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Linkage Map of *Salmonella typhimurium*, Edition VI

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INTRODUCTION

We present the sixth edition of the linkage map of *Salmonella typhimurium*, comprising 532 genes. The first edition of the map contained 133 genes (383), and subsequent editions were in 1967 (380), 1970 (381), 1972 (382), and 1978 (385). All genes presently known to us are included on the linkage map (Fig. 1) and are listed in Table 1, but references presented in earlier editions of the map are not normally included. Alternative gene symbols are listed in Table 2.

In addition, we review some new methods and materials for genetic analysis which are presently available for use in *S. typhimurium*.

THE LINKAGE MAP

The basic structure of early editions of the map was based on interrupted conjugation experiments with Hfr strains, which allowed the placement of P22 transduction linkage groups around a closed circle of 138 min. As more data accumulated on transduction groups, genes placed at nearby locations were found to be linked by transduction. In the fifth edition the map was modified in two ways. First, we changed the map to 100 min, corresponding to the 100-min time-of-entry *Escherichia coli* K-12 map, rather than retaining the 138-min map determined originally in *S. typhimurium*. This was done to facilitate comparisons with the similar linkage map of the closely related species *E. coli*, and because the "minute of entry time," as a measure of map distance, has become less important as measures of transduction frequency and, more recently, of DNA length have become available. Second, the linkage map was presented as 10 linear 10-min intervals, for convenience of display.

We have retained the basic system used in the fifth edition in this version. The 100 min of the map are based on P22 "phage lengths." P22 can encapsulate 26 to 27 megadaltons of DNA (357, 402); this is estimated to be about 1/100 of the *Salmonella* chromosome. The linear distance between genes is calculated from P22 cotransduction data by a formula derived by Kemper (212), $c = 1 - t + (t \times \ln t)$, where c = cotransduction frequency and t = the linear distance between genes measured as the fraction of the length of the DNA of a transducing particle. Phages KB1 and ES18, like P22, were also assigned a 1-min length, but as in *E. coli* the P1 transducing fragment was assigned a 2-min length (24).

The formula used by Bachmann (24) to calculate the *E. coli* map distances, devised by Wu (490), is $c = (1 - d/L)^3$, where c = cotransduction frequency, d = the distance between markers in minutes, and L is the length of the transducing fragment in minutes. The two formulas produce similar estimates of map intervals from cotransduction data. However, where the cotransduction frequency for phage P22 or phage P1 is greater than ca. 30%, the Wu formula estimates slightly longer intervals; when it is less than ca. 30%, the Kemper formula estimates slightly longer intervals. In the *S. typhimurium* map, the net effect of the use of the Kemper formula is to estimate slightly longer transduction linkage groups than would be produced from the Wu formula, but this influence is probably considerably less than other sources of error or variation in the data. Since calculating these distances, we have analyzed the two means of estimating distance. In our opinion, Wu's is the better of the two methods. Kemper's method ignores fragments ending between the markers and capable of carrying only one marker. For

markers that are far apart, this class of fragment will contribute heavily to the total number of selected recombinants and to the calculated cotransduction frequency.

These methods of estimating physical distance from cotransduction frequency assume that the entire length of the transduced fragment is homologous to a chromosomal equivalent, that these fragments are chosen at random, and that recombination is equally probable along this length. When insertion mutations are present in the donor and when deletion mutations are present in the recipient, a segment of the fragment is not homologous to the host chromosome. In these cases it is probably easiest to estimate distance by using a fragment size reduced by the amount of nonhomologous material carried in the donor fragment. Thus, the fragment size used again represents the length of material available for recombination. This is a crude approximation since it does not account for fragments that end within the nonhomologous sequences.

The linkage maps of *E. coli* and *S. typhimurium* show overall homology of gene location, which supported the decision to display the linkage map of *S. typhimurium* as 100 min. Certain map differences have been noted for some years, and these were discussed earlier (385). As analysis of the map intervals becomes more detailed, a larger number of differences may be found. These may turn out to represent the insertion of noncoding sequences into the chromosomes of the two genera at different locations. The following are known or probable differences between the linkage maps.

(i) The gene segment in the ca. 25- to 35-min regions of the linkage maps is inverted between *E. coli* and *S. typhimurium* (77, 382); this has been discussed in detail (385).

(ii) There is no *lac* gene activity in *S. typhimurium*. Lampel and Riley (250) have shown that genetic material corresponding to the *lac* genes is missing from *S. typhimurium*.

In a few cases, genes indicated to have similar phenotypes are given the same name, but are shown at very different locations for the two genera (24; this paper). These include the following: *cod*, cytosine deaminase—69 min on the *S. typhimurium* map, 94 min in *E. coli*; *pck*, phosphoenolpyruvate carboxykinase—13 and 75 min; *pheR*, regulatory gene for *pheA*—64 and 94 min; *pil*, type I somatic pili—14 and 98 min; *ush*, UDP-glucose hydrolase—90 and 11 min. It is possible that these represent homologous genes at different map locations, but it is more likely that they will be found to be nonhomologous genes which affect the same phenotype or homologous genes incorrectly mapped but actually at the same map location.

MATERIALS AND METHODS FOR GENETIC ANALYSIS

A variety of genetic techniques directly applicable to *Salmonella* spp. have been reviewed elsewhere (294, 369, 385). Some recent developments are described below.

Insertion Mutations

Since the last edition of this map, use of transposons as genetic markers has increased greatly. Currently, insertion mutations are available in virtually all of the standard auxotrophic loci (Table 3). In addition, insertion mutations have been isolated near many regions of the chromosome (Table 4). We have tried to compile a list of these mutations to give workers an idea of what material is available. These lists include mutations isolated in our laboratories and those submitted by many other workers. We have not made an exhaustive literature search, so some mutations have certainly been missed. However, the lists presented represent a start, which can be improved and expanded in future editions of the map. We solicit suggested changes and additions. Cultures of these strains are available from the laboratories in which they originated or from K. E. Sanderson, Salmonella Genetic Stock Centre, University of Calgary.

Uses of Transposons

Methods for use of transposable drug resistance elements have been outlined previously (226) and have been updated somewhat since the previous edition of the map (80, 370). Therefore, we discuss here applications which have not had wide use or for which more recent results are available.

(i) Addition of selective markers to plasmids is frequently very useful, especially when dealing with F′ plasmids carrying chromosome regions having few useful selective markers. Insertions of Tn*10* in F-factor sequences have been isolated which do not impair transfer ability or stability of the plasmid (81). The sequences in which these insertions lie share homology with all F′ plasmids regardless of the chromosomal region carried. Thus, the tetracycline resistance phenotype can be added selectively to any F′ plasmid simply by transducing a strain carrying the plasmid to drug resistance. The transduced Tn*10* element is inherited by standard recombination events and is introduced exclusively into the recipient plasmid.

(ii) Use of Tn*10* homology to direct F*lac* insertion and generate an Hfr origin of transfer has proven to be the most rapid and least ambiguous means of placing new genes on the chromosomal linkage map. A Tn*10* insertion is placed in or near the new gene by methods

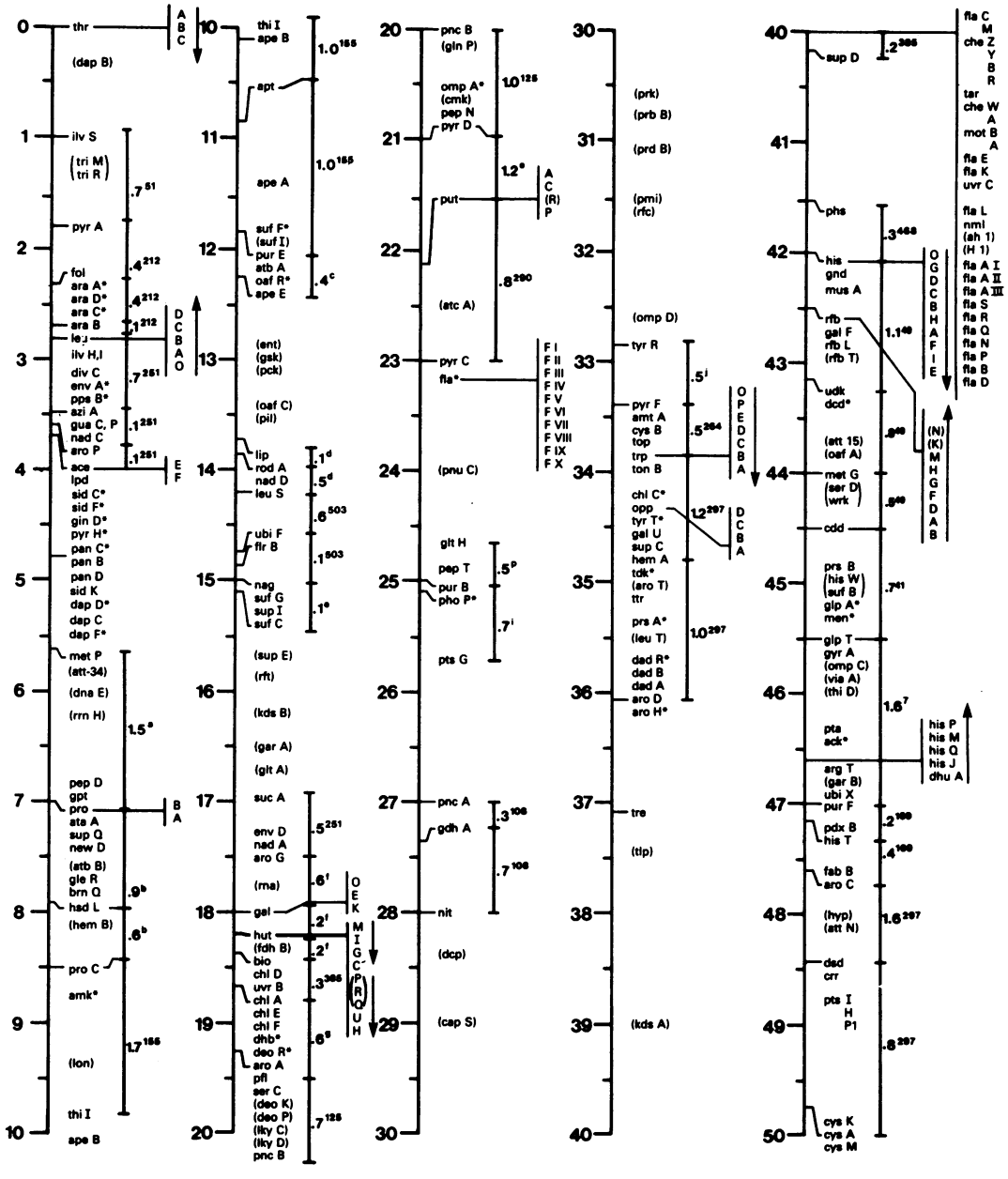

FIG. 1.

described previously (226). An F'*lac* strain carrying a Tn*10* insertion and a temperature-sensitive mutation affecting plasmid replication is transferred into the new mutant. By selection for a derivative that remains Lac$^+$ at 42°C, one selects for Hfr strains which occur by insertion of the plasmid at the chromosomal Tn*10* homology. The origin of transfer of this Hfr strain is at the site of the chromosomal Tn*10* element. Origins of Hfr transfer are easily located by simple plate matings with a series of standard recipient auxotrophs, scoring the gradient of transmission frequencies (81).

(iii) The outward-directed promoters associated with Tn*10* and Tn*5* have provided both a problem and a new use of transposons (44, 47; R. Simons, W. Hoppes, W. McClure, and N. Kleckner, manuscript in preparation). One standard use of these elements has been to provide an absolute block to transcription in operons and

