysis of operator-constitutive mutants of the *H1* operon in *Salmonella typhimurium*. J. Gen. Microbiol. **133**:3071–3080.
144. **Gaitanaris, G. A., M. McCormick, B. H. Howard, and M. E. Gottesman.** 1986. Reconstitution of an operon from overlapping fragments: use of the λSV2 integrative cloning system. Gene **46**:1–11.
145. **Galakatos, N. G., E. Daub, D. Botstein, and C. T. Walsh.** 1986. Biosynthetic *alr* alanine racemase from *Salmonella typhimurium*: DNA and protein sequence determination. Biochemistry **25**:3255–3260.
146. **Galakatos, N. G., and C. T. Walsh.** 1987. Specific proteolysis of native alanine racemases from *Salmonella typhimurium*: identification of the cleavage site and characterization of the clipped two-domain proteins. Biochemistry **26**:8475–8480.
147. **Gardner, M. M., D. O. Hennig, and R. A. Kelln.** 1983. Control of *arg* gene expression in *Salmonella typhimurium* by the arginine repressor from *Escherichia coli* K-12. Mol. Gen. Genet. **189**:458–462.
148. **Geerse, R. H., C. R. J. Ruig, A. R. J. Schuitema, and P. W. Postma.** 1986. Relationship between pseudo-HPr and the PEP: fructose phosphotransferase system in *Salmonella typhimurium* and *Escherichia coli*. Mol. Gen. Genet. **203**:435–444.
149. **Gemmill, R. M., J. W. Jones, G. W. Haughn, and J. M. Calvo.** 1983. Transcription initiation sites of the leucine operons of *Salmonella typhimurium* and *Escherichia coli*. J. Mol. Biol. **170**:39–59.
150. **Gemmill, R. M., M. Tripp, S. B. Friedman, and J. M. Calvo.** 1984. Promoter mutation causing catabolite repression of the *Salmonella typhimurium* leucine operon. J. Bacteriol. **158**:948–953.
151. **Ghangas, G. S., and D. B. Wilson.** 1984. Isolation and characterization of the *Salmonella typhimurium* LT2 xylose regulon. J. Bacteriol. **157**:158–164.
152. **Gibson, M. M., E. M. Ellis, K. A. Graeme-Cook, and C. F. Higgins.** 1987. OmpR and EnvZ are pleiotropic regulatory proteins: positive regulation of the tripeptide permease (*tppB*) of *Salmonella typhimurium*. Mol. Gen. Genet. **207**:120–129.
153. **Gibson, M. M., M. Price, and C. F. Higgins.** 1984. Genetic characterization and molecular cloning of the tripeptide permease (*tpp*) genes of *Salmonella typhimurium*. J. Bacteriol. **160**:122–130.
154. **Gilson, E., C. F. Higgins, M. Hofnung, G. Ferro-Luzzi Ames, and H. Nikaido.** 1982. Extensive homology between membrane-associated components of histidine and maltose transport systems of *Salmonella typhimurium* and *Escherichia coli*. J. Biol. Chem. **257**:9915–9918.
155. **Goldman, R., W. Kohlbrenner, P. Lartey, and A. Pernet.** 1987. Antibacterial agents specifically inhibiting lipopolysaccharide synthesis. Nature (London) **329**:162–164.
156. **Goldman, R. C., and E. M. Devine.** 1987. Isolation of *Salmonella typhimurium* strains that utilize exogenous 3-deoxy-D-*manno*-octulosonate for synthesis of lipopolysaocharide. J. Bacteriol. **169**:5060–5065.
157. **Goldman, R. C., C. C. Doran, and J. O. Capobianco.** 1988. Analysis of lipopolysaccharide biosynthesis in *Salmonella typhimurium* and *Escherichia coli* by using agents which specifically block incorporation of 3-deoxy-D-*manno*-octulosonate. J. Bacteriol. **170**:2185–2191.
158. **Goldrick, D., G.-Q. Yu, S.-Q. Jiang, and J.-S. Hong.** 1988. Nucleotide sequence and transcription start point of the phosphoglycerate transporter gene of *Salmonella typhimurium*. J. Bacteriol. **170**:3421–3426.
159. **Gómez-Eichelmann, M. C., and H. K. Torres.** 1983. Stability of plasmids R1-19 and R100 in hyper-recombinant *Escherichia coli* strains and in *Salmonella typhimurium* strains. J. Bacteriol. **154**:1493–1497.
160. **Goodell, E. W., and C. F. Higgins.** 1987. Uptake of cell wall peptides by *Salmonella typhimurium* and *Escherichia coli*. J. Bacteriol. **169**:3861–3865.
161. **Grenier, F. C., E. B. Waygood, and M. H. Saier, Jr.** 1986. The bacterial phosphotransferase system: kinetic characterization of the glucose, mannitol, glucitol, and N-acetylglucosamine systems. J. Cell. Biochem. **31**:97–105.
162. **Grill, H., N. Weigel, B. J. Gaffney, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Radioactive and electron paramagnetic resonance labelling of the *Salmonella typhimurium* phosphocarrier protein (HPr) at the NH_2-terminal methionine. J. Biol. Chem. **257**:24510–24517.
163. **Groisman, E. A., and M. J. Casadaban.** 1987. Cloning of genes from members of the family *Enterobacteriaceae* with mini-Mu bacteriophage containing plasmid replicons. J. Bacteriol. **169**:687–693.
164. **Grossman, N., M. A. Schmetz, J. Foulds, E. N. Klima, V. Jiminez, L. L. Leive, and K. A. Joiner.** 1987. Lipopolysaccharide size and distribution determine serum resistance in *Salmonella montevideo*. J. Bacteriol. **169**:856–863.
165. **Guerrero, R., and J. Barbé.** 1982. Expression of *recA*-gene dependent SOS functions in *Salmonella typhimurium*. Antonie van Leeuwenhoek J. Microbiol. Serol. **48**:159–167.
166. **Gulig, P. A., and R. Curtiss, III.** 1987. Plasmid-associated virulence of *Salmonella typhimurium*. Infect. Immun. **55**:2891–2901.
167. **Gutterson, N. I., and D. E. Koshland, Jr.** 1983. Replacement and amplification of bacterial genes with sequences altered *in vitro*. Proc. Natl. Acad. Sci. USA **80**:4894–4898.
168. **Haber, L. T., P. P. Pang, D. I. Sobell, J. A. Mankovich, and G. C. Walker.** 1988. Nucleotide sequence of the *Salmonella typhimurium mutS* gene required for mismatch repair: homology of MutS and HexA of *Streptococcus pneumoniae*. J. Bacteriol. **170**:197–202.
169. **Hackett, J., I. Kotlarski, V. Mathan, K. Francki, and D. Rowley.** 1986. The colonization of Peyer's patches by a strain of *Salmonella typhimurium* cured of the cryptic plasmid. J. Infect. Dis. **153**:1119–1125.
170. **Hackett, J., P. Wyk, P. Reeves, and V. Mathan.** 1987. Mediation of serum resistance in *Salmonella typhimurium* by an 11-kilodalton polypeptide encoded by the cryptic plasmid. J. Infect. Dis. **155**:540–549.
171. **Hahn, D. R., and S. R. Maloy.** 1986. Regulation of the *put* operon in *Salmonella typhimurium*: characterization of promoter and operator mutations. Genetics **114**:687–703.
172. **Hammond, S. M., A. Claesson, A. M. Jansson, L.-G. Larsson, B. G. Pring, C. M. Town, and B. Ekström.** 1987. A new class of synthetic antibacterials acting on lipopolysaccharide biosynthesis. Nature (London) **327**:730–732.
173. **Hanau, R., R. K. Koduri, N. Ho, and J. E. Brenchley.** 1983. Nucleotide sequence of the control regions for the *glnA* and *glnL* genes of *Salmonella typhimurium*. J. Bacteriol. **155**:82–89.
174. **Hansen-Hagge, T., V. Lehmann, U. Seydel, B. Lindner, and U. Zähringer.** 1985. Isolation and structural analysis of two lipid A precursors from a KDO deficient mutant of *Salmonella typhimurium* differing in their hexadecanoic acid content. Arch. Microbiol. **141**:353–358.
175. **Harkki, A., H. Karkku, and E. T. Palva.** 1987. Use of λ vehicles to isolate *ompC-lacZ* gene fusions in *Salmonella typhimurium* LT2. Mol. Gen. Genet. **209**:607–611.
176. **Harkki, A., and E. T. Palva.** 1984. Application of phage lambda technology to *Salmonella typhimurium*. Construction of a lambda-sensitive *Salmonella* strain. Mol. Gen. Genet. **195**:256–259.

177. **Harms, E., J.-H. Hsu, C. S. Subrahmanyam, and H. E. Umbarger.** 1985. Comparison of the regulatory regions of *ilvGEDA* operons from several enteric organisms. J. Bacteriol. **164**:207–216.
178. **Haughn, G. W., S. R. Wessler, R. M. Gemmill, and J. M. Calvo.** 1986. High A+T content conserved in DNA sequences upstream of *leuABCD* in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **166**:1113–1117.
179. **Hefferman, E. J., J. Fierer, G. Chikami, and D. Guiney.** 1987. Natural history of oral *Salmonella dublin* infection in BALB/c mice: effect of an 80-kilobase-pair plasmid on virulence. J. Infect. Dis. **155**:1254–1259.
180. **Helmann, J. D., and M. J. Chamberlin.** 1987. DNA sequence analysis suggests that expression of flagellar and chemotaxis genes in *Escherichia coli* and *Salmonella typhimurium* is controlled by an alternative σ factor. Proc. Natl. Acad. Sci. USA **84**:6422–6424.
181. **Helmuth, R., R. Stephan, C. Bunge, B. Hoog, A. Steinbeck, and E. Bulling.** 1985. Epidemiology of virulence-associated plasmids and outer membrane protein patterns within seven common *Salmonella* serotypes. Infect. Immun. **48**:175–182.
182. **Hengge, R., and W. Boos.** 1985. Defective secretion of maltose- and ribose-binding proteins caused by a truncated periplasmic protein in *Escherichia coli*. J. Bacteriol. **162**:972–978.
183. **Hengge, R., T. J. Larson, and W. Boos.** 1983. *sn*-Glycerol-3-phosphate transport in *Salmonella typhimurium*. J. Bacteriol. **155**:186–195.
184. **Herrera, G., A. Urios, V. Aleixandre, and M. Blanco.** 1988. UV light-induced mutability in *Salmonella* strains containing the *umuDC* or the *mucAB* operon: evidence for a *umuC* function. Mutat. Res. **198**:9–13.
185. **Higgins, C. F., C. J. Dorman, D. A. Stirling, L. Waddell, I. R. Booth, G. May, and E. Bremer.** 1988. A physiological role for DNA supercoiling in the osmotic regulation of gene expression in *S. typhimurium* and *E. coli*. Cell **52**:569–584.
186. **Higgins, C. F., and M. M. Gibson.** 1984. Peptide transport in bacteria. Methods Enzymol. **125**:365–377.
187. **Higgins, C. F., and M. M. Hardie.** 1983. Periplasmic protein associated with the oligopeptide permeases of *Salmonella typhimurium* and *Escherichia coli*. J. Bacteriol. **155**:1434–1438.
188. **Higgins, C. F., L. Sutherland, J. Cairney, and I. R. Booth.** 1987. The osmotically regulated *proU* locus of *Salmonella typhimurium* encodes a periplasmic betaine-binding protein. J. Gen. Microbiol. **133**:305–310.
189. **Hildenbrand, K., L. Brand, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Nanosecond fluorescence studies of the phosphocarrier protein (HPr) labelled at the NH_2-terminal methionine. J. Biol. Chem. **257**:14518–14525.
