

# Current Linkage Map of *Escherichia coli*

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INTRODUCTION .....	155
GENETIC NOMENCLATURE .....	155
SUMMARY OF CURRENT MODIFICATIONS AND ADDITIONS TO THE LINKAGE MAP OF <i>E. COLI</i> .....	158
Region from 0 to 15 min .....	158
Region from 15 to 30 min .....	167
Region from 30 to 45 min .....	167
Region from 45 to 60 min .....	167
Region from 60 to 75 min .....	168
Region from 75 to 90 min .....	169
CONCLUDING REMARKS .....	169
LITERATURE CITED .....	170

## INTRODUCTION

A survey of recent literature has shown that the sexually fertile strain K-12 of *Escherichia coli* continues to be a favorite subject for intensive genetic and biochemical investigation. Compared to the 220 genes which were identified and mapped only 2.5 years ago (215), there are now approximately 310 genes described in *E. coli*. Moreover, several of the older genes which were listed in the previous article (215) have now been mapped with much greater precision. This review consists of a comprehensive, updated report on the linkage map of *E. coli*. A similar review of known gene loci in the related species, *Salmonella typhimurium*, appears in a companion report by Sanderson (189).

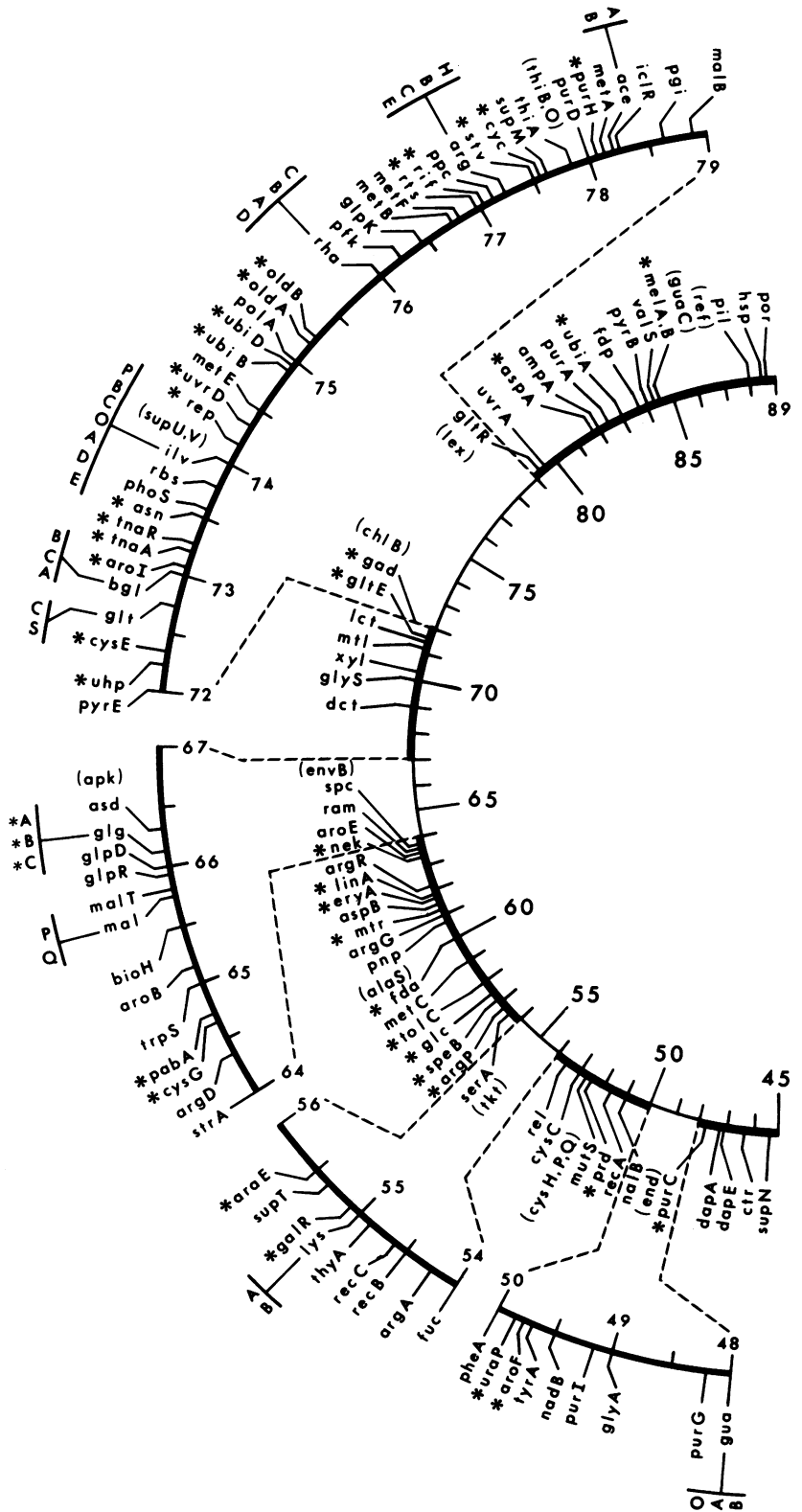
The linkage map of *E. coli*, as of March 1970, is shown in Fig. 1. Although the large size of the map necessitated dividing the figure into right and left halves, it must be kept in mind that the haploid genome of *E. coli* is both genetically and physically a closed, continuous linkage group (31, 118). Accordingly, Fig. 1 should be viewed as an intact circle and not as two separate linkage groups. The functions, insofar as they are known, of all the genes depicted in Fig. 1 are listed in Table 1, together with references to both published and unpublished sources of information. The map scale is marked off in time units, in the manner originated by Jacob and Wollman (118).

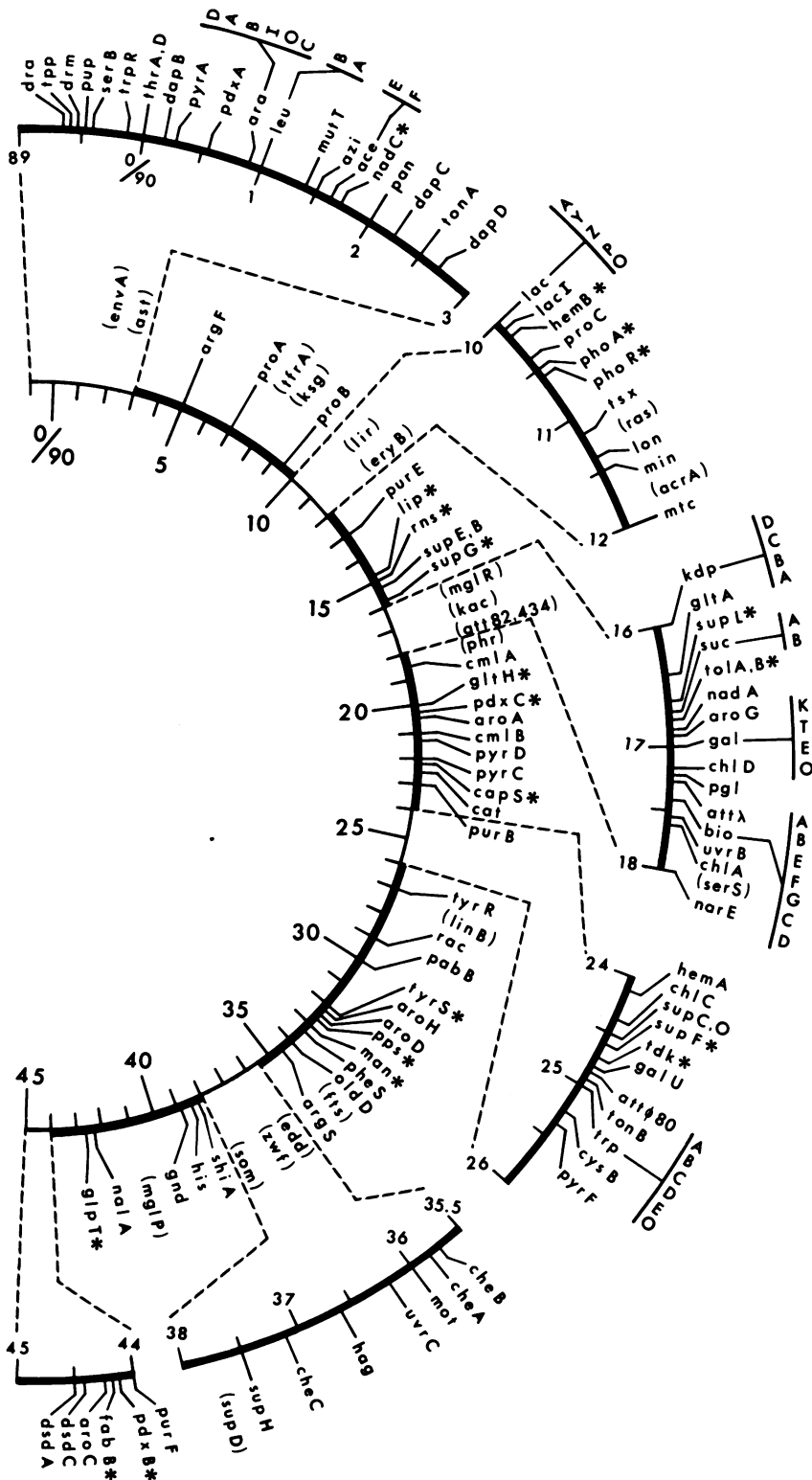
The overall map length of 90 min represents the sum of individual map segments measured by interrupted mating experiments with several different Hfr strains of *E. coli* (214). Precision of the 90-min figure is estimated to be around  $\pm 3\%$ , and it is in good agreement with the results of Fulton (80) who found that a single Hfr donor can transfer a complete chromosome in 90 min.