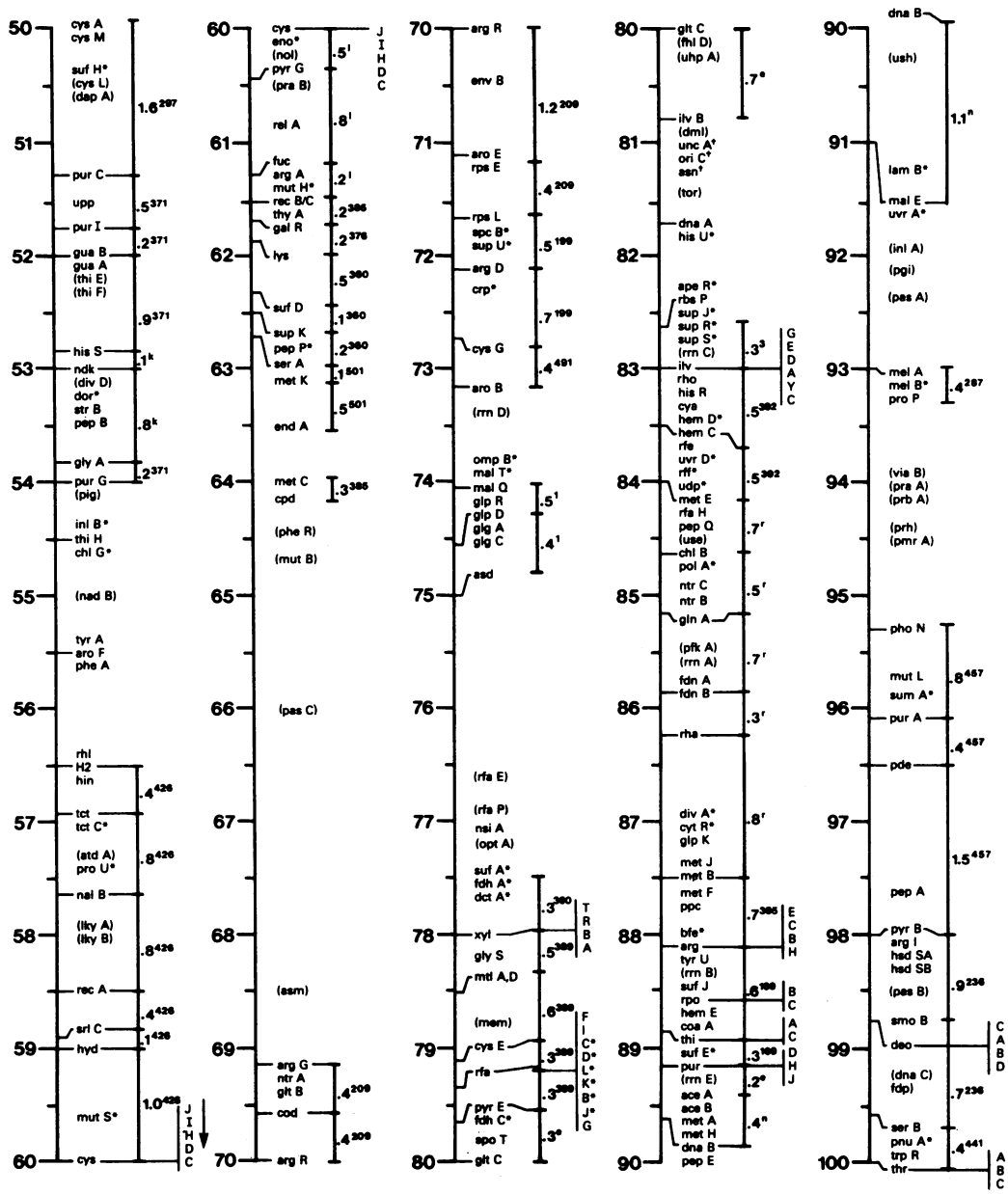

FIG. 1.—*Continued*

FIG. 1. Linkage map of *S. typhimurium*, represented as 10 segments. The scale of 100 min begins at zero for the *thr* loci, as in previous maps (385) and in the linkage map of *E. coli* K-12 (24). The segmented line to the right of the gene symbols 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 frequency of joint transduction and was calculated assuming that the length of P22, KB1, and ES18 transducing fragments is 1 min, whereas that of P1 phage is 2 min, and applying the formula developed by Kemper (212) to convert the percentage of joint transduction to map distance. The superscript to the number indicating the linear distance is the reference giving the data used for calculation: a number refers to Literature Cited and a letter indicates a personal communication, given below. 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 marker 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. The following letters indicate superscripts to the length of the gene interval and show that the interval is based on a personal communication from the following persons: a, T. Mojica-a and P. D. Ayling; b, C. Dignette and C. Colson; c, P. C. Osdoby and C. G. Miller; d, D. Antón; e, J. Roth; f, W. Brill; g, M.-C. Pascal; h, K. L. Strauch and C. G. Miller; i, P. Postma; j, D. Sprinson; k, S. Rodriguez; l, Stephens; m, E. Barrett; n, H. Schmieger; o, J. Foster; p, E. Barrett.

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	Acetate requirement; isocitrate lyase (EC 4.1.3.1)	*aceA*	89	C
aceB	Acetate	Acetate requirement; malate synthase (EC 4.1.3.2)	*aceB*	89	C
aceE	Acetate	Acetate requirement; pyruvate dehydrogenase (pyruvate:cytochrome b_1 oxidoreductase; EC 1.2.2.2)	*aceE*	3	B
aceF	Acetate	Acetate requirement; pyruvate dehydrogenase (pyruvate:lipoate oxidoreductase; EC 1.2.4.1)	*aceF*	3	B
ack		Acetate kinase (ATP:acetate phosphotransferase; E.C.2.7.2.1)	*ackA*	46	267
amk		AMP kinase		9	B
amtA		Resistance to 40 mM 3-amino-1,2,4-triazole in presence of histidine		33	A, B
apeA	Acyl peptide esterase	Acyl amino acid esterase (hydrolyzes *N*-acetyl-L-phenylalanine-β-naphthyl ester)		11	B, 155
apeB	Acyl peptide esterase	Acyl amino acid esterase (hydrolyzes *N*-acetyl-L-phenylalanine-β-naphthyl ester)		10	B, 155
apeE	Acyl peptide esterase	Membrane-bound acyl amino acid esterase, hydrolyzes *N*-acetyl-L-phenylalanine-β-naphthyl ester		12	V
apeR	Acyl peptide esterase	*apeD*; regulatory gene for *apeE*		82	V
aphA		Gene for nonspecific acid phosphatase II		NM	457
apt		Adenine phosphoribosyltransferase		10	B
araA	Arabinose	L-Arabinose isomerase (EC 5.3.1.4)	*araA*	2	B, 171, 257, 351
araB	Arabinose	Ribulokinase (EC 2.7.1.16)	*araB*	2	A, B, 171, 351
araC	Arabinose	Regulatory gene for arabinose catabolic enzymes	*araC*	2	B, 86, 171, 257, 351
araD	Arabinose	L-Ribulosephosphate 4-epimerase (EC 5.1.3.4)	*araD*	2	171, 350, 351, 374
argA	Arginine	*argB*; amino acid acetyltransferase (EC 2.3.1.1)	*argA*	61	A, B
argB	Arginine	*argC*; *N*-acetyl-γ-glutamate kinase (EC 2.7.2.8)	*argB*	88	A, B
argC	Arginine	*argH*; *N*-acetyl-γ-glutamyl phosphate reductase (EC 1.2.1.38)	*argC*	88	A, B
argD	Arginine	*argG*; acetylornithine aminotransferase (EC 2.6.1.11)	*argD*	72	A, B
argE	Arginine	*argA*; acetylornithine deacetylase (EC 3.5.1.16)	*argE*	88	A, B
argG	Arginine	*argE*; argininosuccinate synthetase (EC 6.3.4.5)	*argG*	69	A, B
argH	Arginine	*argF*; argininosuccinate lyase (EC 4.3.2.1)	*argH*	88	A, B
argI	Arginine	Ornithine carbamoyltransferase (EC 2.1.3.3)	*argI*	98	A, B, 74
argP	Arginine	Arginine transport	*argP*	NM	B
argR	Arginine	L-Arginine regulation	*argR*	70	B, 210
argS	Arginine	Arginyl-tRNA synthetase (EC 6.1.1.19)	*argS*	NM	B
argT	Arginine	Lysine-arginine-ornithine binding protein		46	19, 20, 158, 159, 242
aroA	Aromatic	3-Enolpyruvylshikimate 5-phosphate synthetase	*aroA*	19	A, B, 166, 167
aroB	Aromatic	5-Dehydroquinate synthetase	*aroB*	73	A, B
aroC	Aromatic	*aroD*; chorismate synthetase	*aroC*	47	A, B, 238

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]
aroD	Aromatic	*aroE*; 5′-dehydroquinate dehydratase (EC 4.2.1.10)	*aroD*	36	A, B, 16
aroE	Aromatic	*aroC*; 5-dehydroshikimate reductase	*aroE*	71	A, B
aroF	Aromatic	Tyrosine-repressible DAHP synthetase	*aroF*	55	A, B, 429
aroG	Aromatic	Phenylalanine-repressible DAHP synthetase	*aroG*	17	A, B, 429
aroH	Aromatic	Tryptophan-repressible DAHP synthetase	*aroH*	36	A, B
aroP	Aromatic	Aromatic amino acid transport	*aroP*	3	A, B
aroT	Aromatic	Ability to transport tryptophan, phenylalanine, tyrosine	*aroT*	35	A, B
asd		Aspartate-semialdehyde dehydrogenase (EC 1.2.1.11)	*asd*	75	A, B
asm		Unable to assimilate low levels of ammonia; deficient in glutamate synthase and glutamine synthase		68	108, 136
asn	Asparagine	Asparagine synthesis	*asn*	81	195, 507, 508
ataA		*attP22 I*; attachment site for prophage P22	*attP22*	7	A, B, 248, 494
atbA		*attP27 I*; attachment site for prophage P27		12	A, B, 272
atbB		*attP27 II*; second attachment site for prophage P27		7	A, B
atcA		*attP221*; attachment site for prophage P221		22	A, B
atdA		*attP6*$_1$, *attP14*; attachment site for prophage P6$_1$ or P14 in group C *Salmonella* sp.		57	A, B
ats		Arylsulfatase		NM	304, 492
attN		Attachment site for prophage N in *S. montevideo*		48	B
att15	Attachment	Attachment site of phage ε^{15} to chromosome of group E *Salmonella* sp.		43	B
att34	Attachment	Attachment site of phage ε^{34} to chromosome of group E *Salmonella* sp.		5	B
aziA	Azide	Resistant to 3 mM sodium azide on L-methionine	*azi*	3	A, B, 120
bfe		Adsorption of phage BF23 and colicin E	*bfe*	88	B, 298
bio	Biotin	Requirement	*bioA*	18	B
brnQ		*ilvT*; branched-chain amino acid transport	*brnQ*	7	B, 322
capS	Capsule	Capsular polysaccharide synthesis	*capS*	29	391
cdd		Cytidine deaminase (EC 3.5.4.5)	*cdd*	44	A, B
cheA	Chemotaxis	*cheP*; chemotaxis	*cheA*	40	B, 105
cheB	Chemotaxis	*cheX*; chemotaxis; protein-glutamate methylesterase	*cheB*	40	B, 103–106, 140, 213, 253
cheR	Chemotaxis	Chemotaxis; protein-glutamate methyl transferase	*cheX*	40	B, 87, 103–106, 253
cheS	Chemotaxis	Chemotaxis		NM	B, 253
cheW	Chemotaxis	Chemotaxis	*cheW*	40	B, 105, 253, 420
cheY	Chemotaxis	*cheQ*; chemotaxis	*cheY*	40	B, 103–106, 213, 253
cheZ	Chemotaxis	*cheT*; chemotaxis	*cheZ*	40	103–106, 140, 253, 437, 448

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]
chlA	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase	*chlA*	18	A, B
chlB	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, and hydrogen lyase	*chlB*	84	A, B
chlC	Chlorate	Affects nitrate reductase; retains sensitivity to chlorate	*chlC*	34	A, B, 36, 361
chlD	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase	*chlD*	18	A, B
chlE	Chlorate	Resistance		18	A, B
chlF	Chlorate	Resistance		18	A, B
chlG	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase		54	A, B
cmk		Cytidylate kinase (EC 2.7.4.14)		20	B
coaA	Coenzyme A	Pantothenate kinase; coenzyme A synthesis		89	B, 113
cod		Cytosine deaminase (EC 3.5.4.1)		69	A, B, 478
cpd		cAMP phosphodiesterase (EC 3.1.4.17)		64	B, D
crp		cAMP receptor protein	*crp*	72	A, B, 310, 404
crr		Factor III for sugar transport by phosphotransferase IIB′ (*ptsG*) system	*crr*	48	A, B, 119, 310, 344, 406, 407, 418
cya	cAMP	Adenylate cyclase (EC 4.6.1.1)	*cya*	83	A, B, 471
cysA	Cysteine	Sulfate-thiosulfate transport (chromate resistance)	*cysA*	49	A, B, 34, 180, 206, 480
cysB	Cysteine	Cysteine regulation; positive control of L-cystine transport	*cysB*	33	A, B, 28, 34, 120, 180, 187, 330, 331, 423, 479
cysC	Cysteine	Adenylylsulfate kinase (EC 2.7.1.25)	*cysC*	60	A, B, 34, 218–220
cysD	Cysteine	Sulfate adenylyltransferase (EC 2.7.7.4)	*cysD*	60	A, B, 34, 218–220
cysE	Cysteine	Serine acetyltransferase (EC 2.3.1.30)	*cysE*	79	A, B, 34, 85, 181, 238, 331
cysG	Cysteine	Seroheme component of sulfite reductase	*cysG*	72	A, B, 34
cysH	Cysteine	Adenylylsulfate reductase (EC 1.8.99.2)	*cysH*	60	A, B, 34, 218–220, 330
cysI	Cysteine	Heme protein component of sulfite reductase	*cysI*	60	B, 34, 218–220, 330
cysJ	Cysteine	Flavoprotein component of sulfite reductase	*cysJ*	60	A, B, 34, 218–220, 330
cysK	Cysteine	*aziB*, *trzA*; *O*-acetylserine sulfhydrylase-A (resistance to 1,2,4-triazole) (EC 4.2.99.8)	*cysK*	49	A, B, 85, 120, 121, 180, 480
cysL	Cysteine	Resistance to selenate		50	B
cysM	Cysteine	*O*-Acetylserine sulfhydrylase-B (EC 4.2.99.8)		49	121, 180, 479
cytR		Regulatory gene for *deo* operon and *udp* and *cdd* genes	*cytR*	87	A, B, 237