190. **Hiles, I. D., M. P. Gallagher, D. J. Jamieson, and C. F. Higgins.** 1987. Molecular characterization of the oligopeptide permease of *Salmonella typhimurium*. J. Mol. Biol. **195**:125–142.
191. **Hiles, I. D., and C. F. Higgins.** 1986. Peptide uptake by *Salmonella typhimurium*. The periplasmic oligopeptide-binding protein. Eur. J. Biochem. **158**:561–567.
192. **Hiles, I. D., L. M. Powell, and C. F. Higgins.** 1987. Peptide transport in *Salmonella typhimurium*: molecular cloning and characterization of the oligopeptide permease genes. Mol. Gen. Genet. **206**:101–109.
193. **Hill-Chappell, J. M., M. P. Spector, and J. W. Foster.** 1986. The pyridine nucleotide cycle of *Salmonella typhimurium*: genetic characterization of the *pncXA* operon. Mol. Gen. Genet. **205**:507–514.
194. **Hmiel, S. P., M. D. Snavely, C. G. Miller, and M. E. Maguire.** 1986. Magnesium transport in *Salmonella typhimurium*: characterization of magnesium influx and cloning of a transport gene. J. Bacteriol. **168**:1444–1450.
195. **Hoffman, G. R., M. J. Walkowicz, J. M. Mason, and J. F. Atkins.** 1983. Genetic instability associated with the *aroC321* allele in *Salmonella typhimurium* involves genetic duplication. Mol. Gen. Genet. **190**:183–188.
196. **Hoiseth, S. K., and B. A. D. Stocker.** 1985. Genes *aroA* and *serC* of *Salmonella typhimurium* constitute an operon. J. Bacteriol. **163**:355–361.
197. **Holley, E. A., M. P. Spector, and J. W. Foster.** 1985. Regulation of NAD biosynthesis in *Salmonella typhimurium*: expression of *nad-lac* gene fusions and identification of a *nad* regulatory locus. J. Gen. Microbiol. **131**:2759–2770.
198. **Homma, M., H. Fujita, S. Yamaguchi, and T. Iino.** 1984. Excretion of unassembled flagellin by *Salmonella typhimurium* mutants deficient in hook-associated proteins. J. Bacteriol. **159**:1056–1059.
199. **Homma, M., H. Fujita, S. Yamaguchi, and T. Iino.** 1987. Regions of *Salmonella typhimurium* flagellin essential for its polymerization and excretion. J. Bacteriol. **169**:291–296.
200. **Homma, M., and T. Iino.** 1985. Locations of hook-associated proteins in flagellar structures of *Salmonella typhimurium*. J. Bacteriol. **162**:183–189.
201. **Homma, M., and T. Iino.** 1985. Excretion of unassembled hook-associated proteins by *Salmonella typhimurium*. J. Bacteriol. **164**:1370–1372.
202. **Homma, M., T. Iino, K. Kutsukake, and S. Yamaguchi.** 1986. *In vitro* reconstitution of flagellar filaments onto hooks of filamentless mutants of *Salmonella typhimurium* by addition of hook-associated proteins. Proc. Natl. Acad. Sci. USA **83**:6169–6173.
203. **Homma, M., T. Iino, and R. M. Macnab.** 1988. Identification and characterization of the products of six region III flagellar genes (*flaAII.3* through *flaQII*) of *Salmonella typhimurium*. J. Bacteriol. **170**:2221–2228.
204. **Homma, M., Y. Komeda, T. Iino, and R. M. Macnab.** 1987. The *flaFIX* gene product of *Salmonella typhimurium* is a flagellar basal body component with a signal peptide for export. J. Bacteriol. **169**:1493–1498.
205. **Homma, M., K. Kutsukake, and T. Iino.** 1985. Structural genes for flagellar hook-associated proteins in *Salmonella typhimurium*. J. Bacteriol. **163**:464–471.
206. **Homma, M., K. Kutsukake, T. Iino, and S. Yamaguchi.** 1984. Hook-associated proteins essential for flagellar filament formation in *Salmonella typhimurium*. J. Bacteriol. **157**:100–108.
207. **Homma, M., K. Ohnishi, T. Iino, and R. M. Macnab.** 1987. Identification of flagellar hook and basal body gene products (FlaFV, FlaFVI, FlaFVII, and FlaFVIII) in *Salmonella typhimurium*. J. Bacteriol. **169**:3617–3624.
208. **Hong, J.-S., and B. N. Ames.** 1971. Localized mutagenesis of any specific small region of the bacterial chromosome. Proc. Natl. Acad. Sci. USA **68**:3158–3162.
209. **Horowitz, H., J. Van Arsdell, and T. Platt.** 1983. Nucleotide sequence of the *trpD* and *trpC* genes of *Salmonella typhimurium*. J. Mol. Biol. **169**:775–797.
210. **Huffman, G. A., and R. H. Rownd.** 1984. Transition of deletion mutants of the composite resistance plasmid NR1 in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **159**:488–498.
211. **Hughes, D.** 1986. The isolation and mapping of EF-Tu mutations in *Salmonella typhimurium*. Mol. Gen. Genet. **202**:108–111.
212. **Hughes, D.** 1987. Mutant forms of *tufA* and *tufB* independently suppress nonsense mutations. J. Mol. Biol. **197**:611–615.
213. **Hughes, D., J. F. Atkins, and S. Thompson.** 1987. Mutants of elongation factor Tu promote ribosomal frameshifting and nonsense readthrough. EMBO J. **6**:4235–4239.
214. **Hughes, K. T., B. T. Cookson, D. Ladika, B. M. Olivera, and J. R. Roth.** 1983. 6-Aminonicotinamide-resistant mutants of *Salmonella typhimurium*. J. Bacteriol. **154**:1126–1136.
215. **Hughes, K. T., D. Ladika, J. R. Roth, and B. M. Olivera.** 1983. An indispensable gene for NAD biosynthesis in *Salmonella typhimurium*. J. Bacteriol. **155**:213–221.
216. **Hughes, K. T., B. M. Olivera, and J. R. Roth.** 1987. Rec dependence of Mu transposition from P22-transduced fragments. J. Bacteriol. **169**:403–409.
217. **Hughes, K. T., B. M. Olivera, and J. R. Roth.** 1988. Structural gene for NAD synthetase in *Salmonella typhimurium*. J. Bacteriol. **170**:2113–2120.
218. **Hughes, K. T., and J. R. Roth.** 1984. Conditionally transposition-defective derivative of Mu d1(Amp Lac). J. Bacteriol. **159**:

130–137.
219. **Hughes, K. T., and J. R. Roth.** 1985. Directed formation of deletions and duplications using Mud(Ap, *lac*). Genetics **109:** 263–282.
220. **Hughes, K. T., and J. R. Roth.** 1988. Transitory *cis* complementation: a method for providing transposition functions to defective transposons. Genetics **119:**9–12.
221. **Hulanicka, M. D., C. Garrett, G. Jagura-Burdzy, and N. M. Kredich.** 1986. Cloning and characterization of the *cysAMK* region of *Salmonella typhimurium*. J. Bacteriol. **168:**322–327.
222. **Iino, T., Y. Komeda, K. Kutsukake, R. Macnab, P. Matsumura, J. S. Parkinson, M. I. Simon, and S. Yamaguchi.** 1988. New unified nomenclature for the flagellar genes of *Escherichia coli* and *Salmonella typhimurium*. Microbiol. Rev. **52:** 533–535.
223. **Ikeda, T., R. Kamiya, and S. Yamaguchi.** 1984. In vitro polymerization of flagellin excreted by a short-flagellum *Salmonella typhimurium* mutant. J. Bacteriol. **159:**787–789.
224. **Ishiguro, E. E., D. Vanderwel, and W. Kusser.** 1986. Control of lipopolysaccharide biosynthesis and release by *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **168:**328–333.
225. **Jacob, F., S. Brenner, and F. Cuzin.** 1963. On the regulation of DNA synthesis in bacteria. Cold Spring Harbor Symp. Quant. Biol. **28:**329–348.
226. **Jagura-Burdzy, G., and D. Hulanicka.** 1987. Cloning of *cysB* mutant alleles of *S. typhimurium*. Acta Biochim. Pol. **34:**35–44.
227. **Jagura-Burdzy, G., and N. M. Kredich.** 1983. Cloning and physical mapping of the *cysB* region of *Salmonella typhimurium*. J. Bacteriol. **155:**578–585.
228. **Jamieson, D. J., and C. F. Higgins.** 1984. Anaerobic and leucine-dependent expression of a peptide transport gene in *Salmonella typhimurium*. J. Bacteriol. **160:**131–136.
229. **Jamieson, D. J., and C. F. Higgins.** 1986. Two genetically distinct pathways for transcriptional regulation of anaerobic gene expression in *Salmonella typhimurium*. J. Bacteriol. **168:** 389–397.
230. **Jamieson, D. J., R. G. Sawers, P. A. Rugman, D. H. Boxer, and C. F. Higgins.** 1986. Effects of anaerobic regulatory mutations and catabolite repression on regulation of hydrogen metabolism and hydrogenase isoenzyme composition in *Salmonella typhimurium*. J. Bacteriol. **168:**405–411.
231. **Janson, C. A., P. S. Kayne, R. J. Almassy, M. Grunstein, and D. Eisenberg.** 1986. Sequence of glutamine synthetase from *Salmonella typhimurium* and implications for the protein structure. Gene **46:**297–300.
232. **Jenness, D. D., and H. K. Schachman.** 1983. Genetic characterization of the folding domains of the catalytic chains in aspartate transcarbamoylase. J. Biol. Chem. **258:**3266–3279.
233. **Jensen, K. F., R. Fast, O. Karlström, and J. N. Larsen.** 1986. Association of RNA polymerase having increased K_m for ATP and UTP with hyperexpression of the *pyrB* and *pyrE* genes of *Salmonella typhimurium*. J. Bacteriol. **166:**857–865.
234. **Jeter, R. M., B. M. Olivera, and J. R. Roth.** 1984. *Salmonella typhimurium* synthesizes cobalamin (vitamin B_{12}) de novo under anaerobic growth conditions. J. Bacteriol. **159:**206–213.
235. **Jeter, R. M., and J. R. Roth.** 1987. Cobalamin (vitamin B_{12}) biosynthetic genes of *Salmonella typhimurium*. J. Bacteriol. **169:**3189–3198.
236. **Jiang, S.-Q., G.-Q. Yu, Z.-G. Li, and J.-S. Hong.** 1988. Genetic evidence for modulation of the activator by two regulatory proteins involved in the exogenous induction of phosphoglycerate transport in *Salmonella typhimurium*. J. Bacteriol. **170:** 4304–4308.
237. **Jochimsen, B. U., B. Hove-Jensen, B. B. Garber, and J. S. Gots.** 1985. Characterization of a *Salmonella typhimurium* mutant defective in phosphoribosylpyrophosphate synthetase. J. Gen. Microbiol. **131:**245–252.
238. **Jones, G. W., D. K. Rabert, D. M. Svinarich, and H. J. Whitfield.** 1982. Association of adhesive, invasive, and virulent phenotypes of *Salmonella typhimurium* with autonomous 60-megadalton plasmids. Infect. Immun. **38:**476–486.
239. **Jordan, E., H. Saedler, and P. Starlinger.** 1968. O^0 and strong-polar mutations in the *gal* operon are insertions. Mol. Gen. Genet. **102:**353–363.
240. **Jovanovich, S. B.** 1985. Regulation of a *cya-lac* fusion by cyclic AMP in *Salmonella typhimurium*. J. Bacteriol. **161:**641–649.