## GENETIC NOMENCLATURE

The genetic nomenclature employed in this review conforms to the recommendations of Demerec et al. (52) in which gene loci are identified by italicized three-letter symbols, followed by capital letters A through Z to distinguish loci of similar or related function. Occasionally, two or more conventional symbols will crop up in the literature to denote the same gene locus or to identify different allelic states of the same gene. Rather than overcrowd the map with these extra symbols, only the original symbols are shown in Fig. 1 and the alternate symbols are given as cross-referenced entries in Table 1. There are also occasions when an old symbol becomes either misleading or inappropriate and there is good reason to change it altogether. One example is the symbol *nic* which has been used to specify genes determining nicotinic acid requirement. A recent study points out that the *nic* genes do not govern the synthesis of nicotinic acid per se, but

FIG. 1. Scale drawing of the linkage map of *E. coli* adapted from Taylor and Trotter (215). The inner circle, which bears the time scale from 0 through 90 min, depicts the intact circular linkage map. The map is graduated in 1-min intervals beginning arbitrarily with zero at the *thrA* locus. Selected portions of the map (e.g., the 10- to 12-min segment) are displayed on arcs of the outer circle with a 4.5 $\times$  expanded time scale to accommodate all of the markers in crowded regions. Gene symbols are explained in Table 1. Markers in parentheses are only approximately mapped at the positions shown. A gene identified by an asterisk has been mapped more precisely than the markers in parentheses, but its orientation relative to adjacent markers is not yet known.





rather the synthesis of nicotinamide adenine dinucleotide (218). Hence, the *nic* genes are now identified by the more appropriate symbol *nad* (218).

#### SUMMARY OF CURRENT MODIFICATIONS AND ADDITIONS TO THE LINKAGE MAP OF *E. COLI*

Two principal techniques of genetic mapping will be mentioned in the following discussion. The first method is mapping by "time of entry," in which genetic markers are positioned on the chromosome by determining the time when each marker first enters a recipient cell during the course of chromosome transfer from an Hfr donor strain. The method is described in detail by Jacob and Wollman (118), and references to more recent technical refinements are given in (215). Additional information pertaining to specific Hfr strains mentioned below is also available (215). The second, and generally more accurate, mapping technique relies on transduction crosses mediated by bacteriophage P1 of *E. coli*, as described by Lennox (130). Two-factor transduction crosses provide estimates of the distance between pairs of markers separated by not more than 1.5 to 2 min by making use of the empirical "minutes-to-per cent frequency of cotransduction" correlations summarized in Table 9 of reference 215. Multifactor transduction crosses involving three or more cotransducible markers permit precise determinations of the sequence of markers with respect to each other and to the linkage map as a whole. Groups of closely linked markers for which the gene sequence relative to the whole map is known are referred to in this review as having a known orientation.

In the following survey, which will be confined largely to new data appearing since 1967, the discussion begins with markers located in the 0- to 15-min map segment and then proceeds in a clockwise direction through successive 15-min increments of the map.

**Region from 0 to 15 min.** The locus *dapB*, which is placed at minute 0 between *thr* and *pyrA* by three-point transduction crosses, is one of five genes for  $\alpha$ - $\epsilon$ -diaminopimelic acid biosynthesis recently mapped in this laboratory (A. I. Bukhari and A. L. Taylor, *in preparation*). We have assigned the suffix *B* to this *dap* locus since it determines the second enzyme of the biosynthetic pathway (Table 1). A new locus corresponding to an operator region (*araO*) has been added to the arabinose operon at minute 1 (125). This report (125) also indicates the orientation of the *leuA* and *leuB* genes of the leucine operon. The specific transversion-inducing mutator gene, *mutT*, is now accurately mapped at

minute 1.5 (41, 97). A second mutator allele, *ast*, also maps in this general region, but a recent comparative study (245) shows that *ast* strains are phenotypically quite distinct from *mutT* stocks. The *azi* locus, originally described as determining resistance to sodium azide (117, 134), is now known to be associated with additional phenotypic alterations involving resistance to phenethyl alcohol and defective septum formation (242). It seems likely, as suggested by Yura and Wada (242), that the entire *mutT-azi-ace E, F* region may turn out to be a gene complex that specifies part of the structure and function of the cell membrane. The *nadC* locus, one of three known genes which determine nicotinic acid requirement, maps between *azi* and *pan* (85), but its exact position with respect to other nearby markers is not known. The *dapD* and *dapC* loci have been mapped by transduction at points immediately clockwise and anticlockwise, respectively, to *tonA*. The genes probably control the third and fourth steps in diaminopimelic acid biosynthesis, but this has not been confirmed by direct enzyme assays. It appears that certain mutations resulting in defective synthesis of cell wall peptidoglycan also map in the general vicinity of these *dap* genes (143). R. Curtiss (*personal communication*) informs us that the pleiotropic phage resistance gene *tfrA* was incorrectly placed at min 10 in the previous map (215) and that it actually lies somewhere between minutes 7 and 9. The *ksg* (210), *lir*, and *eryB* (9) genes are all concerned with the level of resistance to various antibiotics, and all three map roughly in the region between *leu* and *lac*. The preliminary indication seems to be that they represent at least two new regions, besides the one at minutes 63-64, which code for ribosomal proteins. Positions of the *tsx* and *purE* genes, previously placed at minutes 12.5 and 15, respectively (215), have been changed slightly in accord with new transduction data reported by Donch and Greenberg (56). Several laboratories have described mutants at minutes 11-12 which manifest altered sensitivities to mitomycin C, ultraviolet light, acridines, methylene blue, phenethyl alcohol, triphenyl tetrazolium chloride, or sodium dodecyl sulfate (110, 149, 161, 213). Mutants having defects in septum formation or alterations in radiation sensitivity also map in this region (*see lon, min, and ras*) and it therefore appears that the 11- to 12-min segment may contain a second gene complex concerned with membrane structure and function. Several mutants defective in lipoic acid biosynthesis have been mapped at the *lip* locus at minute 15 (100); the orientation of *lip* with respect to *rns*, also at minute 15 (181), is not known.