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]
dadA		*dad*; D-alanine dehydrogenase (EC 1.4.99.1) (D-histidine, D-methionine utilization)	*dadA*	36	A, B, 472
dadB		Catabolic alanine racemase (EC 5.1.1.1)	*dadB*	36	472
dadR		Insensitivity of *dadA* to catabolite repression		36	B, 481
dam		DNA adenine methylation	*dam*	NM	148
dapA	Diaminopimelate	Dihydropicolinate synthase (EC 4.2.1.52)	*dapA*	50	A, B
dapB	Diaminopimelate	Dihydropicolinate reductase	*dapB*	0	A, B
dapC	Diaminopimelate	Tetrahydropicolinate succinylase	*dapC*	5	A, B
dapD	Diaminopimelate	Succinyl-diaminopimelate aminotransferase (EC 2.6.1.17)	*dapD*	5	A, B
dapF	Diaminopimelate	Diaminopimelate epimerase (EC 5.1.1.7)		5	A, B
dcd		dCTP deaminase (EC 3.5.4.13)	*dcd*	43	B
dcm		DNA cytosine methylation	*dcm*	NM	148
dcp		Dipeptidyl carboxypeptidase	*dcp*	28	466
dctA		Transport of dicarboxylic acids	*dctA*	77	B, 207, 208
deoA	Deoxyribose	*tpp*; thymidine phosphorylase (EC 2.4.2.4)	*deoA*	99	A, B, 190
deoB	Deoxyribose	*drm*; phosphopentomutase (EC 2.7.5.6)	*deoB*	99	A, B, 190
deoC	Deoxyribose	*dra*; phosphodeoxyriboaldolase (EC 4.1.2.4)	*deoC*	99	A, B, 190
deoD	Deoxyribose	*pnu*, *pup*; purine nucleoside phosphorylase (EC 2.4.2.1)	*deoD*	99	A, B, 190
deoK	Deoxyribose	Deoxyribokinase		20	A, B
deoP	Deoxyribose	Deoxyribose transport		20	A, B
deoR	Deoxyribose	Constitutive for enzymes of *deoA*, *-B*, *-C*, and *-D*	*deoR*	19	A, B, 141
dhb		2,3-Dihydroxybenzoic acid requirement		19	B, 35
dhuA	D-Histidine	Utilization; increased activity of histidine-binding protein J		46	A, B, 5, 6, 20, 159, 254
divA	Division	*wrkA*; septum initiation defect		87	A, B, 394
divC	Division	*smoA*; septum initiation defect		3	A, B, 17
divD	Division	Round cell morphology		53	17
dml	D-Malate	Utilization		80	A, B
dnaA	DNA	DNA initiation	*dnaA*	81	B, 25, 26, 176, 195, 284
dnaB	DNA	DNA synthesis	*dnaB*	89	284
dnaC	DNA	DNA synthesis initiation and cell division uncoupling	*dnaC*	99	A, B, 263, 284
dnaE	DNA	DNA synthesis	*dnaE*	6	284
dnaG	DNA	DNA biosynthesis (DNA primase)	*dnaG*	NM	395
dor		Deletion of *r*-determinants from plasmids		53	473
dsd		D-Serine dehydratase (D-serine sensitivity) (EC 4.2.1.14)	*dsd*	48	A, B
dum		dUMP synthesis		NM	B
eca		Enterobacterial common antigen synthesis		NM	B, 8, 66, 110, 111, 306, 362, 445, 484
endA		Endonuclease I	*endA*	63	408, 501
eno	Enolase	Enolase (EC 4.2.1.11)	*eno*	60	142
ent	Enterochelin	*enb*, *asc*; enterochelin (dihydroxybenzoylserine trimer)	*ent*	13	A, B, 495

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]
envA	Envelope	Cell division defect; chain formation	*envA*	3	B, 138
envB	Envelope	*bac*; spherical cells; drug sensitivity	*envB*	70	A, B, 16, 18
envD	Envelope	Autolysis; drug sensitivity; alterations in cell morphology		17	B, 17
fabB	Fatty acid biosynthesis	β-Ketoacyl acyl carrier protein synthetase I (EC 2.3.1.41)	*fabB*	47	A, B
fdhA		Formate hydrogenlyase complex; formate dehydrogenase		77	A, B, 35
fdhB		Formate hydrogenlyase complex; formate dehydrogenase		18	B, 35
fdhC		Formate dehydrogenase associated with both hydrogenase and nitrate reductase		80	35
fdnA		Formate dehydrogenase associated with nitrate reductase		85	35, 37
fdnB		Formate dehydrogenase associated with nitrate reductase		85	35, 37
fdp		Fructose-1,6-diphosphatase (EC 3.1.3.11)	*fdp*	99	A, B
fhlD		Formate dehydrogenase 2 activity		81	B
flaAI	Flagella	Defect in flagellar synthesis	*flaN*	40	A, B, E, 245
flaAII	Flagella	*motC*, *cheV*; defect in flagellar synthesis and chemotactic ability	*flaB*	40	E, 102, 245, 253
flaAIII	Flagella	Defect in flagellar synthesis	*flaC*	40	A, B, 245, 430
flaB	Flagella	Defect in flagellar synthesis	*flaR*	40	A, B, E
flaC	Flagella	Defect in flagellar synthesis	*flaH*	40	A, B, E, 245
flaD	Flagella	Defect in flagellar synthesis	*flaQ*	40	A, B, E
flaE	Flagella	Defect in flagellar synthesis	*flaI*	40	A, B, 245
flaFI	Flagella	Incomplete flagellar basal bodies	*flaU*	23	B, 245
flaFII	Flagella	Defect in flagellar synthesis	*flbA*	23	B
flaFIII	Flagella	Defect in flagellar synthesis	*flaW*	23	B
flaFIV	Flagella	Incomplete flagellar basal bodies	*flaV*	23	B, 245
flaFV	Flagella	Structural gene for the hook protein	*flaK*	23	B, 2, 245, 246
flaFVI	Flagella	Defect in flagellar synthesis	*flaX*	23	B, 245
flaFVII	Flagella	Defect in flagellar synthesis	*flaL*	23	B, 245
flaFVIII	Flagella	Incomplete flagellar basal bodies	*flaY*	23	B, 245
flaFIX	Flagella	Incomplete flagellar basal bodies	*flaM*	23	B, 245
flaFX	Flagella	Defect in flagellar synthesis	*flaZ*	23	B, 245
flaK	Flagella	Defect in flagellar synthesis	*flbB*	40	A, B, 245
flaL	Flagella	No flagella, but hook-basal body complexes detected	*flbD*	40	A, B
flaM	Flagella	Defect in flagellar synthesis	*flaG*	40	A, B, 245
flaN	Flagella	Defect in flagellar synthesis		40	B
flaP	Flagella	Defect in flagellar synthesis		40	A, B
flaQ	Flagella	*cheC*, *cheU*; defect in flagellar synthesis and chemotaxis	*flaA*	40	A, B, 245
flaR	Flagella	Superhooks, mostly without flagellin filament	*flaE*	40	B, 2, 245, 442
flaS	Flagella	Defect in flagellar synthesis	*flaD*	40	B, 245
flaU	Flagella	No flagella, but hook basal-body complexes detected		23	E
flrB	Fluoroleucine resistance	Leucine or isoleucine regulation or both		14	A, B, 133
fol	Folate	Tetrahydrofolate dehydrogenase (folate reductase, trimethoprim resistance) (EC 1.5.1.3)	*folA*	2	A, B

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]
fuc	Fucose	Fucose nonfermenting	*fuc*	61	B
fur		Ferrichrome uptake, regulation of iron uptake; constitutive synthesis of iron-enterochelin and ferrichrome uptake		NM	116
galC	Galactose	Constitutive synthesis of specific galactose permease		NM	B
galE	Galactose	UDP galactose 4-epimerase (EC 5.1.3.2)	*galE*	18	A, B, 144, 147, 154, 276, 303, 318, 338, 349, 396, 489
galF	Galactose	*galE*; modifier of UDP-glucose pyrophosphorylase		42	A, B
galK	Galactose	Galactokinase (EC 2.7.1.6)	*galK*	13	A, B, 176
galO	Galactose	Operator	*galO*	18	A, B
galP	Galactose	Specific galactose permease	*galP*	NM	B, 118, 308
galR	Galactose	Regulation	*galR*	61	A, B
galU	Galactose	Glucose-1-phosphate uridylyltransferase (EC 2.7.7.9)	*galU*	34	A, B
garA	γ resistance	Resistance to γ and UV irradiation; large cells; high RNA and protein content (may be equivalent to *rodA*)		16	182
garB	γ resistance	Resistance to γ and UV irradiation; large cells; high RNA and protein content		46	182
gdhA		Gluamate dehydrogenase (EC 1.4.1.4)	*gdhA*	27	107, 108, 368
gleR		Glycyl-leucine-resistant regulatory gene for transport of branched-chain amino acids		7	321, 322
glgA	Glycogen	Glycogen synthetase (EC 2.4.1.21)	*glgA*	74	B
glgC	Glycogen	Glucose-1-phosphate adenylyltransferase (EC 2.7.7.27)	*glgC*	74	B
glnA		Glutamine synthetase (EC 6.3.1.2)	*glnA*	85	B, 69, 136, 228, 239, 242, 286, 354, 475
glnD		PIIA uridylyltransferase	*glnD*	5	B, 27
glnE	Glutamine	Covalent modification of glutamine synthetase; glutamine synthetase adenylyl transferase (EC 2.7.2.42)		NM	27
glnH	Glutamine	Periplasmic glutamine-binding protein		NM	242
glnP	Glutamine	Glutamine transport (high-affinity system)	*glnP*	20	B, 22
glpA	Glycerol phosphate	Glycerol-3-phosphate dehydrogenase (anaerobic)	*glpA*	45	B
glpD	Glycerol phosphate	Glycerol-3-phosphate dehydrogenase (NAD^+) (EC 1.1.1.8)	*glpD*	74	A, B
glpK	Glycerol phosphate	Glycerol kinase (EC 2.7.1.30)	*glpK*	87	A, B, 355
glpT	Glycerol phosphate	*pgtA*; *sn*-glycerol-3-phosphate transport	*glpT*	45	A, B
glpR	Glycerol phosphate	Regulatory gene for *glpD*, *-K*, and *-T*	*glpR*	74	B
gltA	Glutamate	Requirement	*gltA*	16	A, B
gltB	Glutamate	Glutamate synthase, structural gene	*gltB*	69	130
gltC	Glutamate	Growth on glutamate as sole source of carbon		80	B

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]
gltH	Glutamate	Requirement	*gltH*	25	W
glyA	Glycine	Serine hydroxymethyltransferase (EC 2.1.2.1)	*glyA*	53	A, B, 459
glyS	Glycine	Glycyl-tRNA synthetase (EC 6.1.1.14)	*glyS*	78	B
gnd		Phosphogluconate dehydrogenase (EC 1.1.1.43)	*gnd*	42	A, B, 48, 151
gpd		Glucosamine-6-phosphate deaminase		NM	B
gpsA		*sn*-Glycerol-3-phosphate dehydrogenase [NAD(P)$^+$] (EC 1.1.1.94)		NM	B
gpt		*gxu*; guanine-hypoxanthine phosphoribosyltransferase (EC 2.4.2.8)	*gpt*	6	B, 172
gsk		Guanosine kinase	*gsk*	13	B
guaA	Guanine	GMP synthetase (EC 6.3.4.1)	*guaA*	52	A, B, 141
guaB	Guanine	IMP reductase (EC 1.2.1.14)	*guaB*	52	A, B, 141, 192
guaC		GMP reductase (EC 1.6.6.8)	*guaC*	3	B
guaP	Guanine	Guanine uptake		3	A, B, 43
gyrA	Gyrase	*nalA*; DNA gyrase; resistance or sensitivity to nalidixic acid	*gyrA*	46	B, 331
H1	H antigen	Phase-one flagellar antigen (flagellin)	*hag*	40	A, B, 243, 244, 419–421
H2	H antigen	Phase-two flagellar antigen (flagellin)	None	56	A, B, 183, 243, 244, 417, 419–421, 504
hemA	Heme	δ-Aminolevulinate synthetase (EC 2.3.1.37)	*hemA*	34	A, B, 189, 422
hemB	Heme	Heme deficient	*hemB*	8	A, B
hemC	Heme	Heme deficient; uroporphyrinogen I synthase (EC 4.3.1.8)	*hemC*	83	B
hemD	Heme	Heme deficient; uroporphyrinogen III cosynthase	*hemD*	83	B
hemE	Heme	Accumulates uroporphyrin III	*hemE*	88	B, 109
hin	H inversion	*vh2*; DNA invertase; control of the rate of phase variation	None	56	205, 243, 244, 419, 444, 504–506
hisA	Histidine	*N*-(5′-Phospho-L-ribosylformimino)-5-amino-1-(5′-phosphoribosyl)-4-imidazolecarboxamide isomerase (EC 5.3.1.16)	*hisA*	42	A, B, 48, 82, 151, 230, 232
hisB	Histidine	Imidazoleglycerolphosphate dehydratase (EC 4.2.1.19) plus histidinolphosphatase (EC 3.1.3.15) (bifunctional enzyme)	*hisB*	42	A, B, 48, 83, 230, 232, 432
hisC	Histidine	Histidinol-phosphate aminotransferase (EC 2.6.1.9)	*hisC*	42	A, B, 48, 146, 230, 232
hisD	Histidine	Histidinol dehydrogenase (EC 1.1.1.23)	*hisD*	42	A, B, 11, 31, 33, 48, 63, 83, 229, 230
hisE	Histidine	Phosphoribosyl-ATP pyrophosphohydrolase	*hisE*	42	A, B, 48, 146
hisF	Histidine	Cyclase	*hisF*	42	A, B, 17, 48, 230
hisG	Histidine	ATP phosphoribosyltransferase (EC 2.4.2.17)	*hisG*	42	A, B, 4, 11, 29–33, 48, 61, 146, 170,