241. **Jovanovich, S. B., and J. Lebowitz.** 1987. Estimation of the effect of coumermycin A_1 on *Salmonella typhimurium* promoters by using random operon fusions. J. Bacteriol. **169:**4431–4435.
242. **Jovanovich, S. B., M. Martinell, M. T. Record, Jr., and R. R. Burgess.** 1988. Rapid response to osmotic upshift by osmoregulated genes in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **170:**534–539.
243. **Joys, T. M.** 1985. The covalent structure of the phase-1 flagellar filament protein of *Salmonella typhimurium* and its comparison with other flagellins. J. Biol. Chem. **260:**15758–15761.
244. **Kadam, S. K., M. S. Peppler, and K. E. Sanderson.** 1985. Temperature-sensitive mutants in *rfaI* and *rfaJ*, genes for galactosyltransferase I and glucosyltransferase II, for synthesis of lipopolysaccharide in *Salmonella typhimurium*. Can. J. Microbiol. **31:**861–869.
245. **Kadam, S. K., A. Rehemtulla, and K. E. Sanderson.** 1985. Cloning of *rfaG*, *B*, *I*, and *J* genes for glycosyltransferase enzymes for synthesis of the lipopolysaccharide core of *Salmonella typhimurium*. J. Bacteriol. **161:**277–284.
246. **Kagawa, H., T. Nishiyama, and S. Yamaguchi.** 1983. Motility development of *Salmonella typhimurium* cells with *flaV* mutations after addition of exogenous flagellin. J. Bacteriol. **155:** 435–437.
247. **Kagawa, H., N. Ono, M. Enomoto, and Y. Komeda.** 1984. Bacteriophage Chi sensitivity and motility of *Escherichia coli* K-12 and *Salmonella typhimurium* Fla^- mutants possessing the hook structure. J. Bacteriol. **157:**649–654.
248. **Kaplan, J. B., W. K. Merkel, and B. P. Nichols.** 1985. Evolution of glutamine amidotransferase genes. Nucleotide sequences of the *pabA* genes from *Salmonella typhimurium*, *Klebsiella aerogenes* and *Serratia marcescens*. J. Mol. Biol. **183:**327–340.
249. **Kelln, R. A.** 1984. Evidence for involvement of $pyrH^+$ of an *Escherichia coli* K-12 F-prime factor in inhibiting construction of hybrid merodiploids with *Salmonella typhimurium*. Can. J. Microbiol. **30:**991–996.
250. **Kelln, R. A., J. Neuhard, and L. Stauning.** 1985. Isolation and characterization of pyrimidine mutants of *Salmonella typhimurium* altered in expression of *pyrC*, *pyrD*, and *pyrE*. Can. J. Microbiol. **31:**981–987.
251. **Kemper, J.** 1974. Gene order and co-transduction in the *leu-ara-fol-pyrA* region of the *Salmonella typhimurium* linkage map. J. Bacteriol. **117:**94–99.
252. **Kendrick, K. E., and W. S. Reznikoff.** 1988. Transposition of $IS50_L$ activates gene expression. J. Bacteriol. **170:**1965–1968.
253. **Kingsbury, D. T., and D. R. Helinski.** 1970. DNA polymerase as a requirement for the maintenance of the bacterial plasmid colicinogenic factor E_1. Biochem. Biophys. Res. Commun. **41:** 1538–1544.
254. **Kinney, D. M., and J. W. Foster.** 1985. Identification of a *cis*-acting regulatory region in the *pncB* locus of *Salmonella typhimurium*. Mol. Gen. Genet. **199:**512–517.
255. **Kobori, J. A., and A. Kornberg.** 1982. The *Escherichia coli* *dnaC* gene product. I. Overproduction of the *dnaC* proteins of *Escherichia coli* and *Salmonella typhimurium* by cloning into a high copy number plasmid. J. Biol. Chem. **257:**13757–13762.
256. **Kohno, T., L. Bossi, and J. R. Roth.** 1983. New suppressors of frameshift mutations in *Salmonella typhimurium*. Genetics **103:**23–29.
257. **Kohno, T., L. Bossi, and J. R. Roth.** 1983. Genetic characterization of the *sufJ* frameshift suppressor in *Salmonella typhimurium*. Genetics **103:**31–42.
258. **Krajewska-Grynkiewicz, K., and S. Kustu.** 1983. Regulation of transcription of *glnA*, the structural gene encoding glutamine synthetase, in *glnA*::Mud1(Ap^R, *lac*) fusion strains of *Salmonella typhimurium*. Mol. Gen. Genet. **192:**187–197.
259. **Krajewska-Grynkiewicz, K., and S. Kustu.** 1984. Evidence that nitrogen regulatory gene *ntrC* of *Salmonella typhimurium* is

transcribed from the *glnA* promoter as well as from a separate *ntr* promoter. Mol. Gen. Genet. **193**:135–142.
260. **Kramer, G. F., and B. N. Ames.** 1988. Isolation and characterization of a selenium metabolism mutant of *Salmonella typhimurium*. J. Bacteriol. **170**:736–743.
261. **Kramer, G. F., J. C. Baker, and B. N. Ames.** 1988. Near-UV stress in *Salmonella typhimurium*: 4-thiouridine in tRNA, ppGpp, and ApppGpp as components of an adaptive response. J. Bacteriol. **170**:2344–2351.
262. **Krüger, D. H., S. Hansen, and M. Reuter.** 1983. The *ocr*$^+$ gene function of bacteriophages T3 and T7 counteracts the *Salmonella typhimurium* DNA restriction systems SA and SB. J. Virol. **45**:1147–1149.
263. **Kuchino, Y., Y. Yabusaki, F. Mori, and S. Nishimura.** 1984. Nucleotide sequences of three proline tRNAs from *Salmonella typhimurium*. Nucleic Acids Res. **12**:1559–1562.
264. **Kukral, A. M., K. Strauch, R. A. Maurer, and C. G. Miller.** 1987. Genetic analysis in *Salmonella typhimurium* with a small collection of randomly spaced insertions of transposon Tn*10* Δ16Δ17. J. Bacteriol. **169**:1787–1793.
265. **Kukuruzinska, M. A., W. F. Harrington, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Studies on the molecular weight and association of enzyme I. J. Biol. Chem. **257**:14470–14476.
266. **Kulla, H. G.** 1983. Regulatory citrate lyase mutants of *Salmonella typhimurium*. J. Bacteriol. **153**:546–549.
267. **Kustu, S., J. Hirschman, D. Burton, J. Jelesko, and J. C. Meeks.** 1984. Covalent modification of bacterial glutamine synthetase: physiological significance. Mol. Gen. Genet. **197**: 309–317.
268. **Kutsukake, K., and T. Iino.** 1985. Refined genetic analysis of the region II *che* mutants in *Salmonella typhimurium*. Mol. Gen. Genet. **199**:406–409.
269. **Kwan, H. S., and E. L. Barrett.** 1983. Purification and properties of trimethylamine oxide reductase from *Salmonella typhimurium*. J. Bacteriol. **155**:1455–1458.
270. **Kwan, H. S., and E. L. Barrett.** 1983. Roles for menaquinone and the two trimethylamine oxide (TMAO) reductases in TMAO respiration in *Salmonella typhimurium*: Mud(Ap^r *lac*) insertion mutations in *men* and *tor*. J. Bacteriol. **155**:1147–1155.
271. **Kwan, H. S., and E. L. Barrett.** 1984. Map locations and functions of *Salmonella typhimurium men* genes. J. Bacteriol. **159**:1090–1092.
272. **Kwan, H. S., H. W. Chui, and K. K. Wong.** 1988. *ack*::Mu *d*1–8 (Ap^r *lac*) operon fusions of *Salmonella typhimurium* LT2. Mol. Gen. Genet. **211**:183–185.
273. **Kwan, H. S., and K. K. Wong.** 1986. A general method for isolation of Mu d 1–8(Ap^r *lac*) operon fusions in *Salmonella typhimurium* LT2 from Tn*10* insertion strains: *chlC*::Mu*d*1–8. Mol. Gen. Genet. **205**:221–224.
274. **Lam, S., and J. R. Roth.** 1983. IS200: a *Salmonella*-specific insertion sequence. Cell **34**:951–960.
275. **Lam, S., and J. R. Roth.** 1983. Genetic mapping of IS*200* copies in *Salmonella typhimurium* strain LT2. Genetics **105**: 801–811.
276. **Lam, S., and J. R. Roth.** 1986. Structural and functional studies of insertion element IS*200*. J. Mol. Biol. **187**:157–167.
277. **Lampel, K. A., and M. Riley.** 1982. Discontinuity of homology of *Escherichia coli* and *Salmonella typhimurium* DNA in the *lac* region. Mol. Gen. Genet. **186**:82–86.
278. **LaRossa, R. A., and J. V. Schloss.** 1984. The sulfonylurea herbicide sulfometuron methyl is an extremely potent and selective inhibitor of acetolactate synthase in *Salmonella typhimurium*. J. Biol. Chem. **259**:8753–8757.
279. **LaRossa, R. A., and D. R. Smulski.** 1984. *ilvB*-encoded acetolactate synthase is resistant to the herbicide sulfometuron methyl. J. Bacteriol. **160**:391–394.
280. **LaRossa, R. A., T. K. VanDyk, and D. R. Smulski.** 1987. Toxic accumulation of α-ketobutyrate caused by inhibition of the branched-chain amino acid biosynthetic enzyme acetolactate synthase in *Salmonella typhimurium*. J. Bacteriol. **169**:1372–1378.
281. **Lawton, K. G., and H. W. Taber.** 1984. Isolation of F′ plasmids carrying a portion of the *Salmonella typhimurium* histidine transport operon. J. Bacteriol. **157**:697–702.
282. **Lederberg, E. M., and S. N. Cohen.** 1974. Transformation of *Salmonella typhimurium* by plasmid deoxyribonucleic acid. J. Bacteriol. **119**:1072–1074.
283. **Lee, G. S., and G. F.-L. Ames.** 1984. Analysis of promoter mutations in the histidine transport operon of *Salmonella typhimurium*: use of hybrid M13 bacteriophages for cloning, transformation, and sequencing. J. Bacteriol. **159**:1000–1005.
284. **Lee, J.-H., K. Burke, and G. Wilcox.** 1986. Mutations resulting in promoter-like sequences which enhance the expression of *araC* in *Salmonella typhimurium*. Gene **46**:113–121.
285. **Lee, J.-H., J. Nishitani, and G. Wilcox.** 1984. Genetic characterization of *Salmonella typhimurium* LT2 *ara* mutations. J. Bacteriol. **158**:344–346.
286. **Lee, P. C., B. R. Bochner, and B. N. Ames.** 1983. AppppA, heat-shock stress, and cell oxidation. Proc. Natl. Acad. Sci. USA **80**:7496–7500.
287. **Lee, S. Y., and C. T. Grubmeyer.** 1987. Purification and in vitro complementation of mutant histidinol dehydrogenases. J. Bacteriol. **169**:3938–3944.
288. **Lehner, A. F., S. Harvey, and C. W. Hill.** 1984. Mapping and spacer identification of rRNA operons of *Salmonella typhimurium*. J. Bacteriol. **160**:682–686.
289. **Lehner, A. F., and C. W. Hill.** 1985. Merodiploidy in *Escherichia coli-Salmonella typhimurium* crosses: the role of unequal recombination between ribosomal RNA genes. Genetics **110**:365–380.
290. **Leung, P. S. C., and J. Preiss.** 1987. Cloning of the ADPglucose pyrophosphorylase (*glgC*) and glycogen synthase (*glgA*) structural genes from *Salmonella typhimurium* LT2. J. Bacteriol. **169**:4349–4354.