TABLE 1. List of genetic markers of *E. coli*

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>aceA</i>	Acetate	78	<i>icl</i> ; utilization of acetate: isocitrate lyase	26, 223
<i>aceB</i>	Acetate	78	<i>mas</i> ; utilization of acetate: malate synthetase A	223
<i>aceE</i>	Acetate	2	<i>aceE1</i> ; acetate requirement; pyruvate dehydrogenase (decarboxylase component)	98, 99
<i>aceF</i>	Acetate	2	<i>aceE2</i> ; acetate requirement; pyruvate dehydrogenase (lipoic reductase-transacetylase component)	98, 99
<i>acrA</i>	Acridine	(11)	Sensitivity to acriflavine, phenethyl alcohol, Na dodecyl sulfate	149
<i>alaS</i>	Alanine	(60)	<i>ala-act</i> ; alanyl-tRNA synthetase	150
<i>ampA</i>	Ampicillin	82	Resistance or sensitivity to penicillin	70
<i>apk</i>		(66)	Lysine-sensitive aspartokinase	164
<i>araA</i>	Arabinose	1	L-Arabinose isomerase	129
<i>araB</i>	Arabinose	1	L-Ribulokinase	129
<i>araC</i>	Arabinose	1	Regulatory gene	200, 201
<i>araD</i>	Arabinose	1	L-Ribulose 5-phosphate 4-epimerase	129
<i>araE</i>	Arabinose	56	L-Arabinose permease	67, 156, A
<i>araI</i>	Arabinose	1	Initiator locus	200, 201
<i>araO</i>	Arabinose	1	Operator locus	125
<i>argA</i>	Arginine	54	<i>argB</i> , <i>Arg1</i> , <i>Arg2</i> ; N-acetylglutamate synthetase	90, 111, 215, 226
<i>argB</i>	Arginine	77	<i>argC</i> ; $\alpha$ -N-acetyl-L-glutamate-5-phosphotransferase	87, 88, 133, 226
<i>argC</i>	Arginine	77	<i>argH</i> , <i>arg2</i> ; N-acetylglutamic- $\gamma$ -semialdehyde dehydrogenase	87, 88, 133, 226
<i>argD</i>	Arginine	64	<i>argG</i> , <i>Arg1</i> ; acetylornithine- $\delta$ -transaminase	112, 226, B
<i>argE</i>	Arginine	77	<i>argA</i> , <i>Arg4</i> ; L-ornithine-N-acetylornithine lyase	87, 88, 133, 226
<i>argF</i>	Arginine	5	<i>argD</i> , <i>Arg5</i> ; ornithine transcarbamylase	90, 133, 226
<i>argG</i>	Arginine	61	<i>argE</i> , <i>Arg6</i> ; argininosuccinic acid synthetase	133, 214, 215, 226
<i>argH</i>	Arginine	77	<i>argF</i> , <i>Arg7</i> ; L-argininosuccinate arginine lyase	87, 88, 133, 226
<i>argP</i>	Arginine	57	Arginine permease	132, C
<i>argR</i>	Arginine	62	<i>Rarg</i> ; regulatory gene	90, 119, 133, 226
<i>argS</i>	Arginine	35	Arginyl-tRNA synthetase	40
<i>aroA</i>	Aromatic	21	3-Enolpyruvylshikimate-5-phosphate synthetase	170, 178, 214
<i>aroB</i>	Aromatic	65	Dehydroquinase synthetase	109, 170, 232
<i>aroC</i>	Aromatic	44	Chorismic acid synthetase	170, 214
<i>aroD</i>	Aromatic	32	Dehydroquinase	170, 214
<i>aroE</i>	Aromatic	64	Dehydroshikimate reductase	170, 215, 232
<i>aroF</i>	Aromatic	50	3-Deoxy-D-arabinoheptulosonic acid-7-phosphate (DHAP) synthetase (tyrosine-repressible isoenzyme)	228

<sup>a</sup> Numbers refer to the time scale shown in Fig. 1. Parentheses indicate approximate map locations.

<sup>b</sup> Numbers refer to the Literature Cited section; letters refer to the following: (A) E. Englesberg, *personal communication*; (B) A. L. Taylor, *unpublished data* mentioned in this paper; (C) W. K. Maas and P. Popkins, *personal communication*; (D) R. Curtiss, III, *personal communication*; (E) A. I. Bukhari and A. L. Taylor, *in preparation*; (F) H. Yamaguchi, C. Fetterolf, and J. G. Flaks, *personal communication*; (G) E. J. Murgola, *personal communication*; (H) H. I. Adler, *personal communication*; (I) G. J. Tritz and R. K. Gholson, *personal communication*; (J) T. T. Wu and E. C. Lin, *personal communication*; (K) B. Low, *personal communication*; (L) W. F. Doolittle, *personal communication*; (M) W. K. Maas, *personal communication*; (N) E. Whitney, *personal communication*.

TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min.) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>aroG</i>	Aromatic	17	DHAP synthetase (phenylalanine-repressible isoenzyme)	1, 27, 228
<i>aroH</i>	Aromatic	32	DHAP synthetase (tryptophan-repressible isoenzyme)	228
<i>aroI</i>	Aromatic	73	Function unknown	86
<i>asd</i>	—	66	<i>dap</i> + <i>hom</i> ; aspartic semialdehyde dehydrogenase	38, 95, 196
<i>asn</i>	—	73	Asparagine synthetase	34
<i>aspA</i>	—	82	Aspartase	138
<i>aspB</i>	Aspartate	62	<i>asp</i> ; aspartate requirement	118, 180
<i>ast</i>	Astasia	(4)	Generalized high mutability	244, 245
<i>attλ</i>	Attachment	17	Integration site for prophage λ	118, 184
<i>attφ80</i>	Attachment	25	Integration site for prophage φ80	204
<i>att82</i>	Attachment	(17)	Integration site for prophage 82	118, 184
<i>att434</i>	Attachment	(17)	Integration site for prophage 434	118, 184
<i>azi</i>	Azide	2	<i>pea</i> , <i>fts</i> ; resistance or sensitivity to Na azide or phenethyl alcohol; filament formation at 42C	117, 134, 221, 242
<i>bglA</i>	β-Glucoside	73	<i>β-gIA</i> ; aryl β-glucosidase	192
<i>bglB</i>	β-Glucoside	73	<i>β-gIB</i> ; β-glucoside permease	192
<i>bglC</i>	β-Glucoside	73	<i>β-gIC</i> ; regulatory gene	192
<i>bioA</i>	Biotin	17	Group II; 7-oxo-8-aminopelargonic acid (7KAP) → 7,8-diaminopelargonic acid (DAPA)	1, 51, 65, 182
<i>bioB</i>	Biotin	17	Conversion of dethiobiotin to biotin	1, 51, 65, 182
<i>bioC</i>	Biotin	17	Unknown block prior to 7KAP synthetase	1, 51, 65, 182
<i>bioD</i>	Biotin	17	Dethiobiotin synthetase	1, 51, 64, 65, 182
<i>bioE</i>	Biotin	17	Unknown block prior to 7KAP synthetase	1, 51, 65, 182
<i>bioF, G</i>	Biotin	17	7KAP synthetase	1, 51, 65, 182
<i>bioH</i>	Biotin	66	<i>bioB</i> ; early block prior to 7KAP synthetase	95, 182, 196
<i>capS</i>	Capsule	22	Regulatory gene for capsular polysaccharide synthesis	139
<i>cat</i>	—	23	<i>CR</i> ; catabolite repression	219
<i>cheA</i>	Chemotaxis	36	<i>motA</i> ; chemotactic motility	11, 12
<i>cheB</i>	Chemotaxis	36	<i>motB</i> ; chemotactic motility	11, 12
<i>cheC</i>	Chemotaxis	37	Chemotactic motility	12
<i>chlA</i>	Chlorate	18	<i>narA</i> ; pleiotropic mutations affecting nitrate-chlorate reductase and hydrogen lyase activity	1, 174, 175, 224
<i>chlB</i>	Chlorate	(71)	<i>narB</i> ; pleiotropic mutations affecting nitrate-chlorate reductase and hydrogen lyase activity	174
<i>chlC</i>	Chlorate	25	<i>narC</i> ; structural gene for nitrate reductase	93, 174, 187
<i>chlD</i>	Chlorate	17	<i>narD</i> , <i>narF</i> ; nitrate-chlorate reductase	1, 224
<i>cmlA</i>	Chloramphenicol	19	Resistance or sensitivity to chloramphenicol	178
<i>cmlB</i>	Chloramphenicol	21	Resistance or sensitivity to chloramphenicol	178
<i>ctr</i>	—	46	Mutations affecting the uptake of diverse carbohydrates	231
<i>cyc</i>	Cycloserine	78	Resistance or sensitivity to D-cycloserine	47, D
<i>cysB</i>	Cysteine	25	Pleiotropic mutations affecting cysteine biosynthesis	120, 205, 239
<i>cysC</i>	Cysteine	53	Adenosine 5'-sulfatophosphate kinase	120, 144, 215

TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>cysE</i>	Cysteine	72	Apparently pleiotropic	120
<i>cysG</i>	Cysteine	65	Sulfite reductase	215
<i>cysH</i>	Cysteine	(53)	Adenosine 3'-phosphate 5'-sulfato-phosphate reductase	120
<i>cysP</i>	Cysteine	(53)	Sulfate permease and sulfite reductase	120
<i>cysQ</i>	Cysteine	(53)	Sulfite reductase	120
<i>dapA</i>	Diaminopimelate	47	Dihydrodipicolinic acid synthetase	38, E
<i>dapB</i>	Diaminopimelate	0	Dihydrodipicolinic acid reductase	73, E
<i>dapC</i>	Diaminopimelate	2	Tetrahydrodipicolinic acid → N-succinyl-diaminopimelate	E
<i>dapD</i>	Diaminopimelate	3	Tetrahydrodipicolinic acid → N-succinyl diaminopimelate	E
<i>dapE</i>	Diaminopimelate	47	<i>dapB</i> ; N-succinyl-diaminopimelic acid deacylase	38, E
<i>darA</i>	—	—	See <i>uvrD</i>	222
<i>dct</i>	—	69	Uptake of C <sub>4</sub> -dicarboxylic acids	124
<i>deo</i>	Deoxythymidine	—	See <i>dra</i> , <i>drm</i> , <i>pup</i> and <i>tpg</i>	131
<i>dra</i>	—	89	<i>deoC</i> , <i>thyR</i> ; deoxyriboaldolase	4, 131, 159
<i>drm</i>	—	89	<i>deoB</i> , <i>thyR</i> ; deoxyribomutase	4, 131, 159
<i>dsdA</i>	D-Serine	45	D-Serine deaminase	144
<i>dsdC</i>	D-Serine	45	Regulatory gene	144
<i>edd</i>	—	(35)	Entner-Doudoroff dehydrase (gluconate-6-phosphate dehydrase)	167
<i>end</i>	—	(50)	<i>endI</i> ; endonuclease I	59
<i>envA</i>	Envelope	(3)	Anomalous cell division involving chain formation	155
<i>envB</i>	Envelope	(65)	Anomalous spheroid cell formation	154
<i>eryA</i>	Erythromycin	62	Resistance or sensitivity to erythromycin	F
<i>eryB</i>	Erythromycin	(11)	High level resistance to erythromycin	9
<i>exr</i>	—	—	See <i>lex</i>	
<i>fabB</i>	—	44	Fatty acid biosynthesis	69
<i>fda</i>	—	60	<i>ald</i> ; fructose-1, 6-diphosphate aldolase	20
<i>fdp</i>	—	84	Fructose diphosphatase	77, 78, 240
<i>ftsA</i>	—	—	See <i>azi</i>	221
<i>fts</i>	—	(35)	<i>fts-9</i> ; filamentous growth and inhibition of nucleic acid synthesis at 42 C	221
<i>fuc</i>	Fucose	54	Utilization of L-fucose	71, 215
<i>gad</i>	—	72	Glutamic acid decarboxylase	135, 137
<i>galE</i>	Galactose	17	<i>galD</i> ; uridinediphosphogalactose 4-epimerase	3, 29
<i>galK</i>	Galactose	17	<i>galA</i> ; galactokinase	3, 29
<i>galO</i>	Galactose	17	<i>galC</i> ; operator locus	29, 30
<i>galT</i>	Galactose	17	<i>galB</i> ; galactose 1-phosphate uridyl transferase	3
<i>galR</i>	Galactose	55	<i>Rgal</i> ; regulatory gene	30, 188
<i>galU</i>	Galactose	25	<i>UPDG</i> ; uridine diphosphoglucose pyrophosphorylase	198, 93
<i>glc</i>	Glycolate	58	Utilization of glycolate; malate synthetase G	223
<i>glgA</i>	Glycogen	66	Glycogen synthetase	33, 203
<i>glgB</i>	Glycogen	66	$\alpha$ -1,4-Glucan: $\alpha$ -1,4-glucan 6-glucosyltransferase	33, 203
<i>glgC</i>	Glycogen	66	Adenosine diphosphate glucose pyrophosphorylase	33, 203
<i>glpD</i>	Glycerol phosphate	66	<i>glyD</i> ; L- $\alpha$ -glycerophosphate dehydrogenase	44, 196
<i>glpK</i>	Glycerol phosphate	76	Glycerol kinase	45

TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>glpT</i>	Glycerol phosphate	43	L- $\alpha$ -Glycerophosphate transport system	44
<i>glpR</i>	Glycerol phosphate	66	Regulatory gene	44
<i>gltA</i>	Glutamate	16	<i>glut</i> ; requirement for glutamate; citrate synthase	13, 68, 100
<i>gltC</i>	Glutamate	73	Operator locus	135, 136
<i>gltE</i>	Glutamate	72	Glutamyl-tRNA synthetase	G
<i>gltH</i>	Glutamate	20	Requirement	135
<i>gltR</i>	Glutamate	79	Regulatory gene for glutamate permease	136
<i>gltS</i>	Glutamate	73	Glutamate permease	136
<i>glyA</i>	Glycine	49	Serine hydroxymethyl transferase <sup>c</sup>	48, 215
<i>glyS</i>	Glycine	70	<i>gly-act</i> ; glycyl-tRNA synthetase	21
<i>gnd</i>	—	39	Gluconate-6-phosphate dehydrogenase	167
<i>guaA</i>	Guanine	48	<i>gua</i> ; xanthosine-5'-monophosphate aminase	151, 211, 215
<i>guaB</i>	Guanine	48	<i>gua</i> ; inosine-5'-monophosphate dehydrogenase	151, 211
<i>guaC</i>	Guanine	(88)	Guanosine-5'-monophosphate reductase	151
<i>guaO</i>	Guanine	48	Operator locus	151, 152
<i>hag</i>	H antigen	37	<i>H</i> ; flagellar antigens (flagellin)	12
<i>hemA</i>	Hemin	24	Synthesis of $\delta$ -aminolevulinic acid	93, 190, 191
<i>hemB</i>	Hemin	10	<i>ncf</i> ; synthesis of catalase and cytochromes	191
<i>his</i>	Histidine	39	Requirement	214
<i>hsp</i>	Host specificity	89	<i>hs, rm</i> ; host restriction and modification of DNA	23, 39, 128, 235
<i>icl</i>	—	—	<i>See aceA</i>	26
<i>iclR</i>	—	78	Regulation of the glyoxylate cycle	26
<i>ilvA</i>	Isoleucine-valine	74	<i>ile</i> ; threonine deaminase	169, 177
<i>ilvB</i>	Isoleucine-valine	74	Condensing enzyme (pyruvate + $\alpha$ -ketobutyrate)	169, 177
<i>ilvC</i>	Isoleucine-valine	74	<i>ilvA</i> ; $\alpha$ -hydroxy- $\beta$ -keto acid reductoisomerase	169, 177
<i>ilvD</i>	Isoleucine-valine	74	<i>ilvB</i> ; dehydrase	169, 177
<i>ilvE</i>	Isoleucine-valine	74	<i>ilvC</i> ; transaminase B	169, 177
<i>ilvO</i>	Isoleucine-valine	74	Operator locus for genes <i>ilvA, D, E</i>	176, 177
<i>ilvP</i>	Isoleucine-valine	74	Operator locus for gene <i>ilvB</i>	176, 177
<i>kac</i>	K-accumulation	17	Defect in potassium ion uptake	28
<i>kdpA-D</i>	K-dependent	16	Requirement for a high concentration of potassium	68
<i>ksg</i>	Kasugamycin	(8)	Resistance or sensitivity to kasugamycin (30S ribosomal subunit)	210
<i>lacA</i>	Lactose	10	<i>a, lacAc</i> ; thiogalactoside transacetylase	18, 145, 243
<i>lacI</i>	Lactose	10	<i>i</i> ; regulator gene	49, 145
<i>lacO</i>	Lactose	10	<i>o</i> ; operator locus	49, 145
<i>lacP</i>	Lactose	10	<i>p</i> ; promoter locus	49, 116, 145
<i>lacY</i>	Lactose	10	<i>y</i> ; galactoside permease (M protein)	76, 117, 145
<i>lacZ</i>	Lactose	10	<i>z</i> ; $\beta$ -galactosidase	117, 134, 145
<i>lct</i>	Lactate	71	L-Lactate dehydrogenase	163
<i>leuA</i>	Leucine	1	$\alpha$ -Isopropylmalate synthetase	117, 125, 134
<i>leuB</i>	Leucine	1	$\beta$ -Isopropylmalate dehydrogenase	125
<i>lex</i>	—	(79)	Resistance or sensitivity to X rays and UV light	106
<i>linA</i>	Lincomycin	62	Resistance or sensitivity to lincomycin	F
<i>linB</i>	Lincomycin	(28)	High-level resistance to lincomycin	9
<i>lip</i>	Lipoic acid	15	Requirement	100, 225
<i>lir</i>	—	(11)	Increased sensitivity to lincomycin or erythromycin, or both	9

<sup>c</sup> Enzymatic defect is inferred from studies on the homologous mutant in *S. typhimurium*. Refer to Sanderson (189) for additional references.



TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>lon</i>	Long form	11	<i>capR</i> , <i>dir</i> , <i>muc</i> ; filamentous growth, radiation sensitivity, and regulation of capsular polysaccharide synthesis	2, 56, 108, 140, 221
<i>lysA</i>	Lysine	55	Diaminopimelic acid decarboxylase	118, 214, E
<i>lysB</i>	Lysine	55	Lysine or pyridoxine requirement	E
<i>malB</i>	Maltose	79	<i>mal-5</i> ; maltose permease and phage $\lambda$ receptor site	196, 197
<i>malP</i>	Maltose	66	<i>malA</i> ; maltodextrin phosphorylase	95, 96, 118, 196
<i>malQ</i>	Maltose	66	<i>malA</i> ; amylomaltase	95, 96, 118, 196
<i>malT</i>	Maltose	66	<i>malA</i> ; probably a positive regulatory gene	95, 96, 118, 196
<i>man</i>	Mannose	33	Phosphomannose isomerase	141, 215
<i>melA</i>	Melibiose	84	<i>mel-7</i> ; $\alpha$ -galactosidase	195
<i>melB</i>	Melibiose	84	<i>mel-4</i> ; thiomethylgalactoside permease II	173, 195
<i>metA</i>	Methionine	78	<i>met<sub>3</sub></i> ; homoserine <i>O</i> -transsuccinylase	107, 118, 186, 196
<i>metB</i>	Methionine	77	<i>met-1</i> , <i>met<sub>1</sub></i> ; cystathionine synthetase	87, 118, 186, 214
<i>metC</i>	Methionine	59	Cystathionase	186, 215
<i>metE</i>	Methionine	75	<i>met-B<sub>12</sub></i> ; <i>N</i> <sup>5</sup> -methyltetrahydropteroyl triglutamate-homocysteine methylase <sup>c</sup>	63, 207, 214
<i>metF</i>	Methionine	77	<i>met-2</i> , <i>met<sub>2</sub></i> ; <i>N</i> <sup>5</sup> , <i>N</i> <sup>10</sup> -methyltetrahydrofolate reductase <sup>c</sup>	87, 88, 118, 207
<i>mgIP</i>	Methyl-galactoside	(40)	<i>P-MG</i> ; methyl-galactoside permease	82, 185
<i>mgIR</i>	Methyl-galactoside	(17)	<i>R-MG</i> ; regulatory gene	82
<i>min</i>	Minicell	11	Formation of minute cells containing no DNA	36, H
<i>mot</i>	Motility	36	Flagellar paralysis	12
<i>mtc</i>	Mitomycin C	12	<i>Mb</i> , <i>mbl</i> ; sensitivity to acridines, methylene blue and mitomycin C	110, 161, 213
<i>mtl</i>	Mannitol	71	Utilization of D-mannitol	214
<i>mtr</i>	Methyl tryptophan	61	Resistance to 5-methyltryptophan	103
<i>mutS</i>	Mutator	53	Generalized high mutability	202
<i>mutT</i>	Mutator	1	Generalized high mutability; specifically induces AT $\rightarrow$ CG transversions	41, 97, 202, 206
<i>nadA</i>	Nicotinamide adenine dinucleotide	17	<i>nicA</i> ; nicotinic acid requirement	1, 215
<i>nadB</i>	Nicotinamide adenine dinucleotide	49	<i>nicB</i> ; nicotinic acid requirement	118, 218
<i>nadC</i>	Nicotinamide adenine dinucleotide	2	Quinolinate phosphoribosyl transferase	85, I
<i>nalA</i>	Nalidixic acid	42	Resistance or sensitivity to nalidixic acid	94
<i>nalB</i>	Nalidixic acid	51	Resistance or sensitivity to nalidixic acid	94
<i>nar</i>	Nitrate reductase	—	See <i>chl</i>	
<i>narE</i>	—	18	Nitrate reductase (see also <i>chl</i> )	175, 224
<i>nek</i>	—	63	Resistance to neomycin and kanamycin (30S ribosomal protein)	10
<i>nic</i>	—	—	See <i>nad</i>	
<i>oldA</i>	Oleate degradation	75	<i>old-30</i> ; thiolase	162
<i>oldB</i>	Oleate degradation	75	<i>old-64</i> ; hydroxyacyl-coenzyme A dehydrogenase	162
<i>oldD</i>	Oleate degradation	34	<i>old-88</i> ; acyl-coenzyme A synthetase	162
<i>pabA</i>	<i>p</i> -Aminobenzoate	65	Requirement	109, 232
<i>pabB</i>	<i>p</i> -Aminobenzoate	30	Requirement	109
<i>pan</i>	Pantothenic acid	2	Requirement	53

TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>pdxA</i>	Pyridoxine	1	Requirement	215
<i>pdxB</i>	Pyridoxine	44	Requirement	54
<i>pdxC</i>	Pyridoxine	20	Requirement	B, D
<i>psk</i>	—	76	Structural or regulatory gene for fructose 6-phosphate kinase	146
<i>pgi</i>	—	79	Phosphoglucoisomerase	77
<i>pgl</i>	—	17	6-Phosphogluconolactonase	127
<i>pheA</i>	Phenylalanine	50	Prephenic acid dehydratase	170, 214, 215
<i>pheS</i>	Phenylalanine	33	<i>phe-act</i> ; phenylalanyl tRNA synthetase	22
<i>phoA</i>	Phosphatase	11	<i>P</i> ; alkaline phosphatase	60, D
<i>phoR</i>	Phosphatase	11	<i>R1 pho, R1</i> ; regulatory gene	60, D
<i>phoS</i>	Phosphatase	74	<i>R2 pho, R2</i> ; regulatory gene	8, 60
<i>phr</i>	Photoreactivation	(17)	Photoreactivation of UV-damaged DNA (K12-B hybrids)	222
<i>pil</i>	Pili	88	<i>fim</i> ; presence or absence of pili (fimbriae)	134
<i>pnp</i>	—	61	Polynucleotide phosphorylase	180
<i>polA</i>	Polymerase	75	DNA polymerase	50, 92
<i>por</i>	P1 restriction	89	Restriction of phage P1 DNA	236
<i>ppc</i>	—	77	<i>glu, asp</i> ; succinate, aspartate, or glutamate requirement; phosphoenolpyruvate carboxylase	87, 88, 118
<i>pps</i>	—	33	Utilization of pyruvate or lactate; phosphopyruvate synthetase	25
<i>prd</i>	Propanediol	53	1,2-Propanediol dehydrogenase	237, J
<i>proA</i>	Proline	7	<i>pro</i> ; block prior to L-glutamate semi-aldehyde	46, 214
<i>proB</i>	Proline	9	<i>pro<sub>2</sub></i> ; block prior to L-glutamate semi-aldehyde	46, 214
<i>proC</i>	Proline	10	<i>pro<sub>3</sub></i> ; <i>Pro2</i> ; probably $\Delta$ -pyrroline-5-carboxylate reductase	46
<i>pup</i>	—	89	Purine nucleoside phosphorylase	4
<i>purA</i>	Purine	82	<i>ade<sub>k</sub>, Ad<sub>4</sub></i> ; adenylosuccinic acid synthetase	70, 118
<i>purB</i>	Purine	23	<i>ade<sub>h</sub></i> , adenylosuccinase	205, 211, 214
<i>purC</i>	Purine	48	<i>ade<sub>o</sub></i> ; phosphoribosyl-aminoimidazole-succinocarboxamide synthetase	151, 211
<i>purD</i>	Purine	78	<i>adth<sub>o</sub></i> ; phosphoribosylglycineamide synthetase <sup>c</sup>	211, 214
<i>purE</i>	Purine	13	<i>ade<sub>3</sub></i> ; <i>ade<sub>f</sub></i> ; <i>Pur<sub>2</sub></i> ; phosphoribosyl-aminoimidazole carboxylase	56, 211
<i>purF</i>	Purine	44	<i>purC</i> , <i>ade<sub>u,s</sub></i> ; phosphoribosyl-pyrophosphate amidotransferase <sup>c</sup>	211, 214, 215
<i>purG</i>	Purine	48	<i>adth<sub>o</sub></i> ; phosphoribosylformylglycine-amidine synthetase <sup>c</sup>	211, 218
<i>purH</i>	Purine	78	<i>ade<sub>i</sub></i> ; phosphoribosyl-aminoimidazole-carboxamide formyltransferase	211
<i>purI</i>	Purine	49	Aminoimidazole ribotide synthetase <sup>c</sup>	217, 218
<i>pyrA</i>	Pyrimidine	0	<i>cap, arg + ura</i> ; glutamino-carbamoyl-phosphate synthetase	17, 214, 215
<i>pyrB</i>	Pyrimidine	84	Aspartate transcarbamylase	17, 214
<i>pyrC</i>	Pyrimidine	22	Dihydroorotase	17, 205
<i>pyrD</i>	Pyrimidine	21	Dihydroorotic acid dehydrogenase	17, 205
<i>pyrE</i>	Pyrimidine	72	Orotidylic acid pyrophosphorylase	192, 214
<i>pyrF</i>	Pyrimidine	25	Orotidylic acid decarboxylase	205
<i>rac</i>	Recombination activation	29	Suppressor of <i>recB</i> and <i>recC</i> mutant phenotype	K
<i>ram</i>	Ribosomal ambiguity	64	Nonspecific suppression of all nonsense codons	183

TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>ras</i>	Radiation sensitivity	(11)	Sensitivity to UV and X-ray irradiation	227
<i>rbs</i>	Ribose	74	Utilization of D-ribose	215
<i>recA</i>	Recombination	52	Ultraviolet sensitivity and competence for genetic recombination	233
<i>recB</i>	Recombination	55	Ultraviolet sensitivity and competence for genetic recombination	66, 106, 233, 234
<i>recC</i>	Recombination	55	Ultraviolet sensitivity and competence for genetic recombination	66, 234
<i>ref</i>	Refractory	(88)	<i>refII</i> ; specific tolerance to colicin E2	105
<i>rel</i>	Relaxed	54	<i>RC</i> ; regulation of RNA synthesis	5, 74
<i>rep</i>	Replication	74	Inhibition of lytic replication of temperate phages	32
<i>rhaA</i>	Rhamnose	76	L-Rhamnose isomerase	88, 172
<i>rhaB</i>	Rhamnose	76	L-Rhamnulokinase	88, 172
<i>rhaC</i>	Rhamnose	76	Regulatory gene	88, 172
<i>rhaD</i>	Rhamnose	76	L-Rhamnulose-1-phosphate aldolase	88, 172
<i>rif</i>	Rifampicin	77	DNA-dependent RNA polymerase sensitivity to rifampicin	16, 55, L
<i>rns</i>	Ribonuclease	15	Ribonuclease I	181
<i>rts</i>	—	77	<i>ts-9</i> ; altered electrophoretic mobility of 50S ribosomal subunit	75
<i>serA</i>	Serine	57	3-Phosphoglyceric acid dehydrogenase	214, 215, 220
<i>serB</i>	Serine	89	Phosphoserine phosphatase	215, 220
<i>serS</i>	Serine	(18)	Seryl-tRNA synthetase	104
<i>shiA</i>	Shikimic acid	38	Shikimate and dehydroshikimate permease	171
<i>som</i>	Somatic	(37)	<i>O</i> ; somatic ( <i>O</i> ) antigens	160
<i>spc</i>	Spectinomycin	64	Resistance or sensitivity to spectinomycin	7, 10, 75, 232
<i>speB</i>	Spermidine	57	Putrescine (or spermidine) requirement; agmatine ureohydrolase	M
<i>strA</i>	Streptomycin	64	Resistance, dependence, or sensitivity; "K-character" of the 30S ribosomal subunit	75, 118, 183, 196
<i>stv</i>	Streptovaricin	77	DNA-dependent RNA polymerase sensitivity to streptovaricin	241
<i>sucA</i>	Succinate	17	<i>suc</i> , <i>lys</i> + <i>met</i> ; succinate requirement; $\alpha$ -ketoglutarate dehydrogenase (decarboxylase component)	100, 101, 215
<i>sucB</i>	Succinate	17	<i>suc</i> , <i>lys</i> + <i>met</i> ; succinate requirement; $\alpha$ -ketoglutarate dehydrogenase (dihydrolypoyltranssuccinylase component)	100, 101
<i>supB</i>	Suppressor	16	<i>sub</i> ; suppressor of <i>ochre</i> mutation (not identical to <i>supL</i> )	24
<i>supC</i>	Suppressor	25	<i>suc</i> ; suppressor of <i>ochre</i> mutation (possibly identical to <i>supO</i> )	24, 81, 205, 212
<i>supD</i>	Suppressor	(38)	<i>suI</i> , <i>Su-1</i> ; suppressor of <i>amber</i> mutations	205, 212
<i>supE</i>	Suppressor	16	<i>suII</i> ; suppressor of <i>amber</i> mutations	68, 205
<i>supF</i>	Suppressor	25	<i>suIII</i> , <i>Su-3</i> ; suppressor of <i>amber</i> mutations	83, 212
<i>supG</i>	Suppressor	16	<i>Su-5</i> ; suppressor of <i>ochre</i> mutations	81
<i>supH</i>	Suppressor	38		61, 63
<i>supL</i>	Suppressor	17	Suppressor of <i>ochre</i> mutations	62, 63
<i>supM</i>	Suppressor	78	Suppressor of <i>ochre</i> mutations	62, 63
<i>supN</i>	Suppressor	45	Suppressor of <i>ochre</i> mutations	62, 63, 144
<i>supO</i>	Suppressor	25	Suppressor of <i>ochre</i> mutations (possibly identical to <i>supC</i> )	62, 63
<i>supT</i>	Suppressor	55		63
<i>supU</i>	Suppressor	74	<i>su7</i> ; suppressor of <i>amber</i> mutations	208
<i>supV</i>	Suppressor	74	<i>su8</i> ; suppressor of <i>ochre</i> mutations	208