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]
					198, 225, 227, 230
hisH	Histidine	Amido transferase	*hisH*	42	A, B, 17, 48, 230
hisI	Histidine	Phosphoribosyl-AMP cyclohydrolase (EC 3.5.4.19) (may be bifunctional with *hisE*)	*hisI*	42	A, B, 48
hisJ	Histidine	Periplasmic histidine-binding protein J for high-affinity histidine transport system	*hisJ*	46	A, B, 5, 6, 19, 20, 158, 160, 163, 254, 283
hisM	Histidine	Histidine transport; location of protein not known		46	F, 160
hisO	Histidine	Operator-promoter	*hisO*	42	A, B, 11, 12, 31, 48, 50, 56, 200, 201, 400, 414
hisP	Histidine	High-affinity histidine transport; the P protein in the inner membrane		46	A, B, 5, 6, 19, 20, 160
hisQ	Histidine	Histidine transport; Q, a membrane protein		46	5, 6, 20, 160
hisR	Histidine	tRNA structural gene	*hisR*	83	A, B, 50
hisS	Histidine	Histidyl-tRNA synthetase (EC 6.1.1.21)	*hisS*	53	A, B, 50
hisT	Histidine	Pseudouridine modification of tRNA	*hisT*	47	A, B, 50, 62–64, 197, 334, 353, 366, 379, 458
hisU	Histidine	tRNA maturation		81	A, B, 50, 97–99
hisW	Histidine	tRNA maturation		45	A, B, 50, 100, 101
hpt		Hypoxanthine phosphoribosyltransferase (not EC 2.4.2.8; see *gpt*)	*hpt*	NM	B, 172
hsdL	Host specificity	*hspLT*; restriction-modification system	*hsd*	8	A, B, 39, 72
hsdSA	Host specificity	*hspS*; restriction-modification system	*hsd*	98	A, B, 39, 72, 221, 311
hsdSB	Host specificity	Restriction-modification system	*hsd*	98	B, 72, 311
hutC	Histidine	Utilization; repressor		18	A, B, 57, 277, 342
hutG	Histidine	Formiminoglutamase (EC 3.5.3.8)		18	A B, 57, 277
hutH	Histidine	Histidine ammonia-lyase (EC 4.3.1.3)		18	A, B, 57, 58, 277
hutI	Histidine	Imidazolonepropionase (EC 3.5.2.7)		18	A, B, 57, 58, 277
hutM	Histidine	Utilization; promoter for *hutIGC*		18	A, B, 57, 60, 277
hutP	Histidine	Utilization; promoter for *hutUH*		18	A, B, 57, 60, 277
hutQ	Histidine	Utilization; operator for *hutUH*		18	A, B, 60, 277
hutR	Histidine	Utilization; catabolite insensitivity of *hutUH*		18	A, B, 60, 277
hutU	Histidine	Utilization; urocanate hydratase (EC 4.2.1.49)		18	A, B, 57, 58, 277

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]
hyd		*fhlB*, *fhlC*; hydrogenase	*hyd*	59	B
hyp	Hydrophobic peptide auxotrophy	Hydrophobic polypeptide requirement		48	67
ilvA	Isoleucine	*ile*; threonine dehydratase (EC 4.2.1.16)	*ilvA*	83	A, B, 45, 46, 52, 54, 55, 73, 97, 100, 101
ilvB	Isoleucine-valine	α-Acetohydroxy acid synthase I (valine sensitivity) (EC 4.1.3.18)	*ilvB*	80	A, B, 100, 101, 415, 416
ilvC	Isoleucine-valine	*ilvA*; acetohydroxy acid isomeroreductase	*ilvC*	83	A, B, 46, 52, 97, 223
ilvD	Isoleucine-valine	*ilvB*; dihydroxy acid dehydratase (EC 4.2.1.9)	*ilvD*	83	A, B, 45, 46, 52, 54, 55, 97, 100, 101
ilvE	Isoleucine-valine	*ilvC*; branched-chain amino acid aminotransferase (EC 2.3.1.42)	*ilvE*	83	A, B, 45, 46, 52, 54, 55, 97, 100, 101, 258
ilvG	Isoleucine-valine	α-Acetohydroxy acid synthetase II (feedback inhibition insensitive)	*ilvG*	83	B, 45, 46, 52, 54, 55, 100, 101, 347, 415, 416
ilvH	Isoleucine-valine	Acetolactate synthase II subunit (normally inactive)	*ilvH*	3	G, 100, 101
ilvI	Isoleucine-valine	Acetolactate synthase II subunit (normally inactive)	*ilvI*	3	G
ilvS	Isoleucine	Isoleucyl-tRNA synthetase (EC 6.1.1.5)		1	A, B
ilvY	Isoleucine	Regulation of *ilvC*	*ilvY*	83	53, 55
inlA	Inositol	Fermentation		92	A, B, 324
inlB	Inositol	Fermentation		54	A, B
kat	Catalase	*cls*; catalase (EC 1.11.1.6)		NM	B
kdsA		Ketodeoxyoctonate synthesis		39	B, 259–261, 328, 358, 359, 469
kdsB		CMP ketodeoxyoctonate synthetase		16	H
lamB	Lambda	Determines a protein resembling the lambda receptor	*lamB*	91	335, 337
leuA	Leucine	α-Isopropylmalate synthase (EC 4.1.1.12)	*leuA*	2	A, B, 133, 143, 157, 477
leuB	Leucine	β-Isopropylmalate dehydrogenase	*leuB*	2	A, B, 143, 157
leuC	Leucine	α-Isopropylmalate isomerase subunit	*leuC*	2	A, B, 134, 143, 157
leuD	Leucine	α-Isopropylmalate isomerase subunit	*leuD*	2	A, B, 134
leuO	Leucine	Operator		2	A, B, 157
leuS	Leucine	Leucyl-tRNA synthetase (EC 6.1.1.4)	*leuS*	14	A, B, 157
leuT	Leucine	Leucine transport		35	A, B
lev		Levomycetin resistance		NM	B
lip	Lipoic acid	Requirement	*lip*	13	A, B
liv	Leucine, isoleucine, and valine	Regulatory gene, high-affinity branched-chain amino acid transport system		NM	323

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]
lkyA	Leaky	Leakage of periplasmic proteins		58	B
lkyB	Leaky	Leakage of periplasmic proteins		58	B
lkyC	Leaky	Leakage of periplasmic proteins		20	B
lkyD	Leaky	Leakage of periplasmic proteins; morphology defect		20	B, J, 137, 138
lon		*capR*; filamentous growth; radiation sensitivity; polyamine metabolism; stabilization of abnormal proteins	*lon*	9	B, R, 391
lpd		Lipoamide dehydrogenase (NADH) (EC 1.6.4.3)	*lpd*	3	B
lpp	Lipoprotein	Murein lipoprotein structural gene		NM	309, 493
lys	Lysine	Requirement	*lysA*	62	A, B
malE	Maltose	*malE*; a protein identical to *E. coli* *malE* protein	*malE*	91	A, B, 337
malQ	Maltose	Amylomaltase (EC 2.4.1.25)	*malQ*	74	A, B
malT	Maltose	Regulation of maltose genes	*malT*	74	336
melA	Melibiose	α-Galactosidase (EC 3.2.1.22)	*mel*	93	378
melB	Melibiose	Melibiose permease	*melB*	93	378
mem	Membrane	Sugar transport and membrane protein defective		78	B
men	Menaquinone	Menaquinone deficient; defective in trimethylamine oxide reduction	*men*	45	96, 247
metA	Methionine	*metI*; homoserine transsuccinylase (EC 2.3.1.46)	*metA*	89	A, B
metB	Methionine	Cystathionine γ-synthase (EC 4.2.99.9)	*metB*	87	A, B, 363
metC	Methionine	Cystathionine γ-lyase (EC 4.4.1.1)	*metC*	64	A, B
metE	Methionine	Tetrahydropteroyltriglutamate methyltransferase (EC 2.1.1.14)	*metE*	84	A, B
metF	Methionine	5,10-Methylenetetrahydrofolate reductase (EC 1.1.1.68)	*metF*	87	A, B
metG	Methionine	Methionyl-tRNA synthetase	*metG*	44	A, B
metH	Methionine	B_{12}-dependent homocysteine-N^5-methyltetrahydrofolate transmethylase	*metH*	89	A, B
metJ	Methionine	Methionine analog resistant	*metJ*	87	A, B, 23
metK	Methionine	*S*-Adenosylmethionine synthetase activity (methionine analog resistant)	*metK*	63	A, B, 23, 501
metP	Methionine	High-affinity methionine transport	*metD*	5	A, B, K, 22, 23
mglB	Methyl galactoside	Galactose-binding protein	*mglB*	NM	B, 302
min	Minicells	Cell division	*min*	NM	B
motA	Motility	Nonmotile, although flagellate	*mot*	40	A, B, 186
motB	Motility	Nonmotile, although flagellate	*mot*	40	A, B, 186
mta	*meso*-Tartaric acid	Utilization of and resistance to *meso*-tartaric acid		NM	285
mtlA	Mannitol	D-Mannitol phosphotransferase enzyme IIA	*mtlA*	78	A, B, 265
mtlD	Mannitol	Mannitol-1-phosphate dehydrogenase (EC 1.1.1.17)	*mtlD*	78	B
musA	Mu sensitivity	Sensitivity to phage Mu		42	117
musB	Mu sensitivity	Sensitivity to phage Mu		NM	117
mutB	Mutator	Increased frequency of mutation with alkylating agents		64	413
mutG	Mutator	Increases mutation in host chromosome, not in P22		NM	B
mutH	Mutator	Mutator	*mutH*	61	B, 412, 413
mutL	Mutator	Increases frequency of mutation	*mutL*	96	B, 412, 413
mutS	Mutator	Increased frequency of mutation with alkylating agents	*mutS*	59	412, 413

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]
nadA	Nicotinamide	*nicA*; requirement	*nadA*	17	A, B, 123, 127, 128, 273
nadB	Nicotinamide	*nic*; requirement	*nadB*	55	B, 123, 168, 273
nadC	Nicotinamide	Requirement	*nadC*	3	B, L, 123, 168, 273
nadD	Nicotinamide	Requirement		14	178
nag		*N*-acetylglucosamine nonutilization	*nag*	15	B, 303
nalB	Nalidixic acid	Resistance or sensitivity	*nalB*	57	B, 316
nap		Deficiency for nonspecific acid phosphatase I		NM	457
ndk		Nucleosidediphosphate kinase (EC 2.7.4.6)		53	B, 364
newD		Substitute gene for *leuD*		7	B, 135, 248, 249
nit	Nitrogen	Nitrogen metabolism		28	B, 108
nml	*N*-methyl-lysine	*N*-Methyl-lysine in flagellin		40	A, B
nol		Norleucine resistance; possible defect in valine uptake or regulation		60	B
nsiA	Nicotinamide starvation inducible	NAD metabolism regulation		77	126
ntrA	Nitrogen regulation	*glnF*; repressor-activator for *glnA* expression and for other nitrogen-controlled genes	*glnF*	69	B, 69, 130, 241, 286, 475
ntrB	Nitrogen regulation	*glnR*; regulation of *glnA* expression and other nitrogen-controlled genes	*glnR*	85	239, 241, 242, 286, 475
ntrC	Nitrogen regulation	*glnR*; regulation of *glnA* expression and other nitrogen-controlled genes	*glnR*	85	239, 241, 286, 475
oafA	O-antigen factor	*ofi*; O-5; O-factor 5 (acetyl group)		43	A, B
oafC	O-antigen factor	Determines factor 1 in group E *Salmonella* spp.		13	A, B, 341
oafR	O-antigen factor	Synthesis of O antigen 12^2		12	A, B
ompA		Outer membrane protein 33K (= II* of *E. coli*)	*ompA*	20	B, 274, 317, 332, 386, 440, 456
ompB		Outer membrane protein 36K (= Ib of *E. coli*); regulation of synthesis of outer membrane proteins	*ompR*	74	B, 145, 196, 269, 270, 317, 336, 456
ompC		Outer membrane protein 36K (= Ib of *E. coli*)	*ompC*	45	B, 145, 196, 274, 317, 319, 332, 336, 450, 451, 456
ompD		Outer membrane protein 34K		32	B, 145, 196, 274, 317, 319, 450, 451, 456
ompF		Outer membrane protein 35K(Ia)	*ompF*	NM	145, 319, 352, 393, 456
oppA		Oligopeptide permease	*oppA*	34	165, 264, 433
oppB		Oligopeptide permease	*oppB*	34	161, 165
oppC		Oligopeptide permease	*oppC*	34	161, 165
oppD		Oligopeptide permease	*oppD*	34	161, 165
optA	Oligopeptidase	Oligopeptidase [hydrolyzes *N*-acetyl-(L-alanyl)$_4$]		77	465

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]
oriC	Origin	*poh*; origin of replication of chromosome	*oriC*	81	195, 507, 508
panB	Pantothenic acid	Ketopantoatehydroxymethyl transferase (EC 4.1.2.12)	*panB*	5	A, B, 92, 348
panC	Pantothenic acid	Pantothenate synthetase (EC 6.3.2.1)	*panC*	5	A, B, 92, 348
panD	Pantothenic acid	Ketopantoic acid reductase		5	B, 348
panE	Pantothenic acid	Ketopantoic acid reductase		NM	348
panT	Pantothenic acid	Pantothenate transport		NM	B, M
pasA		6-Aminonicotinic acid sensitive		92	A, B, 124
pasB		6-Aminonicotinic acid sensitive		98	124
pasC		6-Aminonicotinic acid sensitive		66	124
pck		Phosphoenolpyruvate carboxykinase (ATP) (EC 4.1.1.49)		13	A, B
pclA	Permissive for *cly*	Permissive for lytic growth of P22 *cly*		NM	486
pclB	Permissive for *cly*	Permissive for lytic growth of P22 *cly*		NM	486
pclC	Permissive for *cly*	Permissive for lytic growth of P22 *cly*		NM	486
pde	Phosphodiesterase	2′,3′-Cyclic nucleotide 2′-phosphodiesterase		96	457
pdxB		Pyridoxine requirement	*pdxB*	47	A, B
pepA	Peptidase	Peptidase A (similar to aminopeptidase I of *E. coli*)		97	B, 291, 498, 499
pepB	Peptidase	Peptidase B (aminopeptidase)		53	149, 291, 498, 499
pepD	Peptidase	*ptdD, pepH*; (carnosinase); peptidase D (a dipeptidase)	*pepD*	6	A, B, 291, 498, 499
pepE	Peptidase	Peptidase E (splits asp-X peptide bonds)		90	N
pepN	Peptidase	*ptdN*; peptidase N (an aminopeptidase, naphthylamidase)	*pepN*	20	A, B, 291, 293, 498, 499
pepP	Peptidase	*ptdP*; peptidase P (splits X-pro peptide bonds)		63	A, B, 292, 496
pepQ	Peptidase	Peptidase Q (splits X-pro peptide bonds)		84	B, 292, 496
pepT	Peptidase	Peptidase T (a tripeptidase)		25	O
pfkA		6-Phosphofructokinase (EC 2.7.1.11)	*pfkA*	85	B
pfl		Pyruvate formate-lyase	*pfl*	19	B, 166
pgi		Glucosephosphate isomerase (EC 5.3.1.9)	*pgi*	92	B
pheA	Phenylalanine	Chorismate mutase (EC 5.4.99.5)	*pheA*	55	A, B, 446
pheR	Phenylalanine	Regulator gene for *pheA*		64	A, B, 429
phoN	Phosphatase	Nonspecific acid phosphatase I		95	B, 215–217, 457, 476
phoP	Phosphatase	Nonspecific acid phosphatase		25	B, 215–217, 476
phoS	Phosphatase	Periplasmic phosphate-binding protein		NM	B
phs		Hydrogen sulfide production		41	B, 467
pig	Pigment	Brownish colonies		54	A, B
pil		*fim*; pili (fimbriae)	*pil*	14	A, B, 324
pmi	Mannose	Mannosephosphate isomerase (EC 5.3.1.8)		31	A, B
pmrA		Polymyxin resistance		94	B, 460–464
pncA	Pyridine nucleotide cycle	Nicotinamide to nicotinic acid + ammonia	*pncA*	27	B, 127, 128, 222, 273
pncB	Pyridine nucleotide cycle	Nicotinic acid phosphoribosyltransferase		20	B, 125, 127, 128,