291. **Leung, P. S. C., and J. Preiss.** 1987. Biosynthesis of bacterial glycogen: primary structure of *Salmonella typhimurium* ADPglucose synthetase as deduced from the nucleotide sequence of the *glgC* gene. J. Bacteriol. **169**:4355–4360.
292. **Lew, H. C., P. H. Mäkelä, H.-M. Kuhn, H. Mayer, and H. Nikaido.** 1986. Biosynthesis of enterobacterial common antigen requires dTDPglucose pyrophosphorylase determined by a *Salmonella typhimurium rfb* gene and a *Salmonella montevideo rfe* gene. J. Bacteriol. **168**:715–721.
293. **Liljeström, P., M. Luokkamäki, and E. T. Palva.** 1987. Isolation and characterization of a substitution mutation in the *ompR* gene of *Salmonella typhimurium* LT2. J. Bacteriol. **169**: 438–441.
294. **Liljeström, P., P. L. Määttänen, and E. T. Palva.** 1982. Cloning of the regulatory locus *ompB* of *Salmonella typhimurium* LT-2. I. Isolation of the *ompR* gene and identification of its gene product. Mol. Gen. Genet. **188**:184–189.
295. **Lin, H.-C., S.-P. Lei, G. Studnicka, and G. Wilcox.** 1985. The *araBAD* operon of *Salmonella typhimurium* LT2. III. Nucleotide sequence of *araD* and its flanking regions, and primary structure of its product, L-ribulose-5-phosphate 4-epimerase. Gene **34**:129–134.
296. **Lin, H.-C., S.-P. Lei, and G. Wilcox.** 1985. The *araBAD* operon of *Salmonella typhimurium* LT2. I. Nucleotide sequence of *araB* and primary structure of its product, ribulokinase. Gene **34**:111–122.
297. **Lin, H.-C., S.-P. Lei, and G. Wilcox.** 1985. The *araBAD* operon of *Salmonella typhimurium* LT2. II. Nucleotide sequence of *araA* and primary structure of its product, L-arabinose isomerase. Gene **34**:123–128.
298. **Low, B.** 1972. *Escherichia coli* K-12 F-prime factors, old and new. Bacteriol. Rev. **36**:587–607.
299. **Low, K. B.** 1987. Mapping techniques and determination of chromosome size, p. 1184–1189. *In* F. C. Neidhardt, J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
300. **MacAlister, T. J., W. R. Cook, R. Weigand, and L. I. Rothfield.** 1987. Membrane-murein attachment at the leading edge of the

division septum: a second membrane-murein structure associated with morphogenesis of the gram-negative bacterial division septum. J. Bacteriol. **169**:3945–3951.

301. **MacLachlan, P. R., and K. E. Sanderson.** 1985. Transformation of *Salmonella typhimurium* with plasmid DNA: differences between rough and smooth strains. J. Bacteriol. **161**:442–445.
302. **Macnab, R. M.** 1987. Flagella, p. 70–83. *In* F. C. Neidhardt, J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
303. **Madonna, M. J., R. L. Fuchs, and J. E. Brenchley.** 1985. Fine structure analysis of *Salmonella typhimurium* glutamate synthase genes. J. Bacteriol. **161**:353–360.
304. **Mahan, M. J., and L. N. Csonka.** 1983. Genetic analysis of the *proBA* genes of *Salmonella typhimurium*: physical and genetic analyses of the cloned *proB*$^+$*A*$^+$ genes of *Escherichia coli* and of a mutant allele that confers proline overproduction and enhanced osmotolerance. J. Bacteriol. **156**:1249–1262.
305. **Mäkelä, P. H.** 1963. Hfr males in *Salmonella abony*. Genetics **48**:423–429.
306. **Maloy, S. R., and W. D. Nunn.** 1981. Selection for loss of tetracycline resistance by *Escherichia coli*. J. Bacteriol. **145**: 1110–1112.
307. **Maloy, S. R., and J. R. Roth.** 1983. Regulation of proline utilization in *Salmonella typhimurium*: characterization of *put*::Mu *d*(Ap, *lac*) operon fusions. J. Bacteriol. **154**:561–568.
308. **Manoil, C., and J. Beckwith.** 1985. Tn*phoA*: a transposon probe for protein export signals. Proc. Natl. Acad. Sci. USA **82**:8129–8133.
309. **Manoil C., and J. Beckwith.** 1986. A genetic approach to analysing membrane protein topology. Science **233**:1403–1408.
310. **Matsubara, K., K. Ohnishi, and K. Kiritani.** 1987. Location of *livA* gene participating in the high-affinity transport of branched-chain amino acids in *Salmonella typhimurium* LT2. Jpn. J. Genet. **62**:189–196.
311. **Matsubara, K., K. Ohnishi, and K. Kiritani.** 1988. The third general transport system for branched-chain amino acids in *Salmonella typhimurium*. J. Gen. Appl. Microbiol. **34**:183–189.
312. **Maurer, R., B. C. Osmond, and D. Botstein.** 1984. Genetic analysis of DNA replication in bacteria: *dnaB* mutations that suppress *dnaC* mutations and *dnaQ* mutations that suppress *dnaE* mutations in *Salmonella typhimurium*. Genetics **108**:25–38.
313. **Maurer, R., B. C. Osmond, E. Shekhtman, A. Wong, and D. Botstein.** 1984. Functional interchangeability of DNA replication genes in *Salmonella typhimurium* and *Escherichia coli* demonstrated by a general complementation procedure. Genetics **108**:1–23.
314. **Maurer, R., and A. Wong.** 1988. Dominant lethal mutations in the *dnaB* helicase gene of *Salmonella typhimurium*. J. Bacteriol. **170**:3682–3688.
315. **McCarter, L., K. Krajewska-Grynkiewicz, D. Trinh, G. Wei, and S. Kustu.** 1984. Characterization of mutations that lie in the promoter-regulatory region for *glnA*, the structural gene encoding glutamine synthetase. Mol. Gen. Genet. **197**:150–160.
316. **Meadow, N. D., and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Isolation and characterization of a glucose-specific phosphocarrier protein (III^{Glc}) from *Salmonella typhimurium*. J. Biol. Chem. **257**:14526–14537.
317. **Meadow, N. D., J. M. Rosenberg, H. M. Pinkert, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Evidence that *crr* is the structural gene for the *Salmonella typhimurium* glucose-specific phosphocarrier protein III^{Glc}. J. Biol. Chem. **257**:14538–14542.
318. **Meadow, N. D., D. W. Saffen, R. P. Dottin, and S. Roseman.** 1982. Molecular cloning of the *crr* gene and evidence that it is the structural gene for III^{Glc}, a phosphocarrier protein of the bacterial phosphotransferase system. Proc. Natl. Acad. Sci. USA **79**:2528–2532.
319. **Meier, U., and H. Mayer.** 1985. Genetic location of genes encoding enterobacterial common antigen. J. Bacteriol. **163**: 756–762.
320. **Mergeay, M., and J. Gerits.** 1983. Transduction of *Escherichia coli trp* genes in *Salmonella typhimurium* and effect of *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine on transduction with heterogenotic DNA. J. Gen. Microbiol. **129**:321–335.
321. **Messing, J.** 1983. New M13 vectors for cloning. Methods Enzymol. **101**:20–78.
322. **Michaels, G., and R. A. Kelln.** 1983. Construction and use of *pyr*::*lac* fusion strains to study regulation of pyrimidine biosynthesis in *Salmonella typhimurium*. Mol. Gen. Genet. **189**: 463–470.
323. **Michaels, G., R. A. Kelln, and F. E. Nargang.** 1987. Cloning, nucleotide sequence and expression of the *pyrBI* operon of *Salmonella typhimurium* LT2. Eur. J. Biochem. **166**:55–61.
324. **Michiels, T., M. Y. Popoff, S. Durviaux, C. Coynault, and G. Cornelis.** 1987. A new method for the physical and genetic mapping of large plasmids: application to the localisation of the virulence determinants on the 90 kb plasmid of *Salmonella typhimurium*. Microb. Path. **3**:109–116.
325. **Miller, B. E., and N. M. Kredich.** 1987. Purification of the *cysB* protein from *Salmonella typhimurium*. J. Biol. Chem. **262**: 6006–6009.
326. **Miller, C. G., K. L.. Strauch, A. M. Kukral, J. L. Miller, P. T. Wingfield, G. J. Mazzei, R. C. Werlen, P. Graber, and N. R. Movva.** 1987. N-terminal methionine-specific peptidase in *Salmonella typhimurium*. Proc. Natl. Acad. Sci. USA **84**:2718–2722.
327. **Miller, E. S., and J. E. Brenchley.** 1983. Cloning and characterization of *gdhA*, the structural gene for glutamate dehydrogenase of *Salmonella typhimurium*. J. Bacteriol. **157**:171–178.
328. **Miller, J. F., W. J. Dower, and L. S. Tompkins.** 1988. High-voltage electroporation of bacteria: genetic transformation of *Campylobacter jejuni* with plasmid DNA. Proc. Natl. Acad. Sci. USA **85**:856–860.
329. **Misko, T. P., W. J. Mitchell, N. D. Meadow, and S. Roseman.** 1987. Sugar transport by the bacterial phosphotransferase system. J. Biol. Chem. **262**:16261–16266.
330. **Mitchell, W. J., T. P. Misko, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Regulation of other transport systems (lactose and melibiose). J. Biol. Chem. **257**:14553–14564.
331. **Mizuno, T., N. Mutoh, S. M. Panasenko, and Y. Imae.** 1986. Acquisition of maltose chemotaxis in *Salmonella typhimurium* by the introduction of the *Escherichia coli* chemosensory transducer gene. J. Bacteriol. **165**:890–895.
332. **Monroe, R. S., and N. M. Kredich.** 1988. Isolation of *Salmonella typhimurium cys* genes by transduction with a library of recombinant plasmids packaged in bacteriophage P22HT capsids. J. Bacteriol. **170**:42–47.
333. **Morgan, R. W., M. F. Christman, F. S. Jacobson, G. Storz, and B. N. Ames.** 1986. Hydrogen peroxide-inducible proteins in *Salmonella typhimurium* overlap with heat shock and other stress proteins. Proc. Natl. Acad. Sci. USA **83**:8059–8063.
334. **Morisato, D., J. C. Way, H.-J. Kim, and N. Kleckner.** 1983. Tn*10* transposase acts preferentially on nearby transposon ends *in vivo*. Cell **32**:799–807.
335. **Müller, K.-H., T. J. Trust, and W. W. Kay.** 1988. Unmasking of bacteriophage Mu lipopolysaccharide receptors in *Salmonella enteritidis* confers sensitivity to Mu and permits Mu mutagenesis. J. Bacteriol. **170**:1076–1081.
336. **Müller, N., H.-G. Heine, and W. Boos.** 1985. Characterization of the *Salmonella typhimurium mgl* operon and its gene products. J. Bacteriol. **163**:37–45.
337. **Murata-Matsubara, K., K. Ohnishi, and K. Kiritani.** 1985. Genetic and biochemical studies of *livS* mutation affecting the regulation of branched-chain amino acid transport in *Salmonella typhimurium*. Jpn. J. Genet. **60**:11–25.
338. **Nakamura, T., Y. Kon, H. Iwahashi, and Y. Eguchi.** 1983. Evidence that thiosulfate assimilation by *Salmonella typhimurium* is catalyzed by cysteine synthase B. J. Bacteriol. **156**: 656–662.