TABLE 1.—Continued

Gene symbol	Mnemonic	Map position (min) <sup>a</sup>	Alternate gene symbols; phenotypic trait affected	References <sup>b</sup>
<i>tdk</i>	—	25	Deoxythymidine kinase	102
<i>tfrA</i>	T-four	(8)	ϕ <sup>r</sup> ; resistance or sensitivity to phages T4, T3, T7, and λ	46, D
<i>thiA</i>	Thiamine	78	<i>thi</i> ; synthesis of thiazole	122, 211
<i>thiB</i>	Thiamine	(78)	Thiamine phosphate pyrophosphorylase	122
<i>thiO</i>	Thiamine	(78)	Probable operator locus for <i>thiA</i> and <i>thiB</i> genes	123
<i>thrA</i>	Threonine	0	Block between homoserine and threonine	117, 134
<i>thrD</i>	Threonine	0	<i>HS</i> ; aspartokinase I-homoserine dehydrogenase I complex	89, 165, 166
<i>thyA</i>	Thymine	55	Thymidylate synthetase	6, 111, 215
<i>tkt</i>	—	(55)	Transketolase	121
<i>tnaA</i>	—	73	<i>ind</i> ; tryptophanase	84, 168
<i>tnaR</i>	—	73	<i>Rtna</i> ; regulatory gene	84
<i>tolA</i>	Tolerance	17	<i>cim</i> ; <i>tol-2</i> ; tolerance to colicins E2, E3, A, and K	147, 148, 153, 179
<i>tolB</i>	Tolerance	17	<i>tol-3</i> ; tolerance to colicins E1, E2, E3, A, and K	147, 148, 153, 179
<i>tolC</i>	Tolerance	58	<i>colE1-i</i> , <i>tol-8</i> , <i>ref1</i> ; specific tolerance to colicin E1	35, 105, 148, N
<i>tonA</i>	T-one	2	<i>T1</i> , <i>T5 rec</i> ; resistance or sensitivity to phages T1 and T5	46, 53, 134, E
<i>tonB</i>	T-one	25	<i>T1 rec</i> ; resistance to phages T1, ϕ80 and colicins B, I, V; active transport of Fe	91, 204, 230, 239
<i>tpp</i>	—	89	<i>deoA</i> , <i>TP</i> ; thymidine phosphorylase	4, 72, 131
<i>trpA</i>	Tryptophan	25	<i>tryp-2</i> ; tryptophan synthetase, A protein	113, 238, 239
<i>trpB</i>	Tryptophan	25	<i>tryp-1</i> ; tryptophan synthetase, B protein	113, 238, 239
<i>trpC</i>	Tryptophan	25	<i>tryp-3</i> ; indole-3-glycerol phosphate synthetase	113, 238, 239
<i>trpD</i>	Tryptophan	25	<i>tryE</i> ; phosphoribosyl anthranilate transferase	58, 113, 142, 238
<i>trpE</i>	Tryptophan	25	<i>tryD</i> , <i>anth</i> , <i>tryp-4</i> ; anthranilate synthetase	113, 142, 238, 239
<i>trpO</i>	Tryptophan	25	Operator locus	142, 209, 238
<i>trpR</i>	Tryptophan	90	<i>Rtry</i> ; regulatory gene	37, 114, B
<i>trpS</i>	Tryptophan	65	Tryptophanyl-tRNA synthetase	57, 115
<i>tsx</i>	T-six	11	<i>T6 rec</i> ; resistance or sensitivity to phage T6 and colicin K	46, 56, 79, 134
<i>tyrA</i>	Tyrosine	50	Prephenic acid dehydrogenase	170, 214, 215
<i>tyrR</i>	Tyrosine	27	Regulatory gene for <i>tyrA</i> and <i>aroF</i> genes	229
<i>tyrS</i>	Tyrosine	32	Tyrosyl-tRNA synthetase	194
<i>ubiA</i>	Ubiquinone	83	4-Hydroxybenzoate → 3-octaprenyl 4-hydroxybenzoate (OPHB)	42
<i>ubiB</i>	Ubiquinone	75	2-Octaprenylphenol → ubiquinone	42, 43
<i>ubiD</i>	Ubiquinone	75	OPHB decarboxylase	43
<i>uhp</i>	—	72	Uptake of hexose phosphates	126
<i>uraP</i>	Uracil	50	Uracil permease	K
<i>uvrA</i>	Ultraviolet	80	<i>dar-3</i> ; repair of ultraviolet radiation damage to DNA	107, 222
<i>uvrB</i>	Ultraviolet	18	<i>dar-1,6</i> ; repair of ultraviolet radiation damage to DNA	1, 107, 222
<i>uvrC</i>	Ultraviolet	37	<i>dar-4,5</i> ; repair of ultraviolet radiation damage to DNA	12, 107, 222
<i>uvrD</i>	Ultraviolet	74	<i>dar-2</i> , <i>rad</i> ; repair of UV radiation damage to DNA	14, 157, 222
<i>valS</i>	Valine	84	<i>val-act</i> ; valyl-tRNA synthetase	19, 216
<i>xyl</i>	Xylose	70	Utilization of D-xylose	118, 214
<i>zwf</i>	Zwischenferment	(35)	Glucose-6-phosphate dehydrogenase	167

**Region from 15 to 30 min.** The *kdp* loci at minute 16 represent four contiguous complementation groups of mutants that are defective in unknown functions affecting the utilization of potassium ion (68). Mutants of *E. coli* strain B that are specifically deficient in potassium accumulation (*kac* mutants) have also been mapped in the vicinity of minute 16 (28). The correct order of the genes *supE*, *kdpA-D*, *gltA*, *suc*, and *gal* is now known from transduction data presented in references 68 and 100. The *suc* locus for  $\alpha$ -ketoglutarate dehydrogenase has been resolved into two cistrons, *sucA* and *B*, which are functionally analogous to the *aceE* and *F* loci for pyruvate dehydrogenase at minute 2 (101). A new transfer ribonucleic acid (tRNA) synthetase gene, *serS*, has been mapped approximately between minutes 17 and 19 (104). A deletion analysis of the region near *gal* at minute 17 (1) has shown that certain deletions can generate *gal-aroG* multisite mutants that have no requirement for nicotinic acid. Accordingly, the *nadA* (formerly *nicA*) locus has been shifted from its previous position between *gal* and *aroG* (215) to a point slightly anticlockwise to *aroG*. This report (1), together with a follow-up study by Puig et al. (175), also shows that the correct position of the *chlA* gene is now just slightly clockwise from *uvrB* at minute 17.5. Two additional sites of pleiotropic mutations affecting nitrate-chlorate reductase activity, *chlD* [also known as *narF* (1, 224)] and *narE* (175, 224), also map in the 17- to 18-min segment. In vitro complementation tests (15) suggest that the various pleiotropic *chl* genes may code for different structural "subassemblies" which can unite to form a complex endowed with several catalytic functions. A new gene, *pgl*, has been mapped between *att $\lambda$*  and *chlD* by means of overlapping deletion analysis (127). The old *bioA* locus at minute 17.5 is now recognized to contain at least six contiguous genes concerned with biotin synthesis (1, 51, 65, 182). Moreover, these *bio* genes appear to comprise an operon since at least some of the enzymes are regulated in coordinate fashion (64). Current nomenclature, which assigns the symbol *bioB* to one of the genes in this operon, requires us to change the designation of the unlinked *bio* locus at minute 65 from *bioB* to *bioH*.