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]
					168, 222, 273
pnuA	Pyridine nucleotide uptake	*pncC*; NMN to NAD deficient		99	222, 427
pnuB	Pyridine mononucleotide uptake	Growth on lower-than-normal levels of NMN		NM	427
pnuC	Pyridine mononucleotide uptake	No growth on NMN		24	427
polA	Polymerase	DNA nucleotidyltransferase (DNA polymerase I; EC 2.7.7.7)	*polA*	85	A, B, 110, 115, 185, 234, 434, 484
pox		Control of P22 lysogeny		NM	B
ppc		Phosphoenolpyruvate carboxylase (EC 4.1.1.31)	*ppc*	87	A, B, 161, 165
ppsB		Phosphoenolpyruvate synthase	*pps*	3	B
praA		Phage P221-receptor function		94	B
praB		Phage P221-receptor function		60	B
prbA		Phage ES18-receptor function		94	B
prbB		Phage ES18-receptor function		30	B
prdB		Phage PH51-receptor function		31	B
prh		Phage HK009-receptor function		94	B
prk		Phage HK068-receptor function		30	B
proA	Proline	Glutamate to glutamic-γ-semialdehyde	*proA*	7	A, B, 79, 248–250
proB	Proline	Glutamate to glutamic-γ-semialdehyde	*proB*	7	A, B, 79, 248–250
proC	Proline	Pyrroline-5-carboxylate reductase (EC 1.5.1.2)	*proC*	8	A, B
proP	Proline	Minor L-proline permease	*proP*	93	B, 14, 93, 287
proU	Proline	Proline permease function only at high osmolarity		57	P, 93
prsA		Phosphoribosylpyrophosphate synthetase	*prs*	35	Q, 194
prsB		Phosphoribosylpyrophosphate synthetase		44	339
psuA		Suppressor of polarity		NM	B
pta		Sensitive to alizarin yellow; acetyl CoA orthophosphate acetyl transferase (EC 2.3.1.8)	*pta*	46	267
ptsF	Phosphotransferase system	Fructose phosphotransferase enzyme IIA	*ptsF*	NM	B, 235, 375, 377
ptsG	Phosphotransferase system	*glu*, *gpt*, *cat*; glucose phosphotransferase enzyme IIB′-factor III (*crr*) system (methyl-β-D-glucoside)	*ptsG*	25	B, 343, 356, 375, 405
ptsH	Phosphotransferase system	*carB*; phosphohistidine protein-hexose phosphotransferase (EC 2.7.1.69)	*ptsH*	49	A, B, 152, 219, 235, 281, 344, 345, 375, 377, 404, 418
ptsI	Phosphotransferase system	*carA*; enzyme I of the phosphotransferase system	*ptsI*	49	A, B, 152, 219, 235, 281, 344, 345, 375, 377, 378, 404, 418
ptsM	Phosphotransferase system	*manA*; mannose-glucose phosphotransferase enzyme IIA (2-deoxyglucose)	*ptsM*	NM	B, 356
ptsP1	Phosphotransferase system	Promoter for *ptsH,I*		49	B

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]
purA	Purine	Adenylosuccinate synthetase (EC 6.3.4.4)	*purA*	96	A, B
purB	Purine	Adenylosuccinate lyase (EC 4.3.2.2)	*purB*	25	A, B
purC	Purine	Phosphoribosylaminoimidazole-succinocarboxamide synthetase (EC 6.3.2.6)	*purC*	51	A, B
purD	Purine	Phosphoribosylglycinamide synthetase (EC 6.3.1.13)	*purD*	89	A, B
purE	Purine	Phosphoribosylaminoimidazole carboxylase (EC 4.1.1.21)	*purE*	11	A, B, 449
purF	Purine	Amidophosphoribosyltransferase (EC 2.1.2.14)	*purF*	47	A, B, 141
purG	Purine	Phosphoribosylglycinamidine synthetase (EC 6.3.5.3)	*purL*	54	A, B
purH	Purine	Phosphoribosylaminoimidazolecarboxamide formyltransferase (EC 2.1.2.3)	*purH*	89	A, B
purI	Purine	Phosphoribosylaminoimidazole synthetase (EC 6.3.3.1)	*purM*	51	A, B
purJ	Purine	IMP cyclohydrolase (EC 3.5.4.10)		89	A, B
putA	Proline	*putB*; utilization; proline oxidase and pyrroline-5-carboxylate dehydrogenase; bifunctional enzyme (proline utilization)		21	A, B, 288, 289
putC	Proline	Utilization; constitutive synthesis of *putA* and *putB* enzymes		21	A, B
putP	Proline	Utilization; major L-proline permease		21	A, B, 14, 93, 287, 289
putR	Proline	Utilization; catabolite repression insensitivity of *putA* and *putB* enzymes		21	A, B
pyrA	Pyrimidine	*argD*, *ars*, *aus*; carbamoyl-phosphate synthase (glutamine) (arginine + uracil requirement) (EC 2.7.2.9)	*car*	1	A, B, 74, 94, 425
pyrB	Pyrimidine	Aspartate carbamoyltransferase (EC 2.1.3.2)	*pyrB*	98	A, B, 82, 94, 191, 193, 425
pyrC	Pyrimidine	Dihydro-orotase (EC 3.5.2.3)	*pyrC*	23	A, B, 74, 191, 425
pyrD	Pyrimidine	Dihydro-orotate oxidase (EC 1.3.3.1)	*pyrD*	20	A, B, 74, 191, 425
pyrE	Pyrimidine	Orotate phosphoribosyltransferase (EC 2.4.2.10)	*pyrE*	79	A, B, 193, 238, 276, 425
pyrF	Pyrimidine	Orotidine-5′-phosphate decarboxylase (EC 4.1.1.23)	*pyrF*	33	A, B, 191, 238, 425
pyrG	Pyrimidine	Cytidine triphosphate synthetase (EC 6.3.4.2)	*pyrG*	60	A, B
pyrH	Pyrimidine	Uridine monophosphate kinase	*pyrH*	5	A, B, 191, 502
rbsP	Ribose	Ribose-binding protein	*rbsP*	82	B, 300
recA		Recombination deficient; degrades DNA	*recA*	58	A, B, 8, 66, 110, 111, 306, 362, 445, 484
recB/C		Recombination deficient; exonuclease V	*recB/C*	61	B
relA	RNA relaxed	RC; regulation of RNA synthesis	*relA*	61	B, 98, 346, 373, 379
rfaB	Rough	UDP-D-galactose:lipopolysaccharide α-1,6-D-galactosyl transferase		79	488

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]
rfaC	Rough	Lipopolysaccharide core defect; proximal heptose deficient	*rfa*	79	B, 67, 88, 153, 280, 386, 431, 438
rfaD	Rough	D-Glycero-D-manno-heptose epimerase	*rfaD*	79	B, 177, 280, 438
rfaE	Rough	Lipopolysaccharide core defect; proximal heptose deficient		76	A, B, 88, 131, 153, 280, 386, 396, 397, 431, 438
rfaF	Rough	Lipopolysaccharide core defect; distal heptose deficient		79	A, B, 162, 176, 177, 276, 280, 398, 438
rfaG	Rough	Lipopolysaccharide core defect; glucose I transferase		79	A, B, 91, 162, 278, 280, 396, 438
rfaH	Rough	Lipopolysaccharide core defect; (galactose I deficient); repression of F-factor function	*sfrB*	84	A, B, 90, 266, 271, 280, 390, 439
rfaI	Rough	Lipopolysaccharide core defect; (galactose I deficient)		78	280
rfaJ	Rough	Lipopolysaccharide core defect; glucose II transferase		79	A, B, 162, 280, 396
rfaK	Rough	Lipopolysaccharide core defect; glucose II deficient		79	A, B, 280
rfaL	Rough	Lipopolysaccharide core defect; *O*-translocase		79	A, B, 162, 280, 301, 338, 396
rfaP	Rough	Lipopolysaccharide core defect; phosphorylation of heptose		77	A, B
rfbA	Rough	TDP-glucose pyrophosphorylase		42	A, B, 275, 280, 320, 438
rfbB	Rough	TDP-glucose oxidoreductase		42	A, B, 280
rfbD	Rough	TDP-rhamnose synthetase		42	A, B, 280
rfbF	Rough	Glucose-1-phosphate cytidylyltransferase (EC 2.7.7.33)		42	A, B, 280
rfbG	Rough	CDP-glucose oxidoreductase		42	A, B
rfbH	Rough	CDP-abequose synthetase		42	A, B, 307
rfbK	Rough	Phosphomannomutase (*man-2*)	*rfb*	42	A, B
rfbL	Rough	Phosphomannomutase B		42	A, B
rfbM	Rough	Mannose-1-phosphate guanylyltransferase (EC 2.7.7.22)		42	A, B
rfbN	Rough	Galactose-diphosphoglycosyl carrier lipid synthetase		42	B
rfbT	Rough	*O*-Translocase		42	A, B
rfc	Rough	*rouC*; O-repeat unit not polymerized		31	A, B, 320
rfe	Rough	Defect in synthesis of enterobacterial common antigen, the T1 antigen, and O-side chains of *Salmonella* sp. groups L and C1	*rfe*	83	A, B, 71, 188, 268
rff	Rough	Block in synthesis of enterobacterial common antigen	*rff*	84	B, 268
rft	Rough	"Transient" T1 forms		15	A, B, 71, 164
rfu	Rough	"Transient" T1 forms		NM	71, 391
rha	Rhamnose	Utilization	*rha*	86	A, B, 324
rho		*psu*; polarity suppressor; transcription terminator factor rho	*rho*	83	84, 173, 174

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]
rhl		Regulator gene for *Hl*		56	A, B
rna		*rnsA*; RNase I	*rna*	17	B, 10, 78, 262, 329
rnc		RNase III	*rnc*	NM	B, 10, 262, 329
rodA	Rod	Round cell morphology	*rodA*	13	S, 17
rpoB		*rif*; RNA polymerase, β subunit (EC 2.7.7.6)	*rpoB*	89	A, B, 65, 193, 452
rpoC		RNA polymerase, β-prime subunit (EC 2.7.7.6)	*rpoC*	89	B, 65, 193, 500
rpoD	RNA polymerase	RNA polymerase, σ subunit		NM	395
rpsE	Ribosomal protein, small	*spcA*; 30S ribosomal subunit protein S5	*rpsE*	71	A, B
rpsL	Ribosomal protein, small	*strA*; 30S ribosomal subunit protein S12	*rpsL*	71	A, B, 179
rrnA	rRNA	rRNA operon	*rrnA*	86	10, 262, 329
rrnB	rRNA	rRNA operon	*rrnB*	89	10, 262, 329
rrnC	rRNA	rRNA operon	*rrnC*	82	10, 262, 329
rrnD	rRNA	rRNA operon	*rrnD*	73	329
rrnE	rRNA	rRNA operon	*rrnE*	89	10, 262, 329
rrnH	rRNA	rRNA operon	*rrnH*	6	10, 329
serA	Serine	Phosphoglycerate dehydrogenase (EC 1.1.1.95)	*serA*	62	A, B, 501
serB	Serine	Phosphoserine phosphatase (EC 3.1.3.3)	*serB*	99	A, B
serC	Serine	Requirement	*serC*	19	166
serD	Serine	Requirement for pyridoxine plus L-serine or glycine		44	B
sidC		Siderochrome utilization; ferrichrome transport; albomycin resistance		4	B
sidF		Siderochrome utilization; ferrichrome transport; albomycin resistance		4	B
sidK		Siderochrome utilization; albomycin resistance; receptor of phage ES18 in *S. typhimurium* and of T5 in *S. paratyphi* B	*tonA*	5	B, 68
smoB		Smooth colony morphology in histidine-constitutive mutants		99	A, B, 236
spcB	Spectinomycin	Resistance; nonribosomal		72	A, B
spoT	Spot	Guanosine 5′-diphosphate, 3′-diphosphate pyrophosphatase	*spoT*	79	T, 99, 373
srlC	Sorbitol	*gut*; regulatory gene	*srlC*	58	A, B
strB	Streptomycin	Low-level resistance plus auxotrophy; nonribosomal		53	A, B
strC		Streptomycin resistance, not *strA* or *-B*		NM	B
sucA	Succinate	*suc*, *lys* + *met*; succinate requirement; α-ketoglutarate dehydrogenase (decarboxylase component)	*sucA*	17	A, B
sufA		Frameshift suppressor affecting proline tRNA and correcting +1 frameshifts at runs of C in the message		77	A, B
sufB		Frameshift suppressor affecting proline tRNA and correcting +1 frameshifts at runs of C in the message		45	A, B