339. **Naumann, D., C. Schultz, J. Born, H. Labischinski, K. Brandenburg, G. von Busse, H. Brade, and U. Seydel.** 1987. Inves-

tigations into the polymorphism of lipid A from lipopolysaccharides of *Escherichia coli* and *Salmonella minnesota* by Fourier-transform infrared spectroscopy. Eur. J. Biochem. **164**:159–169.

340. **Negre, D., J. C. Cortay, P. Donini, and A. J. Cozzone.** 1988. Inaccurate protein synthesis in a mutant of *Salmonella typhimurium* defective in transfer RNA pseudouridylation. FEBS Lett. **234**:165–168.
341. **Nelson, S. O., A. R. J. Schuitema, R. Benne, L. H. T. van der Ploeg, J. S. Plijter, F. Aan, and P. W. Postma.** 1984. Molecular cloning, sequencing, and expression of the *crr* gene: the structural gene for III^{Glc} of the bacterial PEP:glucose phosphotransferase system. EMBO J. **3**:1587–1593.
342. **Neuhard, J., K. F. Jensen, and E. Stauning.** 1982. *Salmonella typhimurium* mutants with altered expression of the *pyrA* gene due to changes in RNA polymerase. EMBO J. **1**:1141–1145.
343. **Neuhard, J., R. A. Kelln, and E. Stauning.** 1986. Cloning and structural characterization of the *Salmonella typhimurium pyrC* gene encoding dihydroorotase. Eur. J. Biochem. **157**: 335–342.
344. **Neuhard, J., E. Stauning, and R. A. Kelln.** 1985. Cloning and characterization of the *pyrE* gene and of *PyrE*::Mud1 (Ap^R *lac*) fusions from *Salmonella typhimurium*. Eur. J. Biochem. **146**: 597–603.
345. **Newbury, S. F., N. H. Smith, E. C. Robinson, I. D. Hiles, and C. F. Higgins.** 1987. Stabilization of translationally active mRNA by prokaryotic REP sequences. Cell **48**:297–310.
346. **Niedhardt, F. C., J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.).** 1987. *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
347. **Nixon, B. T., C. W. Ronson, and F. M. Ausubel.** 1986. Two-component regulatory systems responsive to environmental stimuli share strongly conserved domains with the nitrogen assimilation regulatory genes *ntrB* and *ntrC*. Proc. Natl. Acad. Sci. USA **83**:7850–7854.
348. **Nnalue, N. A., and B. A. D. Stocker.** 1986. Some *galE* mutants of *Salmonella choleraesuis* retain virulence. Infect. Immun. **54**:635–640.
349. **Nnalue, N. A., and B. A. D. Stocker.** 1987. Test of the virulence and live-vaccine efficacy of auxotrophic and *galE* derivatives of *Salmonella choleraesuis*. Infect. Immun. **55**:955–962.
350. **Noel, K. D., and G. F.-L. Ames.** 1978. Evidence for a common mechanism for the insertion of the Tn*10* transposon and for the generation of Tn*10*-stimulated deletions. Mol. Gen. Genet. **166**:217–223.
351. **Novotny, M. J., W. L. Frederickson, E. B. Waygood, and M. H. Saier, Jr.** 1985. Allosteric regulation of glycerol kinase by enzyme III^{Glc} of the phosphotransferase system in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **162**:810–816.
352. **Nyman, K., K. Nakamura, H. Ohtsubo, and E. Ohtsubo.** 1981. Distribution of the insertion sequence IS1 in gram-negative bacteria. Nature (London) **289**:609–612.
353. **Ohnishi, K., A. Hasegawa, K. Matsubara, T. Date, T. Okada, and K. Kiritani.** 1988. Cloning and nucleotide sequence of the *brnQ* gene, the structural gene for a membrane-associated component of the LIV-II transport system for branched-chain amino acids in *Salmonella typhimurium*. Jpn. J. Genet. **63**:343–357.
354. **Ohnishi, K., M. Homma, K. Kutsukake, and T. Iino.** 1987. Formation of flagella lacking outer rings by *flaM*, *flaU*, and *flaY* mutants of *Escherichia coli*. J. Bacteriol. **169**:1485–1488.
355. **O'Reilly, C., P. D. Turner, P. F. Smith-Keary, and D. J. McConnell.** 1984. Molecular cloning of genes involved in purine biosynthetic and salvage pathways of *Salmonella typhimurium*. Mol. Gen. Genet. **196**:152–157.
356. **Orr, J. C., D. W. Bryant, D. R. McCalla, and M. A. Quilliam.** 1985. Dinitropyrene-resistant *Salmonella typhimurium* are deficient in an acetyl-CoA acetyltransferase. Chem.-Biol. Interactions **54**:281–288.
357. **Orrego, C., and E. Eisenstadt.** 1987. An inducible pathway is required for mutagenesis in *Salmonella typhimurium* LT2. J. Bacteriol. **169**:2885–2888.
358. **Ostrowski, J., G. Jagura-Burdzy, and N. M. Kredich.** 1987. DNA sequences of the *cysB* regions of *Salmonella typhimurium* and *Escherichia coli*. J. Biol. Chem. **262**:5999–6005.
359. **Overbye, K. M., S. K. Basu, and P. Margolin.** 1983. Loss of DNA topoisomerase I activity alters many cellular functions in *Salmonella typhimurium*. Cold Spring Harbor Symp. Quant. Biol. **47**:785–791.
360. **Overbye, K. M., and P. Margolin.** 1981. Role of the *supX* gene in ultraviolet light-induced mutagenesis in *Salmonella typhimurium*. J. Bacteriol. **146**:170–178.
361. **Ow, D. W., V. Sundaresan, D. M. Rothstein, S. E. Brown, and F. M. Ausubel.** 1983. Promoters regulated by the *glnG* (*ntrC*) and *nifA* gene products share a heptameric consensus sequence in the −15 region. Proc. Natl. Acad. Sci. USA **80**:2524–2528.
362. **Pang, P. P., A. S. Lundberg, and G. C. Walker.** 1985. Identification and characterization of the *mutL* and *mutS* gene products of *Salmonella typhimurium* LT2. J. Bacteriol. **163**: 1007–1015.
363. **Pang, P. P., S. D. Tsen, A. S. Lundberg, and G. C. Walker.** 1984. The *mutH*, *mutL*, *mutS*, and *uvrD* genes of *Salmonella typhimurium* LT2. Cold Spring Harbor Symp. Quant. Biol. **49**: 597–602.
364. **Pang, P. P., and G. C. Walker.** 1983. Identification of the *uvrD* gene product of *Salmonella typhimurium* LT2. J. Bacteriol. **153**:1172–1179.
365. **Pang, P. P., and G. C. Walker.** 1983. The *Salmonella typhimurium* LT2 *uvrD* gene is regulated by the *lexA* gene product. J. Bacteriol. **154**:1502–1504.
366. **Park, U. E., J. R. Roth, and B. M. Olivera.** 1988. *Salmonella typhimurium* mutants lacking NAD pyrophosphatase. J. Bacteriol. **170**:3725–3730.
367. **Paveglio, M. T., J. S. Tang, R. E. Unger, and E. L. Barrett.** 1988. Formate-nitrate respiration in *Salmonella typhimurium*: studies of two *rha*-linked *fdn* genes. J. Bacteriol. **170**:213–217.
368. **Payne, G. M., E. N. Spudich, and G. F.-L. Ames.** 1985. A mutational hot-spot in the *hisM* gene of the histidine transport operon in *Salmonella typhimurium* is due to deletion of repeated sequences and results in an altered specificity of transport. Mol. Gen. Genet. **200**:493–496.
369. **Peterson, A. A., and E. J. McGroarty.** 1985. High-molecular-weight components in lipopolysaccharides of *Salmonella typhimurium*, *Salmonella minnesota*, and *Escherichia coli*. .J. Bacteriol. **162**:738–745.
370. **Pflugrath, J. W., and F. A. Quiocho.** 1988. The 2 Å resolution structure of the sulfate-binding protein involved in active transport in *Salmonella typhimurium*. J. Mol. Biol. **200**:163–180.
371. **Pizarro, R. A., G. O. Boselli, and L. V. Orce.** 1985. Biochemical and phenotypical correction of an envelope mutant of *Salmonella typhimurium*. Arch. Int. Physiol. Biochim. **92**:333–337.
372. **Plamann, L. S., and G. V. Stauffer.** 1987. Nucleotide sequence of the *Salmonella typhimurium metR* gene and the *metR-metE* control region. J. Bacteriol. **169**:3932–3937.
373. **Poland, J., and P. D. Ayling.** 1984. Methionine and glutamine transport systems in D-methionine utilising revertants of *Salmonella typhimurium*. Mol. Gen. Genet. **194**:219–226.
374. **Popoff, M. Y., I. Miras, C. Coynault, C. Lasselin, and P. Pardon.** 1984. Molecular relationships between virulence plasmids of *Salmonella* serotypes *typhimurium* and *dublin* and large plasmids of other *Salmonella* serotypes. Ann. Microbiol. (Paris) **135**:389–398.
375. **Postma, P. W., W. Epstein, A. R. J. Schuitema, and S. O. Nelson.** 1984. Interaction between III^{Glc} of the phosphoenolpyruvate:sugar phosphotransferase system and glycerol kinase of *Salmonella typhimurium*. J. Bacteriol. **158**:351–353.
376. **Postma, P. W., H. G. Keizer, and P. Koolwijk.** 1986. Transport of trehalose in *Salmonella typhimurium*. J. Bacteriol. **168**: 1107–1111.
377. **Postma, P. W., and J. W. Lengeler.** 1985. Phosphoenolpyruvate:carbohydrate phosphotransferase system of bacteria. Microbiol. Rev. **49**:232–269.

378. **Powers, D. A., and S. Roseman.** 1984. The primary structure of *Salmonella typhimurium* HPr, a phosphocarrier protein of the phosphoenolpyruvate:glycose phosphotransferase system. A correction. J. Biol. Chem. **259**:15212–15214.
379. **Pruss, G. J.** 1985. DNA topoisomerase I mutants. Increased heterogeneity in linking number and other replicon-dependent changes in DNA supercoiling. J. Mol. Biol. **185**:51–63.
380. **Pruss, G. J., and K. Drlica.** 1985. DNA supercoiling and suppression of the *leu-500* promoter mutation. J. Bacteriol. **164**:947–949.
381. **Pruss, G. J., and K. Drlica.** 1986. Topoisomerase I mutants: the gene on pBR322 that encodes resistance to tetracycline affects plasmid DNA supercoiling. Proc. Natl. Acad. Sci. USA **83**:8952–8956.
382. **Purcell, B. K., J. Pruckler, and S. Clegg.** 1987. Nucleotide sequences of the genes encoding type 1 fimbrial subunits of *Klebsiella pneumoniae* and *Salmonella typhimurium*. J. Bacteriol. **169**:5831–5834.
383. **Qureshi, N., K. Takayama, D. Heller, and C. Fenselau.** 1983. Position of ester groups in the lipid A backbone of lipopolysaccharides obtained from *Salmonella typhimurium*. J. Biol. Chem. **258**:12947–12951.
384. **Raetz, C. R. H., S. Purcell, M. V. Meyer, N. Qureshi, and K. Takayama.** 1985. Isolation and characterization of eight lipid A precursors from a 3-deoxy-D-manno-oculosonic acid-deficient mutant of *Salmonella typhimurium*. J. Biol. Chem. **260**:16080–16088.
385. **Ravid, S., and M. Eisenbach.** 1984. Direction of flagellar rotation in bacterial cell envelopes. J. Bacteriol. **158**:222–230.
386. **Reeve, C. A., A. T. Bockman, and A. Matin.** 1984. Role of protein degradation in the survival of carbon-starved *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **157**:758–763.