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 message		15	A, B
sufD		Frameshift suppressor affecting glycine tRNA and correcting +1 frameshift mutations at runs of G in the message		62	A, B
sufE		Frameshift suppressor correcting +1 frameshift mutations at runs of G in the message		89	A, B
sufF		Recessive frameshift suppressor correcting +1 frameshift mutations at runs of G in the message		11	A, B
sufG		Frameshift suppressor correcting +1 mutations at runs of A in the message		15	B, 231
sufH		Frameshift suppressor		50	B
sufI		Frameshift suppressor		11	B
sufJ		Frameshift suppressor		88	B, 62, 64
sumA		Suppressor of missense		96	B
supC	Suppressor	Ochre suppressor	*supC*	34	A, B, 487
supD	Suppressor	Amber suppressor; serine insertion	*supD*	40	A, B, 487
supE	Suppressor	*supY*; amber suppressor; glutamine insertion	*supE*	15	A, B, 63, 487
supF		See *tyrT*			B
supG	Suppressor	Ochre suppressor; lysine insertion	*supG*	NM	B, 487
supI		Nonsense suppressor induced by ICR-191 and allelic to *sufG*		15	B
supJ	Suppressor	*supH*; amber suppressor; leucine insertion	*supJ*	82	B, 487
supK		*supT*; recessive UGA suppressor; also corrects some frameshift mutations		62	A, B
supM		see *tyrU*			A, B
supQ		Suppressor of nonsense and deletion mutations of *leuD*		7	A, B, 135, 248, 249
supR		Amber suppressor; haploid lethal		82	A, B
supS		UGA suppressor; haploid lethal		82	A, B
supU		Suppressor of UGA mutations, may be due to alteration of ribosome structure		72	199
tar		Chemotaxis transduction polypeptide; aspartate receptor	*tar*	40	103, 104, 470
tctA		Tricarboxylic acid transport		57	426
tctC		Tricarboxylic acid transport; citrate-binding protein		57	B, 21, 443
tdk		Thymidine kinase (EC 2.7.1.75)	*tdk*	34	A, B
thiA	Thiamine	*thiG*; thiamine or thiazole moiety	*thiA*	89	A, B
thiC	Thiamine	*thiA*; thiamine or pyrimidine moiety	*thiC*	89	A, B
thiD	Thiamine	Thiamine requirement		46	A, B
thiE	Thiamine	Thiazole type		52	A, B
thiF	Thiamine	Thiazole type		52	A, B
thiH	Thiamine	*thiB*; thiamine requirement		54	B
thiI	Thiamine	*thiC*; thiazole type		10	B
thrA	Threonine	*thrCD*; aspartokinase I (EC 2.7.2.4) and homoserine dehydrogenase I (EC 1.1.1.3)	*thrA*	0	A, B
thrB	Threonine	*thrA*; homoserine kinase (EC 2.7.1.39)	*thrB*	0	A, B
thrC	Threonine	*thrB*; threonine synthase (EC 4.2.99.2)	*thrC*	0	A, B
thyA	Thymine	Requirement	*thyA*	61	B
tkt		Transketolase (EC 2.2.1.1)	*tkt*	NM	B

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]
tlp		Loss of protease II		37	B, 156
tlr		Thiolutin resistance; P22 development at high temperature		NM	202–204
tonB		*chr*; determines resistance to ES18; determines a salmonellocin; affects iron transport; regulates levels of some outer membrane proteins	*tonB*	34	A, B, 68, 440
top	DNA topoisomerase I	*supX*; topoisomerase	*topA*	33	A, B, 132, 264, 333, 454, 455
tor		Trimethylamine oxide reductase		80	247
tre	Trehalose	Utilization	*tre*	37	A, B
triM		Tricarboxylic acid metabolism		1	B
triR		Tricarballylic acid transport		1	B
trpA	Tryptophan	*trpC*; tryptophan synthetase, component α (EC 4.2.1.20)	*trpA*	34	A, B, 296, 312, 315, 403, 436, 485
trpB	Tryptophan	*trpD*; tryptophan synthetase, component β (EC 4.2.1.20)	*trpB*	34	A, B, 89, 296, 403, 409
trpC	Tryptophan	*trpE*; *N*-(5-phosphoribosyl) anthranilate isomerase and indole-3-glycerol phosphate synthase (EC 4.1.1.48)	*trpC*	34	A, B, 409
trpD	Tryptophan	*trpB*; anthranilate phosphoribosyltransferase (EC 2.4.2.18)	*trpD*	34	A, B, 112, 150, 252, 313, 446, 447
trpE	Tryptophan	*trpA*; anthranilate synthase (EC 4.1.3.27)	*trpE*	34	A, B, 75, 132, 150, 252, 314, 447, 497
trpO	Tryptophan	Operator	*trpO*	34	A, B, 42, 70, 325
trpP	Tryptophan	Promoter		34	A, B, 15, 42, 70, 132, 325, 326
trpR	Tryptophan	Resistance to 5-methyltryptophan; derepression of tryptophan enzymes	*trpR*	99	A, B
ttr		Tetrathionate reductase		35	A, B
tuf		Protein chain elongation factor Tu	*tuf*	NM	139
tyn		Tyramine oxidase		NM	304
tyrA	Tryosine	Requirement	*tyrA*	55	A, B, 429, 446
tyrO	Tyrosine	Operator for *aroF* and *tyrA*	*aroK*	55	A, B, 429
tyrR	Tyrosine	Regulator gene for *aroF* and *tyrA*	*tyrR*	32	A, B, 429
tyrT	Tyrosine	*supC*; ochre suppressor; tyrosine tRNA1	*tyrT*	34	B, 487
tyrU	Tyrosine	*supM*; ochre suppressor; tyrosine tRNA2	*tyrU*	88	B, 487
ubiF	Ubiquinone	*cad*; deficient in ubiquinone synthesis; accumulates 2-octaprenyl-3-methyl-6-methoxy-1,4-benzoquinone	*ubiF*	14	211, 503
ubiX	Ubiquinone	Polyprenyl *p*-hydrobenzoate carboxylase; growth stimulation by *p*-hydroxybenzoic acid		46	B, 19, 20, 38, 175
udk		Uridine kinase (EC 2.7.1.48)	*udk*	43	A, B, 483
udp		Uridine phosphorylase (EC 2.4.2.3)	*udp*	84	A, B

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]
uhpA		Utilization of hexose phosphate		80	B, U
uhpT		Hexosephosphate transport	*uhpT*	NM	B
umuC		Induction of mutations by UV; sensitivity to UV	*umuC*	NM	114
uncA	Uncoupling	Membrane-bound (Mg^{2+}-Ca^{2+}) ATPase	*unc*	81	195, 507, 508
upp		Uracil phosphoribosyltransferase (EC 2.4.2.9)	*upp*	51	A, B, 483
use	Uracil sensitivity	Altered expression of genes *pyrA*, *pyrC*, *pyrD*, and *argI*		84	74
ush		UDP-glucose hydrolase		90	B, 295
usp	Ureidosuccinate	Permeability to ureidosuccinate (i.e., carbamyl aspartate)		NM	A, B, 503
uvrA	UV	Repair of UV damage to DNA; UV endonuclease, component A	*uvrA*	91	A, B, 95, 233, 240, 435
uvrB	UV	Repair of UV damage to DNA; UV endonuclease, component B	*uvrB*	18	A, B, 8, 306, 445
uvrC	UV	Repair of UV damage to DNA	*uvrC*	40	B
uvrD	UV	Repair of UV damage to DNA; increased sensitivity to mutagenesis by alkylating agents	*uvrB*	84	B, 340, 412, 413
valS		Valyl-tRNA synthetase (EC 6.1.1.9)	*valS*	NM	B
viaA		*ViA*; Vi antigen		46	A, B
viaB		*ViB*; Vi antigen (in *S. typhosa*)		94	A, B
xylA	D-Xylose	Xylose isomerase (EC 5.3.1.5)	*xyl*	78	B, 410, 411
xylB	D-Xylose	Xylulokinase (EC 2.7.1.17)		78	B, 410, 411
xylR	D-Xylose	Regulation		78	B, 410, 411
xylT	D-Xylose	Transport		78	B, 299, 410, 411

[a] Abbreviations: acetyl CoA, acetyl coenzyme A; DAHP, 3-deoxy-D-arabinoheptulosonic acid-7-phosphate; NMN, nicotinamide mononucleotide.

[b] The homologous gene in *E. coli* is described in reference 24.

[c] Map positions are shown in Fig. 1. NM, Not mapped.

[d] The numbers refer to references in Literature Cited. Letters A and B indicate that references on the gene are given in earlier reviews: A refers to reference 382; B refers to reference 385. Letters C through W refer to personal communications from the following sources: C, S. Maloy and J. R. Roth; D, W. W. Kay; E, K. Kutsukake, S. Yamaguchi, and T. Iino; F, G. F.-L. Ames; G, J. Calvo; H, H. Dasgupta and M. J. Osborn; J, J. Fung and L. Rothfield; K, P. D. Ayling; L, K. Hughes and J. R. Roth; M, S. Dunn and E. E. Snell; N, T. H. Carter and C. G. Miller; O, K. S. Strauch and C. G. Miller; P, L. Csonka; Q, J. Gots; R, J. Roth; S, A. T. DeMicheli and D. Antón; T, B. Bochner and J. R. Roth; U, R. O. Burns; V, P. D. Osdoby and C. G. Miller; W, J. Foster.

has therefore permitted identification of polycistronic transcription units and assignment of transcription direction. In general, the outward-directed promoters associated with these elements have not interfered with this sort of experiment because in most genes, these transcripts terminate at *rho*-dependent termination sites before they reach the next cistron (84). In a few locations, however, expression of downstream genes has been noted. In at least some of these cases, expression is due to promoters introduced by the transposon (44, 84). Apparently at these positions no terminators exist to prevent readthrough. Because of these observations, the failure to see a polar effect of transposon insertion should not be taken as unequivocal evidence that no operon exists. When the transposon causes a strong polar block, however, it still seems reasonable to conclude that a polycistronic message exists.

It may be possible to use the outward promoters associated with Tn*5* and Tn*10*. Recently we have selected for expression of a *his* gene whose promoter is deleted. The most prevalent class of mutant by far is due to insertion of Tn*10* and Tn*5* (or their components IS*10* and IS*50*) immediately upstream of the gene in question (A. Wang, personal communication). It may prove valuable to be able to express late genes in operons or to express particular genes independent of their

TABLE 2. Alternative gene symbols[a]

Alternative symbol	Symbol in Table 1
apeD	*apeR*
argA	*argE*
argB	*argA*
argC	*argB*
argD	*pyrA*
argE	*argG*
argF	*argH*
argG	*argD*
argH	*argC*
aroC	*aroE*
aroD	*aroC*
aroE	*aroD*
ars	*pyrA*
asc	*ent*
attP14	*atdA*
attP22 I	*ataA*
attP22I	*atcA*
attP27 I	*atbA*
attP27 II	*atbB*
attP6 I	*atdA*
aus	*pyrA*
aziB	*cysK*
bac	*envB*
cad	*ubiF*
capR	*lon*
carA	*ptsI*
carB	*ptsH*
cat	*ptsG*
cheC	*flaQ*
cheP	*cheA*
cheQ	*cheY*
cheT	*cheZ*
cheU	*flaQ*
cheV	*flaAII*
cheX	*cheB*
chr	*tonB*
cls	*kat*
dad	*dadA*
deg	*lon*
dra	*deoC*
drm	*deoB*
enb	*ent*
fhlB	*hyd*
fhlC	*hyd*
fim	*pil*
galE	*galF*
glnF	*ntrA*
glnR	*ntrB*
glnR	*ntrC*
glu	*ptsG*
gpt	*ptsG*
gut	*srlC*
gxu	*gpt*
hspLT	*hsdL*
hspS	*hsdSA*
ile	*ilvA*
ilvA	*ilvC*
ilvB	*ilvD*
ilvC	*ilvE*
ilvT	*brnQ*
lys + met	*sucA*
manA	*ptsM*
metI	*metA*
motC	*flaAII*
nalA	*gyrA*
nic	*nadB*
nicA	*nadA*
ofi	*oafA*
pepH	*pepD*
pgtA	*glpT*
pncC	*pnuA*
pnu	*deoD*
poh	*oriC*
psu	*rho*
ptdD	*pepD*
ptdN	*pepN*
ptdP	*pepP*
pup	*deoD*
putB	*putA*
rif	*rpoB*
rnsA	*rna*
rouC	*rfc*
smoA	*divC*
spcA	*rpsE*
strA	*rpsL*
suc	*sucA*
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*
trpA	*trpE*
trpB	*trpD*
trpC	*trpA*
trpD	*trpB*
trpE	*trpC*
trzA	*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 equivalent symbols, listed and described in detail in Table 1, be used in future.

normal control elements. In such cases the transposons may prove to be valuable as "portable promoters."