387. **Richardson, S. M. H., C. F. Higgins, and D. M. J. Lilley.** 1984. The genetic control of DNA supercoiling in *Salmonella typhimurium*. EMBO J. **3**:1745–1752.
388. **Rick, P. D., B. A. Neumeyer, and D. A. Young.** 1983. Effect of altered lipid A synthesis on the synthesis of the OmpA protein in *Salmonella typhimurium*. J. Biol. Chem. **258**:629–635.
389. **Rick, P. D., S. Wolski, K. Barr, S. Ward, and L. Ramsay-Sharer.** 1988. Accumulation of a lipid-linked intermediate involved in enterobacterial common antigen synthesis in *Salmonella typhimurium* mutants lacking dTDP-glucose pyrophosphorylase. J. Bacteriol. **170**:4008–4014.
390. **Riggs, D., and S. Artz.** 1984. The *hisD-hisC* gene border of the *Salmonella typhimurium* histidine operon. Mol. Gen. Genet. **196**:526–529.
391. **Riggs, D. L., R. D. Mueller, H.-S. Kwan, and S. W. Artz.** 1986. Promoter domain mediates guanosine tetraphosphate activation of the histidine operon. Proc. Natl. Acad. Sci. USA **83**: 9333–9337.
392. **Riley, M., and S. Krawiec.** 1987. Genome organization, p. 967–981. *In* F. C. Neidhardt, J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
393. **Riley, M., C. O'Reilly, and D. McConnell.** 1983. Physical map of *Salmonella typhimurium* LT2 DNA in the vicinity of the *proA* gene. J. Bacteriol. **157**:655–657.
394. **Ritchie, L. J., R. M. Hall, and D. M. Podger.** 1986. Mutant of *Salmonella typhimurium* LT2 deficient in DNA adenine methylation. J. Bacteriol. **167**:420–422.
395. **Roof, D. M., and J. R. Roth.** 1988. Ethanolamine utilization in *Salmonella typhimurium*. J. Bacteriol. **170**:3855–3863.
396. **Rosenthal, E. R., and J. M. Calvo.** 1987. Transcription termination sites at the distal end of the *leu* operon of *Salmonella typhimurium*. J. Mol. Biol. **194**:443–452.
397. **Rothstein, S. J., R. A. Jorgensen, K. Postle, and W. S. Reznikoff.** 1980. The inverted repeats of Tn*5* are functionally different. Cell **19**:795–805.
398. **Rowen, L., J. A. Kobori, and S. Scherer.** 1982. Cloning of bacterial DNA replication genes. Mol. Gen. Genet. **187**:501–509.
399. **Roy, S., and M. Chakravorty.** 1986. Spontaneous deletions of drug-resistance determinants from *Salmonella typhimurium* in *Escherichia coli*. J. Med. Microbiol. **22**:119–123.
400. **Rudd, K. E., B. R. Bochner, M. Cashel, and J. R. Roth.** 1985. Mutations in the *spoT* gene of *Salmonella typhimurium*: effects on *his* operon expression. J. Bacteriol. **163**:534–542.
401. **Rudd, K. E., and R. Menzel.** 1987. *his* operons of *Escherichia coli* and *Salmonella typhimurium* are regulated by DNA supercoiling. Proc. Natl. Acad. Sci. USA 84:517–521.
402. **Russo, A. F., and D. E. Koshland, Jr.** 1983. Separation of signal transduction and adaptation functions of the aspartate receptor in bacterial sensing. Science **220**:1016–1020.
403. **Russo, A. F., and D. E. Koshland, Jr.** 1985. Identification of the *tip*-encoded receptor in bacterial sensing. J. Bacteriol. **165**: 276–282.
404. **Sabe, H., T. Miwa, T. Kodaki, K. Izui, S. Hiraga, and H. Katsuki.** 1984. Molecular cloning of the phosphoenolpyruvate carboxylase gene, *ppc*, of *Escherichia coli*. Gene **31**:279–283.
405. **Saier, M. H., Jr., F. C. Grenier, C. A. Lee, and E. B. Waygood.** 1985. Evidence for the evolutionary relatedness of the proteins of the bacterial phosphoenolpyruvate:sugar phosphotransferase system. J. Cell. Biochem. **27**:43–56.
406. **Saier, M. H., Jr., M. J. Novotny, D. Comeau-Fuhrman, T. Osumi, and J. D. Desai.** 1983. Cooperative binding of the sugar substrates and allosteric regulatory protein (enzyme III^{Glc} of the phosphotransferase system) to the lactose and melibiose permeases in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **155**:1351–1357.
407. **Saier, M. H., Jr., D. L. Wentzel, B. U. Feucht, and J. J. Judice.** 1975. A transport system for phosphoenolpyruvate, 2-phosphoglycerate, and 3-phosphoglycerate in *Salmonella typhimurium*. J. Biol. Chem. **250**:5089–5096.
408. **Sanderson, K. E.** 1967. Revised linkage map of *Salmonella typhimurium*. Bacteriol. Rev. **31**:354–372.
409. **Sanderson, K. E.** 1970. Current linkage map of *Salmonella typhimurium*. Bacteriol. Rev. **34**:176–193.
410. **Sanderson, K. E.** 1972. Linkage map of *Salmonella typhimurium*, edition IV. Bacteriol. Rev. **36**:558–586.
411. **Sanderson, K. E., and M. Demerec.** 1965. The linkage map of *Salmonella typhimurium*. Genetics **51**:897–913.
412. **Sanderson, K. E., and P. E. Hartman.** 1978. Linkage map of *Salmonella typhimurium*, edition V. Microbiol. Rev. **42**:471–519.
413. **Sanderson, K. E., and J. A. Hurley.** 1987. Linkage map of *Salmonella typhimurium*, p. 877–918. *In* F. C. Neidhardt, J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
414. **Sanderson, K. E., J. Janzer, and J. Head.** 1981. Influence of lipopolysaccharide and protein in the cell envelope on recipient capacity in conjugation of *Salmonella typhimurium*. J. Bacteriol. **148**:283–293.
415. **Sanderson, K. E., S. K. Kadam, and P. R. MacLachlan.** 1983. Derepression of F factor function in *Salmonella typhimurium*. Can. J. Microbiol. **29**:1205–1212.
416. **Sanderson, K. E., and P. R. MacLachlan.** 1983. F-mediated conjugation, F^+ strains, and Hfr strains of *Salmonella typhimurium* and *Salmonella abony*, p. 1138–1144. *In* F. C. Neidhardt, J. L. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli* and *Salmonella typhimurium*: cellular and molecular biology. American Society for Microbiology, Washington, D.C.
417. **Sanderson, K. E., H. Ross, L. Ziegler, and P. H. Mäkelä.** 1972. F^+, Hfr, and F′ strains of *Salmonella typhimurium* and *Salmonella abony*. Bacteriol. Rev. **36**:608–637.
418. **Sanderson, K. E., and J. R. Roth.** 1983. Linkage map of *Salmonella typhimurium*, edition VI. Microbiol. Rev. **47**:410–453.
419. **Sarno, M. V., L. G. Tenn, A. Desai, A. M. Chin, F. C. Grenier, and M. H. Saier, Jr.** 1984. Genetic evidence for glucitol-specific enzyme III, an essential phosphocarrier protein of the

Salmonella typhimurium glucitol phosphotransferase system. J. Bacteriol. **157**:953–955.

420. **Sawers, R. G., D. J. Jamieson, C. F. Higgins, and D. H. Boxer.** 1986. Characterization and physiological roles of membrane-bound hydrogenase isoenzymes from *Salmonella typhimurium*. J. Bacteriol. **168**:398–404.
421. **Schloss, J. V., D. E. Van Dyk, J. F. Vasta, and R. M. Kutny.** 1985. Purification and properties of *Salmonella typhimurium* acetolactate synthase isozyme II from *Escherichia coli* HB101/pDU9. Biochemistry **24**:4952–4959.
422. **Schmid, M. B., and J. R. Roth.** 1983. Selection and endpoint distribution of bacterial inversion mutations. Genetics **105**: 539–557.
423. **Schmid, M. B., and J. R. Roth.** 1983. Genetic methods for analysis and manipulation of inversion mutations in bacteria. Genetics **105**:517–537.
424. **Schmid, M. B., and J. R. Roth.** 1987. Gene location affects expression level in *Salmonella typhimurium*. J. Bacteriol. **169**: 2872–2875.
425. **Schmidt, C., and H. Schmieger.** 1984. Selective transduction of recombinant plasmids with cloned *pac* sites by *Salmonella* phage P22. Mol. Gen. Genet. **196**:123–128.
426. **Schmieger, H.** 1982. Packaging signals for phage P22 on the chromosome of *Salmonella typhimurium*. Mol. Gen. Genet. **187**:516–518.
427. **Schmieger, H.** 1984. *pac* sites are indispensable for in vivo packaging of DNA by phage P22. Mol. Gen. Genet. **195**:252–255.
428. **Schmitz, G., P. Dürre, G. Mullenbach, and G. F.-L. Ames.** 1987. Nitrogen regulation of transport operons: analysis of promoters *argTr* and *dhuA*. Mol. Gen. Genet. **209**:403–407.
429. **Schoner, B., and R. G. Schoner.** 1981. Distribution of IS*5* in bacteria. Gene **16**:347–352.
430. **Schroeder, C. J., and W. J. Dobrogosz.** 1986. Cloning and DNA sequence analysis of the wild-type and mutant cyclic AMP receptor protein genes from *Salmonella typhimurium*. J. Bacteriol. **167**:616–622.
431. **Schulte, L. L., L. T. Stauffer, and G. V. Stauffer.** 1984. Cloning and characterization of the *Salmonella typhimurium metE* gene. J. Bacteriol. **158**:928–933.
432. **Scott, T. N., and M. I. Simon.** 1982. Genetic analysis of the mechanism of the *Salmonella* phase variation site specific recombination system. Mol. Gen. Genet. **188**:313–321.
433. **Searles, L. L., S. R. Wessler, and J. M. Calvo.** 1983. Transcription attenuation is the major mechanism by which the *leu* operon of *Salmonella typhimurium* is controlled. J. Mol. Biol. **163**:377–394.
434. **Sengstag, C., and W. Arber.** 1983. IS2 insertion is a major cause of spontaneous mutagenesis of the bacteriophage P1: a non-random distribution of target sites. EMBO J. **2**:67–71.
435. **Shapiro, J. A.** 1969. Mutations caused by the insertion of genetic material into the galactose operon of *Escherichia coli*. J. Mol. Biol. **40**:93–105.
436. **Shen, M. M., E. A. Raleigh, and N. Kleckner.** 1987. Physical analysis of Tn*10*- and IS*10*-promoted transpositions and rearrangements. Genetics **116**:359–369.
437. **Simms, S. A., E. W. Cornman, J. Mottonen, and J. Stock.** 1987. Active site of the enzyme which demethylates receptors during bacterial chemotaxis. J. Biol. Chem. **262**:29–31.
438. **Simms, S. A., M. G. Keane, and J. Stock.** 1985. Multiple forms of the CheB methylesterase in bacterial chemosensing. J. Biol. Chem. **260**:10161–10168.
439. **Simons, R. W., B. C. Hoopes, W. R. McClure, and N. Kleckner.** 1983. Three promoters near the termini of IS*10*: pIN, pOUT, and pIII. Cell **34**:673–682.
440. **Skavronskaya, A. G., N. F. Stepanova, and I. V. Andreeva.** 1982. UV-mutable hybrids of *Salmonella* incorporating *Escherichia coli* region adjacent to *tryptophan* operon. Mol. Gen. Genet. **185**:315–318.