(iv) Complementation tests, using chromo-

TABLE 3. Insertion mutations

Mutation designation	Element inserted	Map position (min)	Strain no. to request[a]	Lab of origin[b]
thr	Tn*10*	0	TT191	J.R.
thr	Tn*5*	0	TT2384	J.R.
pyrA	Tn*10*	1	TT136	J.R.
pyrA	Tn*5*	1	TT2760	J.R.
leu	Tn*10*	2	TT206	J.R.
leu	Tn*5*	2	TT2383	J.R.
ilvHI	Tn*9*	2		C. Berg
pan	Tn*10*	5	TT421	J.R.
proA(B^+)	Tn*10*	7	JL2659	L. Csonka, J. Ingraham
proB(A^-)	Tn*10*	7	JL2520	L. Csonka, J. Ingraham
purE	Tn*10*	11	TT289	J.R.
apeE	Tn*10*	12	TN964	P.C. Osdoby, C. Miller
nadA	Tn*10*	17	TT398	J.R.
bio	Tn*10*	18	TT402	J.R.
pncB	Tn*10*	20	TT6197	J.R.
pepN	Tn*10*	20	TN770	C. Miller
pyrD	Tn*10*	21	TT468	J.R.
pyrD	Tn*5*	21	TT2289	J.R.
put	Tn*10*	22	TT946	J.R.
put	Tn*5*	22	TT2292	J.R.
pyrC	Tn*10*	23	TT459	J.R.
purB	Tn*10*	25	TT282	J.R.
ptsG	Tn*10*	25	PP1120	P.W. Postma
pncA	Tn*10*	27	TT6195	J.R.
pyrF	Tn*10*	33	TT464	J.R.
trp	Tn*10*	34	TT1333	J.R.
trp	Tn*5*	34	TT2377	J.R.
aroD	Tn*10*	36	TT1454	J.R.
tre	Tn*10*	37	TT1518	J.R.
tlp	Tn*5*	37	TN921	P.C. Osdoby, C. Miller
hisD	Tn*10*	42	TT27	J.R.
gnd	Tn*10*	42	NK114	J.R.
hisP	Tn*10*	46	TA3090	G. Ferro-Luzzi Ames
purF	Tn*10*	47	TT317	J.R.
hisT	Tn*5*	47	TT5866	J.R.
cysA	Tn*10*	49	NK186	N. Kleckner
cysA	Tn*5*	50	TT2373	J.R.
purC	Tn*10*	51	TT287	J.R.
purI	Tn*10*	51	TT11	J.R.
guaB	Tn*10*	52	TT275	J.R.
guaA	Tn*10*	52	TT278	J.R.
gua	Tn*5*	52	TT2707	J.R.
glyA	Tn*10*	53	TT418	J.R.
purG	Tn*10*	54	TT315	J.R.
nadB	Tn*10*	55	TT399	J.R.
tyrA	Tn*10*	55	TT126	J.R.
pheA	Tn*10*	55	TT1339	J.R.
proU	Tn*10*	57		L. Csonka
srl	Tn*10*	58	TT520	J.R.
srl	Tn*5*	58	TT2979	J.R.
cysJ	Tn*10*	60	TT173	J.R.
relA	Tn*10*	60	TT7542	J.R.
argA	Tn*10*	61	TT146	J.R.
recBC	Tn*10*	61	DB4659	D. Botstein
lysA	Tn*10*	61	TT215	J.R.
lysA	Tn*5*	61	TT2376	J.R.
pepP	Tn*10*	62	TN853	C. Miller
serA	Tn*10*	62	TT169	J.R.
metC	Tn*10*	64	TT14	J.R.
argG	Tn*10*	69	TT142	J.R.
ntrA	Tn*10*	69	JB1472	J.E. Brenchley
ntrA	Tn*10*	69	SK282	S. Kustu

TABLE 3—*Continued*

Mutation designation	Element inserted	Map position (min)	Strain no. to request[a]	Lab of origin[b]
crp	Tn*10*	72	PP1037	P.W. Postma
cysG	Tn*10*	72	TT172	J.R.
cysG	Tn*5*	72	TT2290	J.R.
ilvB	Tn*5*	80		C. Berg
apeR	Tn*5*	82	TN901	P.C. Osdoby, C. Miller
ilvA	Tn*10*	83	TT58	C. Berg
ilvD	Tn*10*	83	TT81	C. Berg
ilvE	Tn*10*	83	TT71	C. Berg
ilvG	Tn*10*	83	TT4	C. Berg
cya	Tn*10*	83	PP1002	P.W. Postma
metE	Tn*10*	84	TT218	J.R.
metE	Tn*5*	84	TT2370	J.R.
glnA120	Tn*10*	85	SK389	S. Kustu
ntrB137	Tn*10*	85	SK398	S. Kustu
ntrC352	Tn*10*	85	SK835	S. Kustu
metB	Tn*10*	87	TT225	J.R.
metF	Tn*10*	87	TT233	J.R.
metF	Tn*5*	87	TT2381	J.R.
argH	Tn*10*	88	TT137	J.R.
thiA	Tn*10*	88	TT501	J.R.
purD	Tn*10*	89	TT311	J.R.
purD	Tn*5*	89	TT2798	J.R.
purH	Tn*10*	89	TT292	J.R.
purH	Tn*5*	89	TT2792	J.R.
metA	Tn*10*	89	TT256	J.R.
aceA	Tn*10*	90	TT8027	J.R.
malE	Tn*10*	91	TS616	T. Palva
mel	Tn*10*	93	TT1662	J.R.
mutL	Tn*10*	94		G. Walker
purA	Tn*10*	96	TT273	J.R.
pyrB	Tn*10*	98	TT460	J.R.
argI	Tn*10*	98	TT147	J.R.
argI	Tn*5*	98	TT2374	J.R.
serB	Tn*10*	99	TT21	J.R.

[a] These strains are available from the laboratory of origin or from Ken Sanderson, *Salmonella* Genetic Stock Centre.

[b] J.R., John Roth.

somal duplications instead of plasmid exogenotes, are possible by appropriate use of transposons. This method of complementation testing has two main attractions. First, the copy numbers of the two alleles in question are held very close to a 1:1 ratio. The second advantage is the ease with which the structure of the diploid can be verified. Duplications are unstable and segregate both haplotypes among their progeny. Thus, all markers present in the diploid are revealed. With exogenote diploids, segregants reveal only the chromosomal markers; testing of the exogenote markers is more cumbersome since it requires another transfer step.

We have used duplications for complementation tests in three different situations. The first two involve dominant suppressor mutations, one near *spc* (199) and one near *rif* (62). The third case involves recessive *nadD* mutations near *lys* and *leuS* (K. Hughes, D. Ladika, J. Roth, and B. Olivera, manuscript in preparation). In each of these cases transposons were used for construction of the duplication and addition of markers to the diploid. Since each situation was distinct, we will not go through the methods in detail here.

(v) The ability of the Tn*10* element to generate deletions adjacent to its insertion site provides a means of generating deletion mutations. The method is extremely powerful when combined with a positive selection for Tet^s derivatives (59, 282).

Recently, several observations make this method somewhat less appealing for some applications. First, the endpoints of the Tn*10* deletions do not appear to be randomly generated.

TABLE 4. Insertions near genes

Mutation designation	Element inserted	Approximate map position (min)	Linkage[a]	Strain no.	Lab of origin[b]
zjj-1004[c]	Tn*10*	0	66% to *pnuA* 15% to *thr* 50% to *serB*	TT6736	J.R.
zad-803	Tn*10*	2	30% to *leu*	TN745	L. Green, C. Miller
zad-1022	Tn*10*	3	67% to *nadC*	TT7170	J.R.
zaf	Tn*10*	5	38%(P1) to *metP*	HU521	A. Cottam, P. Ayling
zaf-305	Tn*10*	6	50% to *dnaE*	DB9069	R. Maurer, D. Botstein
zah-807	Tn*10*	7	40% to *proAB*	TN986	K. Strauch, C. Miller
zah-806	Tn*10*	7	75% to *pepD*	TN801	L. Green, C. Miller
zai-808	Tn*10*	8	30% to *proC* on *proB* side	TN1004	T. Carter, C. Miller
zaj-1034	Tn*10*	9	50% to *lon*	TT8024	J.R.
zba-883	Tn*10*	10	50% to *apeB21*	TN1785	P. Osdoby, C. Miller
zba-284	Tn*10*	10	90% to *thiI*	TN924	P. Osdoby, C. Miller
zbb-876	Tn*10*	11	75% to *apeA*	TN1781	P. Osdoby, C. Miller
zbc-809	Tn*10*	12	40% to *purE*	TN780	T. Carter, C. Miller
zbc-854	Tn*10*	12	25% to *apeE*	TN1338	P. Osdoby, C. Miller
zbd-873	Tn*10*	12	60% to *apeE*	TN1744	P. Osdoby, C. Miller
zbe-1023	Tn*10*	14	55% to *nadD* 90% to *lip*	TT7247	J.R.
zbf-99	Tn*10*	15	74% to *supE*	TT2342	J.R.
zbj	Tn*10*	15	75% to *sufG*	DB4289	D. Botstein
zbh-1009	Tn*10*	17	Linked to *nadA*	TT6577	J.R.
zbi-812	Tn*10*	18	90% to *galE*	TN1117	P. Osdoby, C. Miller
zbj-1048	Tn*10*	20	94% to *pncB*	TT7445	J.R.
zca-843	Tn*10*	20	30% to *pepN*	TN799	L. Green, C. Miller
zcc	Tn*10*	23	50% to *pyrC*	DB4672	D. Botstein
zcd	Tn*10*	23	86% to *gdhA*	JB1177	J.E. Brenchley
zce-850	Tn*10*	25	50% to *purB*	TN1358	K. Strauch, C. Miller
zci-845	Tn*10*	28	40% to *dcp*		E. Vimr, C. Miller
zde-815	Tn*10*	34	50% to *trp*	TN817	T. Carter, C. Miller
zde-605	Tn*10*	34	54% to *supC*	TT2345	J.R.
zde-94	Tn*10*	35	50% to *supF*	TT2337	J.R.
zea-81	Tn*10*	40	75% to *H1*	TT1952	J.R.
zea	Tn*10*	40	66% to *cheR* 84% to *flaC*	ST314	D.E. Koshland
zeb-618	Tn*10*	40	90% to *supD*	TR2070	J.R.
zeb	Tn*10*	40	33% to *flaR* 46% to *cheR*	ST322	D.E. Koshland
zec-1	Tn*10*	42	50% to *his* at *hisE* end	NK397	N. Kleckner
zec-2	Tn*10*	42	50% to *his* at *hisO* end	TT513	J.R.
zee-78	Tn*10*	44	80% to *metG*	TT2242	J.R.
zeh-754	Tn*10*	45	90% to *hisW*	TT5371	J.R.
zeh	Tn*10*	45	95% to *gyrA*	DB9031	R. Maurer, D. Botstein
zei-102	Tn*10*	46	80% to *dhuA*	TA3088	G. Ames
zei-608	Tn*10*	47	40% to *aroC* 27% to *hisT*	TA3092	G. Ames
zea-1031	*Tn10*	48	95% to *ptsI* between *cysA* and *ptsI*	TT7293	J.R.
zfd-801	Tn*10*	53	33% to *strB* 65% to *pepB* 10% to *glyA*	TN858	L. Green, C. Miller
zfe-801	Tn*10*	53	12% to *glyA*		S. Rodriguez, J. Ingraham
zff-789	Tn*10*	55	55% to *nadB*	TT6581	J.R.
zfg-82	Tn*10*	56	95% to *H2*	TT1896	J.R.
zfi	Tn*10*	57	Between *nalB* and *tct*		J.M. Somers, W.W. Kay
zge-607	Tn*10*	60	13% to *relA*	TA2437	B.N. Ames

TABLE 4—*Continued*

Mutation designation	Element inserted	Approximate map position (min)	Linkage[a]	Strain no.	Lab of origin[b]
zga-1041	Tn*5*	61	70% to *relA*	TT8051	J.R.
zgc-732	Tn*10*	62	90% to *sufD*		J.R.
zgc-18	Tn*10*	62	84% to *thyA* 67% to *recBC* 3% to *argA*		J.R.
zge	Tn*10*	64	30% to *metC* 30% to *cpd*		W.W. Kay
zgc-866	Tn*5*	62	60% to *serA*	TN1655	K. Strauch, C. Miller
zgi	Tn*10*	69	60% to *gltB*	JB1709	J.E. Brenchley
zgi-201	Tn*10*	69	90% to *ntrA*	SK191	S. Kustu
zhb	Tn*10*	72	50% to *crp*		R.O. Burns
zhj-1036	Tn*10*	79	90% to *spoT*	TT7433	J.R.
zia-748	Tn*10*	81	30% to *hisU*	TT3920	J.R.
zhj-1024	Tn*5*	79	75% to *pyrE*	TT7244	J.R.
zhj-1025	Tn*10*	79	70% to *pyrE*	TT7245	J.R.
zhj-1026	Tn*5*	80	40% to *gltC*	TT7246	J.R.
zia	Tn*10*	80	50% to *ilvB*		R.O. Burns
zia-1040	Tn*5*	81	30% to *hisU*	TT8038	J.R.
zia-748	Tn*10*	81	30% to *hisU*	TT3920	J.R.
zib	Tn*10*	81	95% to *dnaA*	DB9048	R. Maurer, D. Botstein
zic-867	Tn*10*	82	60% to *apeR*	TN1741	P. Osdoby, C. Miller
zic-851	Tn*10*	82	73% to *apeR*	TN1239	P. Osdoby, C. Miller
zid-62	Tn*10*	83	95% to *cya* 40% to *hisR*	TT2104	J.R.
zid-64	Tn*10*	83	40% to *ilv*	TT2010	J.R.
zie-822	Tn*5*	84	70% to *metE*	TN1064	K. Strauch, C. Miller
zig-205	Tn*10*	85	40% to *glnA*	SK273	S. Kustu
zig-214	Tn*10*	85	75% to *ntrC*	SK811	S. Kustu
zii-614	Tn*10*	88	30% to *argH* 40% to *sufJ*	TT2385	J.R.
zja-861	Tn*10*	91	5% to *malB* 3% to *pepE*	TN1425	T. Carter, C. Miller
zjd-27	Tn*10*	93	60% to *proP* 60% to *mel*	TT1800	J.R.
zjh-33	Tn*10*	98	83% to *pyrB*	TT563	J.R.
zji-842	Tn*5*	98	88% to *argI* 44% to *pyrB* 28% to *pepA*	TN1040	T. Carter, C. Miller
zji-841	Tn*10*	97	45% to *pepA*	TN797	L. Green, C. Miller
zji-1072	Tn*5*	98	20% to *hsdSA* 7% to *argI*	TT8082	J.R.
zji-1073	Tn*10*	98	15% to *argI*	TT8088	J.R.
zjj	Tn*10*	99	95% to *dnaC*	DB9159	R. Maurer, D. Botstein

[a] Linkage data are joint transductions of the insertion sequence with the gene indicated, using phage P22, except where another phage (P1) is indicated.