441. **Skovgaard, O., and F. G. Hansen.** 1987. Comparison of *dnaA* nucleotide sequences of *Escherichia coli*, *Salmonella typhimurium*, and *Serratia marcescens*. J. Bacteriol. **169**:3976–3981.
442. **Smith, B. P., M. Reina-Guerra, S. K. Hoiseth, B. A. D. Stocker, F. Habasha, E. Johnson, and F. Merritt.** 1984. Aromatic-dependent *Salmonella typhimurium* as modified live vaccines for calves. Am. J. Vet. Res. **45**:59–66.
443. **Smith, G. R., C. M. Roberts, and D. W. Schultz.** 1986. Activity of Chi recombinational hotspots in *Salmonella typhimurium*. Genetics **112**:429–439.
444. **Smith, H. R., G. O. Humphreys, N. D. F. Grindley, J. N. Grindley, and E. S. Anderson.** 1973. Molecular studies of an *fi*$^+$ plasmid from strains of *Salmonella typhimurium*. Mol. Gen. Genet. **126**:143–151.
445. **Smith, J. M., E. H. Rowsell, J. Shioi, and B. L. Taylor.** 1988. Identification of a site of ATP requirement for signal processing in bacterial chemotaxis. J. Bacteriol. **170**:2698–2704.
446. **Smith, N. H., P. B. Crichton, D. C. Old, and C. F. Higgins.** 1988. Ribosomal-RNA patterns of *Escherichia coli*, *Salmonella typhimurium* and related Enterobacteriaceae. J. Med. Microbiol. **25**:223–228.
447. **Somers, J. M., and W. W. Kay.** 1983. Genetic fine structure of the tricarboxylate transport (*tct*) locus of *Salmonella typhimurium*. Mol. Gen. Genet. **190**:20–26.
448. **Spector, M. P., Z. Aliabadi, T. Gonzalez, and J. W. Foster.** 1986. Global control in *Salmonella typhimurium*: two-dimensional electrophoretic analysis of starvation-, anaerobiosis-, and heat shock-inducible proteins. J. Bacteriol. **168**:420–424.
449. **Spector, M. P., J. M. Hill, E. A. Holley, and J. W. Foster.** 1985. Genetic characterization of pyridine nucleotide uptake mutants of *Salmonella typhimurium*. J. Gen. Microbiol. **131**:1313–1322.
450. **Spector, M. P., Y. K. Park, S. Tirgari, T. Gonzalez, and J. W. Foster.** 1988. Identification and characterization of starvation-regulated genetic loci in *Salmonella typhimurium* by using Mu d-directed *lacZ* operon fusions. J. Bacteriol. **170**:345–351.
451. **Squires, C. H., M. DeFelice, C. T. Lago, and J. M. Calvo.** 1983. *ilvHI* locus of *Salmonella typhimurium*. J. Bacteriol. **154**:1054–1063.
452. **Stackhouse, T. M., J. J. Onuffer, C. R. Matthews, S. A. Ahmed, and E. W. Miles.** 1988. Folding of homologous proteins: conservation of the folding mechanism of the α subunit of tryptophan synthase from *Escherichia coli*, *Salmonella typhimurium*, and five interspecies hybrids. Biochemistry **27**:824–832.
453. **Stalker, D. M., W. R. Hiatt, and L. Comai.** 1985. A single amino acid substitution in the enzyme 5-enolpyruvylshikimate-3-phosphate synthase confers resistance to the herbicide glyphosate. J. Biol. Chem. **260**:4724–4728.
454. **Stan-Lotter, H., and P. D. Bragg.** 1985. Subunit distribution of the sulfhydryl groups of the F_1 adenosine triphosphatase of *Salmonella typhimurium*. Arch. Biochem. Biophys. **239**:280–285.
455. **Stern, M. J., C. F. Higgins, and G. F.-L. Ames.** 1984. Isolation and characterization of *lac* fusions to two nitrogen-regulated promoters. Mol. Gen. Genet. **195**:219–227.
456. **Stock, A., T. Chen, D. Welsh, and J. Stock.** 1988. CheA protein, a central regulator of bacterial chemotaxis, belongs to a family of proteins that control gene expression in response to changing environmental conditions. Proc. Natl. Acad. Sci. USA **85**:1403–1407.
457. **Stock, A., D. E. Koshland, Jr., and J. Stock.**1985. Homologies between the *Salmonella* typhimurium CheY protein and proteins involved in the regulation of chemotaxis, membrane protein synthesis, and sporulation. Proc. Natl. Acad. Sci. USA **82**:7989–7993.
458. **Stock, A., J. Mottonen, T. Chen, and J. Stock.** 1987. Identification of a possible nucleotide binding site in CheW, a protein required for sensory transduction in bacterial chemotaxis. J. Biol. Chem. **262**:535–537.
459. **Stock, A., E. Schaeffer, D. E. Koshland, Jr., and J. Stock.** 1987. A second type of protein methylation reaction in bacterial chemotaxis. J. Biol. Chem. **262**:8011–8014.
460. **Stock, A. M., and J. B. Stock.** 1987. Purification and characterization of the CheZ protein of bacterial chemotaxis. J. Bacteriol. **169**:3301–3311.
461. **Stock, J., G. Kersulis, and D. E. Koshland, Jr.** 1985. Neither methylation nor demethylating enzymes are required for bac-

terial chemotaxis. Cell **42**:683–690.
462. **Stock, J. B., E. B. Waygood, N. D. Meadow, P. W. Postma, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. The glucose receptors of the *Salmonella typhimurium* phosphotransferase system. J. Biol. Chem. **257**: 14543–14552.
463. **Storz, G., M. F. Christman, H. Sies, and B. N. Ames.** 1987. Spontaneous mutagenesis and oxidative damage to DNA in *Salmonella typhimurium*. Proc. Natl. Acad. Sci. USA **84**:8917–8921.
464. **Stover, C. K., J. Kemper, and R. C. Marsh.** 1988. Molecular cloning and characterization of *supQ/newD*, a gene substitution system for the *leuD* gene of *Salmonella typhimurium*. J. Bacteriol. **170**:3115–3124.
465. **Strain, S. M., I. M. Armitage, L. Anderson, K. Takayama, N. Qureshi, and C. R. H. Raetz.** 1985. Location of polar substituents and fatty acyl chains on lipid A precursors from a 3-deoxy-D-manno-octulosonic acid-deficient mutant of *Salmonella typhimurium*. Studies by ^{1}H, ^{13}C, and ^{31}P nuclear magnetic resonance. J. Biol. Chem. **260**:16089–16098.
466. **Strauch, K. L., T. H. Carter, and C. G. Miller.** 1983. Overproduction of *Salmonella typhimurium* peptidase T. J. Bacteriol. **156**:743–751.
467. **Strauch, K. L., J. B. Lenk, B. L. Gamble, and C. G. Miller.** 1985. Oxygen regulation in *Salmonella typhimurium*. J. Bacteriol. **161**:673–680.
468. **Strauch, K. L., and C. G. Miller.** 1983. Isolation and characterization of *Salmonella typhimurium* mutants lacking a tripeptidase (peptidase T). J. Bacteriol. **154**:763–771.
469. **Sukupolvi, S., I. M. Helander, R. Hukari, M. Vaara, and P. H. Mäkelä.** 1985. Lipopolysaccharides of three classes of supersensitive mutants of *Salmonella typhimurium*. FEMS Microbiol. Lett. **30**:341–345.
470. **Sukupolvi, S., and D. O'Connor.** 1987. Amino acid alterations in a hydrophobic region of the TraT protein of R6-5 increase the outer membrane permeability of enteric bacteria. Mol. Gen. Genet. **210**:178–180.
471. **Sukupolvi, S., D. O'Connor, and M. F. Edwards.** 1986. The *traT* protein is able to normalize the phenotype of a plasmid-carried permeability mutation of *Salmonella typhimurium*. J. Gen. Microbiol. **132**:2079–2085.
472. **Sukupolvi, S., M. Vaara, I. M. Helander, P. Viljanen, and P. H. Mäkelä.** 1984. New *Salmonella typhimurium* mutants with altered outer membrane permeability. J. Bacteriol. **159**:704–712.
473. **Sutherland, L., J. Cairney, M. J. Elmore, I. R. Booth, and C. F. Higgins.** 1986. Osmotic regulation of transcription: induction of the *proU* betaine transport gene is dependent on accumulation of intracellular potassium. J. Bacteriol. **168**:805–814.
474. **Sverdlov, E. D., N. A. Lisitsyn, S. O. Guryev, and G. S. Monastyrskaia.** 1986. Nucleotide sequence of the *Salmonella typhimurium rpoB* gene encoding the β-subunit of RNA polymerase. Dokl. Akad. Nauk. SSSR **287**:232–236. (Translated by abstracting service.)
475. **Sverdlov, E. D., N. A. Lisitsyn, S. O. Guryev, Y. V. Smirnov, V. M. Rostapshov, and G. S. Monastyrskaya.** 1986. Genes coding the β-subunit of bacterial RNA polymerases. I. Primary structure of the *rpoB* gene EcoRI-C fragment from *Salmonella typhimurium*. Bioorg. Khim. **12**:699–707. (In Russian with abstract in English.)
476. **Sweet, G. D., C. M. Kay, and W. W. Kay.** 1984. Tricarboxylate-binding proteins of *Salmonella typhimurium*. Purification, crystallization, and physical properties. J. Biol. Chem. **259**: 1586–1592.
477. **Swenson, P. A., L. Riester, and T. V. Palmer.** 1983. Role of the *supX* gene in sensitizing *Salmonella typhimurium* cells to respiration shutoff induced by far ultraviolet irradiation. Photochem. Photobiol. **38**:305–310.
478. **Szekely, E., and M. Simon.** 1983. DNA sequence adjacent to flagellar genes and evolution of flagellar-phase variation. J. Bacteriol. **155**:74–81.
479. **Taillon, B. E., R. Little, and R. P. Lawther.** 1988. Analysis of the functional domains of biosynthetic threonine deaminase by comparison of the amino acid sequences of three wild-type alleles to the amino acid sequence of biodegradative threonine deaminase. Gene **63**:245–252.
480. **Takayama, K., N. Qureshi, and P. Mascagni.** 1983. Complete structure of lipid A obtained from the lipopolysaccharides of the heptoseless mutant of *Salmonella typhimurium*. J. Biol. Chem. **258**:12801–12803.
481. **Theisen, M., R. A. Kelln, and J. Neuhard.** 1987. Cloning and characterization of the *pyrF* operon of *Salmonella typhimurium*. Eur. J. Biochem. **164**:613–619.
482. **Tirgari, S., M. P. Spector, and J. W. Foster.** 1986. Genetics of NAD metabolism in *Salmonella typhimurium* and cloning of the *nadA* and *pnuC* loci. J. Bacteriol. **167**:1086–1088.
483. **Tittawella, I. P. B.** 1984. Evidence for clustering of RNA polymerase and ribosomal protein genes in six species of Enterobacteria. Mol. Gen. Genet. **195**:215–218.
484. **Tittawella, I. P. B.** 1985. *Salmonella typhimurium rpoB* and *rpoC* genes cloned on λ phages. FEBS Lett. **185**:33–36.
485. **Tribhuwan, R. C., M. S. Johnson, and B. L. Taylor.** 1986. Evidence against direct involvement of cyclic GMP or cyclic AMP in bacterial chemotactic signaling. J. Bacteriol. **168**:624–630.
486. **Turnbough, Jr., C. L., and B. R. Bochner.** 1985. Toxicity of the pyrimidine biosynthetic pathway intermediate carbamyl aspartate in *Salmonella typhimurium*. J. Bacteriol. **163**:500–505.
487. **Urbanowski, M. L., L. S. Plamann, and G. V. Stauffer.** 1987. Mutations affecting the regulation of the *metB* gene of *Salmonella typhimurium* LT2. J. Bacteriol. **169**:126–130.
488. **Urbanowski, M. L., M. D. Plamann, L. T. Stauffer, and G. V. Stauffer.** 1984. Cloning and characterization of the gene for *Salmonella typhimurium* serine hydroxymethyltransferase. Gene **27**:47–54.
489. **Urbanowski, M. L., and G. V. Stauffer.** 1985. Nucleotide sequence and biochemical characterization of the *metJ* gene from *Salmonella typhimurium* LT2. Nucleic Acids Res. **13**: 673–685.
490. **Urbanowski, M. L., and G. V. Stauffer.** 1985. Cloning and initial characterization of the *metJ* and *metB* genes from *Salmonella typhimurium* LT2. Gene **35**:187–197.
491. **Urbanowski, M. L., and G. V. Stauffer.** 1986. The *metH* gene from *Salmonella typhimurium* LT2: cloning and initial characterization. Gene **44**:211–217.
492. **Urbanowski, M. L., and G. V. Stauffer.** 1986. Autoregulation by tandem promoters of the *Salmonella typhimurium* LT2 *metJ* gene. J. Bacteriol. **165**:740–745.
493. **Urbanowski, M. L., and G. V. Stauffer.** 1987. Regulation of the *metR* gene of *Salmonella typhimurium*. J. Bacteriol. **169**:5841–5844.
494. **Urbanowski, M. L., L. T. Stauffer, L. S. Plamann, and G. V. Stauffer.** 1987. A new methionine locus, *metR*, that encodes a *trans*-acting protein required for activation of *metE* and *metH* in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **169**:1391–1397.
495. **Vaara, M., P. Viljanen, S. Sukupolvi, and T. Vaara.** 1985. Does polymyxin B nonapeptide increase outer membrane permeability in antibiotic supersensitive enterobacterial mutants? FEMS Microbiol. Lett. **26**:289–294.
496. **Vandenbosch, J. L., D. K. Rabert, and G. W. Jones.** 1987. Plasmid-associated resistance of *Salmonella typhimurium* to complement activated by the classical pathway. Infect. Immun. **55**:2645–2652.
497. **Van Dyk, T. K., and R. A. LaRossa.** 1986. Sensitivity of a *Salmonella typhimurium aspC* mutant to sulfometuron methyl, a potent inhibitor of acetolactate synthase II. J. Bacteriol. **165**: 386–392.
498. **Van Dyk, T. K., and R. A. LaRossa.** 1987. Involvement of *ack-pta* operon products in alpha-ketobutyrate metabolism by *Salmonella typhimurium*. Mol. Gen. Genet. **207**:435–440.
499. **Van Dyk, T. K., D. R. Smulski, and Y.-Y. Chang.** 1987. Pleiotropic effects of *poxA* regulatory mutations of *Escherichia coli* and *Salmonella typhimurium*, mutations conferring sulfometuron methyl and alpha-ketobutyrate hypersensitivity. J. Bacteriol. **169**:4540–4546.

500. **Van Gijsegem, F., and A. Toussaint.** 1982. Chromosome transfer and R-prime formation by an RP4::mini-Mu derivative in *Escherichia coli*, *Salmonella typhimurium*, *Klebsiella pneumoniae*, and *Proteus mirabilis*. Plasmid **7**:30–44.
501. **Verma, N. K., N. B. Quigley, and P. R. Reeves.** 1988. O-antigen variation in *Salmonella* spp.: *rfb* gene clusters of three strains. J. Bacteriol. **170**:103–107.
502. **Vogel, W., and H. Schmieger.** 1986. Selection of bacterial *pac* sites recognized by *Salmonella* phage P22. Mol. Gen. Genet. **205**:563–567.
503. **Wasserman, S. A., E. Daub, P. Grisafi, D. Botstein, and C. T. Walsh.** 1984. Catabolic alanine racemase from *Salmonella typhimurium*: DNA sequence, enzyme purification, and characterization. Biochemistry **23**:5182–5187.
504. **Waxman, L., and A. L. Goldberg.** 1985. Protease La, the *lon* gene product, cleaves specific fluorogenic peptides in an ATP-dependent reaction. J. Biol. Chem. **260**:12022–12028.
505. **Way, J. C., M. A. Davis, D. Morisato, D. E. Roberts, and N. Kleckner.** 1984. New Tn*10* derivatives for transposon mutagenesis and for construction of *lacZ* operon fusions by transposition. Gene **32**:369–379.
506. **Waygood, E. B., B. Reiche, W. Hengstenberg, and J. S. Lee.** 1987. Characterization of mutant histidine-containing proteins of the phosphoenolpyruvate:sugar phosphotransferase system of *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **169**:2810–2818.
507. **Wei, L.-N., and T. M. Joys.** 1985. Covalent structure of three phase-1 flagellar filament proteins of *Salmonella*. J. Mol. Biol. **186**:791–803.
508. **Wei, L.-N., and T. M. Joys.** 1986. The nucleotide sequence of the H-1r gene of *Salmonella rubislaw*. Nucleic Acids Res. **14**: 8227.
509. **Weigel, N., M. A. Kukuruzinska, A. Nakazawa, E. B. Waygood, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Phosphoryl transfer reactions catalyzed by enzyme I of *Salmonella typhimurium*. J. Biol. Chem. **257**:14477–14491.
510. **Weigel, N., D. A. Powers, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Primary structure and active site of a general phosphocarrier protein (HPr) from *Salmonella typhimurium*. J. Biol. Chem. **257**: 14499–14509.
511. **Weigel, N., E. B. Waygood, M. A. Kukuruzinska, A. Nakazawa, and S. Roseman.** 1982. Sugar transport by the bacterial phosphotransferase system. Isolation and characterization of enzyme I from *Salmonella typhimurium*. J. Biol. Chem. **257**: 14461–14469.
512. **Weinberg, R. A., and R. O. Burns.** 1984. Regulation of expression of the *ilvB* operon in *Salmonella typhimurium*. J. Bacteriol. **160**:833–841.
513. **West, T. P., T. W. Traut, M. S. Shanley, and G. A. O'Donavan.** 1985. A *Salmonella typhimurium* strain defective in uracil catabolism and beta-alanine synthesis. J. Gen. Microbiol. **131**: 1083–1090.
514. **Whalen, W. A., and C. M. Berg.** 1984. Gratuitous repression of *avtA* in *Escherichia coli* and *Salmonella typhimurium*. J. Bacteriol. **158**:571–574.
515. **Widenhorn, K. A., W. Boos, J. M. Somers, and W. W. Kay.** 1988. Cloning and properties of the *Salmonella typhimurium* tricarboxylate transport operon in *Escherichia coli*. J. Bacteriol. **170**:883–888.
516. **Widenhorn, K. A., J. M. Somers, and W. W. Kay.** 1988. Expression of the divergent tricaryboxylate transport operon (*tctI*) of *Salmonella typhimurium*. J. Bacteriol. **170**:3223–3227.
517. **Wilson, R. B., and S. R. Maloy.** 1987. Isolation and characterization of *Salmonella typhimurium* glyoxylate shunt mutants. J. Bacteriol. **169**:3029–3034.
518. **Wollin, R., E. S. Creeger, L. I. Rothfield, B. A. D. Stocker, and A. A. Lindberg.** 1983. *Salmonella typhimurium* mutants defective in UDP-D-galactose:lipopolysaccharide α1,6-D-galactosyltransferase. Structural, immunochemical, and enzymologic studies of *rfaB* mutants. J. Biol. Chem. **258**:3769–3774.
519. **Wong, A., L. Kean, and R. Maurer.** 1988. Sequence of the *dnaB* gene of *Salmonella typhimurium*. J. Bacteriol. **170**:2668–2675.
520. **Wu, T. T.** 1966. A model for three-point analysis of random general transduction. Genetics **54**:405–410.
521. **Wylie, D., A. Stock, C.-Y. Wong, and J. Stock.** 1988. Sensory transduction in bacterial chemotaxis involves phosphotransfer between *che* proteins. Biochem. Biophys. Res. Commun. **151**: 891–896.
522. **Yamaguchi, S., S.-I. Aizawa, M. Kihara, M. Isomura, C. J. Jones, and R. M. Macnab.** 1986. Genetic evidence for a switching and energy-transducing complex in the flagellar motor of *Salmonella typhimurium*. J. Bacteriol. **168**:1172–1179.
523. **Yamaguchi, S., H. Fujita, A. Ishihara, S.-I. Aizawa, and R. M. Macnab.** 1986. Subdivision of flagellar genes of *Salmonella typhimurium* into regions responsible for assembly, rotation, and switching. J. Bacteriol. **166**:187–193.
524. **Yamaguchi, S., H. Fujita, K. Sugata, T. Taira, and T. Iino.** 1984. Genetic analysis of H2, the structural gene for phase-2 flagellin in *Salmonella*. J. Gen. Microbiol. **130**:255–265.
525. **Yamaguchi, S., H. Fujita, T. Taira, K. Kutsukake, M. Homma, and T. Iino.** 1984. Genetic analysis of three additional *fla* genes in *Salmonella typhimurium*. J. Gen. Microbiol. **130**:3339–3342.
526. **Yamamoto, N., and M. L. Droffner.** 1985. Mechanisms determining aerobic or anaerobic growth in the facultative anaerobe *Salmonella typhimurium*. Proc. Natl. Acad. Sci. USA **82**:2077–2081.
527. **Yang, Y.-L., D. Goldrick, and J.-S. Hong.** 1988. Identification of the products and nucleotide sequences of two regulatory genes involved in the exogenous induction of phosphoglycerate transport in *Salmonella typhimurium*. J. Bacteriol. **170**: 4299–4303.
528. **Yanisch-Perron, C., J. Vieira, and J. Messing.** 1985. Improved M13 phage cloning vectors and host strains: nucleotide sequences of M13mp18 and pUC19 vectors. Gene **33**:103–119.
529. **Youderian, P., P. Sugiono, K. L. Brewer, N. P. Higgins, and T. Elliott.** 1988. Packaging specific segments of the *Salmonella* chromosome with locked-in Mu*d*-P22 prophages. Genetics **118**:581–592.
530. **Yu, C.-Q., and J.-S. Hong.** 1986. Identification and nucleotide sequence of the activator gene of the externally induced phosphoglycerate transport system of *Salmonella typhimurium*. Gene **45**:51–57.
531. **Yutani, K., T. Sato, K. Ogasahara, and E. W. Miles.** 1984. Comparison of denaturation of tryptophan synthase alpha-subunits from *Escherichia coli*, *Salmonella typhimurium*, and an interspecies hybrid. Arch. Biochem. Biophys. **229**:448–454.
532. **Zaror, I., I. Gomez, G. Castillo, A. Yudelevich, and A. Venegas.** 1988. Molecular cloning and expression in *E. coli* of a *Salmonella typhi* porin gene. FEBS Lett. **229**:77–81.
533. **Zhu, N., B. M. Olivera, and J. R. Roth.** 1988. Identification of a repressor gene involved in the regulation of NAD de novo biosynthesis in *Salmonella typhimurium*. J. Bacteriol. **170**:117–125.
534. **Zinder, N. D.** 1960. Sexuality and mating in *Salmonella*. Science **131**:924–926.
535. **Zukin, R. S., M. F. Klos, and R. E. Hirsch.** 1986. Conformational dynamics of two histidine-binding proteins of *Salmonella typhimurium*. Biophys. J. **49**:1229–1235.