[b] Strains can be obtained from the laboratory of origin or from Ken Sanderson, *Salmonella* Genetic Stock Centre. J.R. is John Roth.

[c] Allele numbers have not been inserted for designations that do not comply with standard nomenclature. All mutants numbered by the *z . .* system should be assigned different allele numbers; blocks of allele numbers can be obtained from Ken Sanderson, *Salmonella* Genetic Stock Centre, University of Calgary. *zaa* indicates a mutation or insertion at unit 0 on the linkage map; *zab*, at unit 1; and so on. If allele numbers are non-overlapping, *z . .* designations can be changed as map positions are refined. However, although the map positions of some of the insertions in this table have been modified, the *z . . .* designations are normally left unaltered to avoid confusion when data are already published.

Instead the endpoints seem to preferentially end at hot spots for Tn*10* insertion (5). Thus, if one desires a set of randomly distributed deletion endpoints for use in deletion mapping, Tn*10* is not the method of choice. A second problem with Tn*10*-generated deletions is that the selection of Tet^s derivatives yields inversions as well as deletions (226). Unless one has a series of

point mutants to use in crosses for characterization of the mutations recovered, it is not always simple to determine whether one has isolated a deletion or an inversion with a breakpoint in the gene of interest.

(vi) Although most transposon work in *Salmonella* spp. has involved Tn*10*, it is frequently useful to have a second type of transposon available. The kanamycin resistance element Tn*5* has been used for this purpose. Several methods have been used for making Tn*5* insertions in *Salmonella* spp. We have made transposition mutants by general transduction of Tn*5* into a recipient background with no homology for standard recombination events (289). In this situation transposition is the only way of inheriting kanamycin resistance. We have prevented homologous recombination by using a donor strain (i.e., TT6695 or TT1780) which carries Tn*5* inserted in the *lac* operon of an F'*lac* plasmid. An F^- *Salmonella* sp. recipient strain has no sequences homologous to the site of the donor Tn*5* element; therefore, inheritance of kanamycin resistance can only occur by transposition of the donated element. Alternatively, one can use a *recA*$^-$ recipient to block recombination; in this case any Tn*5* insertion mutant can be used as donor. The above methods are possible, despite the relatively low frequency of Tn*5* transduction, because of the increase in transposition activity of Tn*5* which occurs in these crosses (see below). A more elaborate vehicle for introduction of Tn*5* has been constructed by Nancy Kleckner. Use of this vehicle is described by Shanabruch et al. (412). A marked P22 phage was constructed which carries Tn*5*; the phage genome carries mutations which prevent its lysogeny and growth in the recipient such that the Tn*5* element can only be stably inherited if it transposes from the phage to the host chromosome. This method works well but is, in our opinion, more complicated than the general transduction method.

One problem with use of Tn*5* is its frequent transposition upon initial introduction into a new recipient (49). For example, if *trp*::Tn*5* is transduced into a new recipient, selecting for Kan^r, approximately 10% of the Kan^r transductants will inherit the Kan^r phenotype by transposition of Tn*5* to new sites in the recipient. Therefore, when constructing strains with Tn*5* insertions, it is important to characterize transductants carefully. The tendency of Tn*5* to transpose can be substantially reduced for crosses such as the example given above if the cross is performed at 40°C, since Tn*5* transposes poorly at high temperature. Once a Tn*5* mutation is established in a strain, the transposition activity is inhibited and the strain can be used in much the same ways as for a Tn*10* mutant, provided that it is not retransduced to a new strain. The transposition problem is only associated with initial introduction of Tn*5* into a new strain.

Construction of Operon Fusions

Gene and operon fusions are certainly among the most powerful genetic techniques for the investigation of gene regulation and for the discovery of new genes responding to a particular regulatory signal. The most easily manipulated means of forming such fusions is through the use of hybrid Mu phages constructed by Casadaban and Cohen (76). The original "Mud" phage, Mu *d*1 (Amp^r Lac$^+$), has been used in a wide array of circumstances to form operon fusions. Other Mu derivatives have been constructed which permit protein fusions to the *lacZ* gene (M. Casadaban, personal communication).

Initially these techniques were not available for *Salmonella* work since phage Mu does not infect *Salmonella* spp. However, a variety of means have been used to circumvent the difficulty. One is to move the Mu *d*1 phage from *E. coli* into *Salmonella* spp. by P1-mediated transduction (305, 365). A second method is to move Mu from *E. coli* on an F' factor (257). Another has been to select mutants of *Salmonella* spp. that are sensitive to Mu infection (117). A fourth method has been to use Mu *d*1 with a helper Mu phage which has tail fiber genes derived from P1. The lysates generated by this method have the host range of P1 and can be used to infect appropriately marked *Salmonella* strains (94). Once one Mu *d*1 lysogen is generated by any of the above methods, all host range problems can be overcome by using phage P22 to move the defective Mu *d*1 hybrid into any *S. typhimurium* strain by transduction (168; B. Morrison, unpublished data). In these crosses, P22 carries the hybrid (no Mu helper is required) into the new recipient, and the resulting Mu *d*1 genome is zygotically induced and transposes with high frequency in the new recipient to generate a variety of new fusion lysogens. Since P22 is only slightly larger than the Mu *d*1 prophage, single transduced fragments rarely carry an entire Mu *d*1 prophage. However, the transducing activity of the P22HT105 mutant (see below) is so great that two P22 fragments, each carrying a portion of Mu, frequently cooperate to regenerate the Mu prophage in the recipient, with the net result that transduction frequencies are attained that are high enough to pose no practical problem (K. Hughes, unpublished data).

One of the practical problems of using Mu *d*1 has been its instability. The prophage transposes spontaneously even when temperatures are maintained at 30°C to preserve its temperature-sensitive repressor protein. This makes it impossible to simply select constitutive mutations with

higher levels of *lacZ* expression, since one obtains secondary fusions which place *lacZ* under control of a more active promoter. We have recently constructed a derivative of Mu *d*1 which circumvents this problem. By a two-step selection, mutations were added to the Mu genes that are required for transposition. The resulting Mu *d*1 prophage is unable to transpose as long as it is maintained in strains lacking an amber suppressor. In such strains, the Mu *d*1 (Amp^r Lac^+) prophage can be manipulated in much the same way as a Tn*10* insertion. It can be transduced by P22 into new genetic backgrounds and can be used directly for selection of regulatory mutations, even at high temperature. However, in suppressor-carrying strains, the prophage transposes actively and new fusions are easily generated (K. Hughes and J. R. Roth, unpublished data).

Transductional Methods

The chief technical advantage of *Salmonella* sp. as an object of genetic analysis has been the ease with which transductional crosses can be done with phage P22. Initially, the chief advantages of P22 were high transduction frequencies, simple methods of preparation of high-titer stocks, and, importantly, the stability of phage stocks during storage. Newer additions to the advantages of P22 are *int* mutations, which prevent integration and permit simple recovery of phage-sensitive transductants (424). After this, the HT mutations were found to increase the frequency with which P22 packages host chromosome (402). With these mutations, any chromosomal marker can be transduced at a frequency of approximately 10^{-4}. This frequency makes it possible to generate on the order of 10^5 transductants on a single plate. The resolution of mapping crosses is therefore extremely high. Recombination between mutations separated by the order of 10 base pairs can easily be scored on a single plate. In the words of John Scott, "P22HT takes the worry out of being close" (J. Scott, personal communication).

The second consequence of this high transducing frequency is the ability to detect two-fragment transduction events. If single mutations are restored by recombination at a frequency of 10^{-4} transductants per phage particle, the two-fragment events can be expected at about 10^{-7} double transductants per phage particle (when crosses are performed at a multiplicity of 10). Thus, if 10^9 phage are used on a single selection plate at a multiplicity of 10, one can expect to observe on the order of 100 double transductants. This can be a problem when a low cotransduction frequency is being measured, since even unlinked markers show apparent cotransduction when each is carried into the cell on a separate fragment. For example, under the conditions described above, a cotransduction frequency of 0.1% is meaningless since two-fragment events are expected at this frequency. In these cases one needs to repeat the cross with a low multiplicity of infection (<1). If cotransduction is still seen, one can conclude that true linkage is being observed.

The possibility of detecting two-fragment transduction events can be exploited to good advantage. Deletions larger than one fragment can be repaired by such two-fragment events. If each of two fragments carries a portion of the material, the whole region can be reassembled by recombination. Such events should show the expected dependence on multiplicity of infection. We have used these two-fragment transductions in two situations. One is in the repair of large inversion mutations for which one fragment is needed to repair each breakpoint of the inversion (372). The two-fragment transduction events also contribute to P22's ability to transduce the large Mu *d*1 prophage into new strains as outlined above (Hughes and Roth, unpublished data).

Surprisingly, the HT mutation appears to give P22 the ability to transduce small plasmids such as pBR322 from one strain to another. This is a very convenient means of strain construction since it eliminates the need to prepare plasmid DNA and transform cells. Initial attempts to transduce plasmids with P22 suggested the need for homology between P22 DNA and the plasmid to be transduced (327). These initial experiments were performed with standard P22 phages (no HT mutation). P22HT can transduce plasmids which have no homology with the P22 genome. This phenomenon has been studied by Rolf Menzel and Royce Johnson (personal communication). They found that plasmid transduction is independent of the state of the *recA* gene in either the donor or the recipient. The transduced fragment contains a concatenate of plasmid genomes repeated several times. A possible explanation of this process may be that P22 infection induces rolling-circle replication of the plasmid and P22HT can encapsulate the resulting concatenate. Transductants carry the standard plasmid, with most copies being present as monomers.

Removing a P22 prophage from a lysogenic strain is occasionally desirable. We have found that an easy method for doing this is to transduce deletion *proAB47* into the lysogenic recipient strain. Since this deletion removes the P22 attachment site, inheritance of the deletion necessitates loss of the P22 prophage. The deletion can be transduced selectively since it removes the gene *gpt* and therefore generates the 8-azaguanine-resistant phenotype. A transducing

lysate grown on a strain carrying the *proAB47* mutation can transduce any recipient to 8-azaguanine resistance on plates containing 8-azaguanine and proline. Transductants (which are present above a background of spontaneous resistant mutants) are Pro$^-$ and can be freed of phage by being streaked.

A procedure has been developed which allows the propagation of generalized transducing phage of P22 directly on cells growing on solid media. The donor cells can be killed with chloroform, the phage transferred directly to recipient cells, and transductants selected (367).

Recently, coliphage λ has been successfully used in *Salmonella* spp. Growth of λ in *Salmonella* spp. requires overcoming two obstacles: failure to adsorb and failure of the antitermination protein N of λ to function in *Salmonella* spp. (129). The first problem has been solved by introducing the *lamB* gene of *E. coli* in place of the *Salmonella* sp. equivalent (337). This adds the surface features required for λ infection. The second problem is solved by using N-independent (*nin*) mutants of λ. This is easily arranged since most of the λ cloning vectors are *nin* mutants. Maurer and Botstein have used lambda extensively for the cloning of *Salmonella* sp. DNA replication genes (R. Maurer, personal communication).

Conjugation Methods

Methods of F-mediated conjugation were reviewed in edition V of the linkage map (385). A list of Hfr strains was presented earlier (388). In addition, as noted above, Tn*10* homology can be used to direct F*lac* insertion and generate Hfr origin of transfer (81). The frequency of F-mediated transconjugant formation between strains of *S. typhimurium* LT2 is the same as for *E. coli* K-12 (ca. 1.0 per donor cell) if two barriers to mating are overcome. The first barrier, due to repression of F by a plasmid labeled pSLT (earlier called the "cryptic plasmid," the LT plasmid, or MP10), reduces the number of cells with F pili and reduces mating efficiency by a factor of 100 to 1,000 (9, 387, 428). This barrier can be overcome by use of donor strains lacking plasmid pSLT or by use of strains in which the F carries mutations in the *traO* or the *finP* gene (122), making them insensitive to pSLT repression (387). The second barrier to conjugation is the somatic side chains on the lipopolysaccharide of the normally "smooth" strains of *S. typhimurium*, which reduce the frequency of mating aggregation and of transconjugant formation, especially when mating is in broth rather than on solid medium. The frequency of mating aggregation and of transconjugant formation is increased by a factor of 20 in "rough" mutants which have lost the somatic side chains of the lipopolysaccharide (386, 474). Such strains can be readily isolated by selecting for resistance to bacteriophages such as FO (Felix-O) (482).

Transformation Methods

Transformation with plasmid DNA was reported earlier by Lederberg and Cohen (255). Transformation was reported to be more efficient with *galE* mutants, which have rough lipopolysaccharide, than with smooth strains (G. F.-L. Ames, personal communication). Early-log-phase L-broth cultures of rough strains of class *rfaF* (chemotype Rd2), as well as *galE* mutants, yield 10^5 to 10^6 transformants per μg of plasmid DNA, 10 to 100 times that obtained with smooth strains and with most classes of rough mutants (276). With mutations affecting the heptose region of the lipopolysaccharide core, transformation was lower than for smooth strains by a factor of ca. 10.

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