Two new genes, *cmlA* and *cmlB*, that determine resistance to chloramphenicol are located at minutes 18.5 and 21, respectively (178). The *pdxC* locus at minute 20 represents a third and hitherto unmapped gene for pyridoxine requirement. The *Pdx<sup>-</sup>* allele was present in strain X961, kindly given to us by R. Curtiss. Preliminary results (A. L. Taylor, unpublished data) indicate

that *pdxC* is very close to *aroA* (95 to 97% cotransduction of these two markers). It is not known, however, whether *pdxC* lies clockwise or anticlockwise to *aroA*. Other additions to the 22- to 25-min segment of the map include *capS* (139), *cat* (219), and *hemA* (190, 191). The *chlC* locus, (174), also known as *narC* (93), has now been mapped with greater precision at a point between *hemA* and *supC* (93). A new regulatory gene, *tyrR*, that controls expression of the structural genes *aroF* and *tyrA*, has been mapped at minute 27 by time of entry with Hfr Hayes (229). The *tyrR* locus appears to be quite distinct from the tyrosyl-tRNA synthetase gene, *tyrS*, which maps 5 min away at minute 32 (194). The *rac* gene at minute 29 has the curious effect of suppressing the recombination-deficient phenotype of mutants at the *recB* and *recC* loci (B. Low, personal communication).

**Region from 30 to 45 min.** The symbol *oldD* is proposed here to identify the locus of the *old-88* allele which was recently mapped at minute 34 by time-of-entry experiments (162). Two genes for chemotactic motility, which had been previously designated *motA* and *B* (215), are now renamed *cheA* and *cheB* in accord with the nomenclature of Armstrong and Adler (12). The symbol *mot* at minute 36 now serves to identify flagellar paralysis mutants (12). This latter report also describes a third chemotaxis gene, *cheC*, at minute 37 and it further proposes that the symbol *fla* be reserved to denote unmapped genes that control the formation of flagella. The *nalA* locus at minute 42 is one of two new genes which determine resistance to nalidixic acid; the second gene, *nalB*, maps at minute 51 (94). The *pdxB* locus has been mapped by three-point transduction crosses at minute 44, between *purF* and *aroC* (54). A mutation affecting unsaturated fatty acid biosynthesis (*fabB*) has also been mapped in the short interval between *aroC* and *purF*, but the relative order of *fabB* and *pdxB* is not known (69).

**Region from 45 to 60 min.** The position and orientation of *dapA* and *dapE* at minute 47 are based on the results of three-point transduction crosses performed in this laboratory (A. I. Bukhari and A. L. Taylor, in preparation). The locus previously designated as *dapB* (215) is renamed *dapE*, as it codes for the fifth enzyme of the diaminopimelic acid pathway (38). Nijkamp and Oskamp (152) recently presented further evidence that the two genes for guanine biosynthesis at minute 48 comprise a true operon. The *nicB* locus, now renamed *nadB* as recommended by Tritz et al. (218), has been moved from its former incorrect location at minute 46 (215) to minute 49.5. In addition to mapping *nadB*,

Tritz et al. (217, 218) identified and mapped a new purine locus of *E. coli*, *purI*, at a point between *glyA* and *nadB*. Three new cysteine genes, *cysH*, *P*, and *Q*, have been mapped near *cysC* at minute 53 (120). Although fine-structure genetics of the region remains to be done, it seems likely that all four of these *cys* loci will make up a contiguous gene cluster analogous to the *cysCDHIJ* cluster in *S. typhimurium* (189). Three genes that affect genetic recombination have been accurately mapped. The *recA* locus is placed at minute 51.5, slightly closer to *cysC* than to *pheA* on the basis of transduction data reported by Willetts et al. (233). The position and orientation of the closely linked *recB* and *recC* loci at minute 54.5 were determined by means of multifactor transduction crosses (66, 234). A recent study by Oishi (158) indicates that mutations in the *recB* and *recC* genes are associated with the loss of a specific deoxyribonuclease activity. The structural gene for diaminoipimelic acid decarboxylase at minute 55 has been divided into two regions, *lysA* and *lysB*, to identify two phenotypically distinct classes of mutants which will be described in detail elsewhere (A. I. Bukhari and A. L. Taylor, *in preparation*). A new structural (or regulatory) gene for agmatine ureohydrolase, which catalyzes the second step in spermidine synthesis from arginine, has been named *speB* and mapped near *serA* at minute 56.5; its orientation with respect to *serA* and *argP* is not yet known (W. K. Maas, *personal communication*). The *tolC* locus, also known as *refI* (105), has been mapped with good precision at minute 58 both by time-of-entry and by transduction methods (E. Whitney, *personal communication*).

**Region from 60 to 75 min.** Mutants resistant to the antimetabolite 5-methyl tryptophan have been mapped very close to *argG* at minute 61, but the orientation of *mtr* was not determined (103). The *mtr* mutants are phenotypically distinct from resistant strains resulting from mutations in the *trpR* or *trpO* genes (103). The region from minutes 63 to 64 contains at least four genes that specify components of the 30S ribosome: these are *nek* (10), *ram* (183), *spc* (10, 75), and *strA* (75). The orientation of *ram*, *spc*, and *strA* with respect to each other and to *aroE* is based on recombination and transduction data presented in several recent reports (7, 10, 183, 232). The *nek* locus appears to be closely linked and anticlockwise to *spc*, but its orientation relative to *aroE* is not known (10).

The *argD* locus, previously mapped near the *strA* region (226), is now precisely located at minute 64.5 by transduction analyses done in this laboratory (A. L. Taylor and S. Paigen, *unpub-*

*lished data*). Following the lead of Itikawa et al. (112), we obtained a number of *argD* mutants of *E. coli* among proline-independent "revertants" of a proline auxotroph blocked at a step prior to glutamic  $\gamma$ -semialdehyde. One of these double mutants (strain AT 3141, *proA*<sup>-</sup> *argD*<sup>-</sup>) was used in multifactor transduction crosses with strains carrying mutant alleles at several loci close to *strA*. The results, in summary form, of one cross showed that *aroE* was cotransduced with *strA* and *argD* at frequencies at 64 and 14%, respectively; the gene order indicated was *aroE-strA-argD*. In a second cross, *cysG* was cotransduced at a frequency of 41% with *argD*, 22% with *strA*, and 13% with *malP*, and the indicated gene order was *strA-argD-cysG-malP*.

The structural gene for a tryptophanyl-tRNA synthetase lies about midway between *strA* and *malP*, as shown by transduction studies (57, 115). The locus formerly designated as *malA* at minute 66 (215) has been shown to contain two structural genes, *malQ* and *malP*, and a regulatory gene, *malT* (95). Present evidence suggests that *malQ* and *malP* form an operon, and that *malT* probably does not belong to this operon, even though it maps immediately adjacent to *malP* (95). As explained earlier in this survey, the *bioB* gene at minute 65.5 is renamed *bioH*. Three closely linked genes that affect the formation of glycogen have been mapped at minute 66 between *glpD* and *asd* (33, 203); orientation of the *glgA*, *B*, and *C* genes has not yet been determined and it is not known whether the three genes represent an operon (33). The *dct* locus, which controls the transport of several four-carbon dicarboxylic acids, maps at minute 69 by time-of-entry determination (124). A new structural (or regulatory) gene for lactate dehydrogenase has been named *lct* and mapped at minute 71.5 by transduction (163). Other new genes in the 71- to 72-min region include *gad* (137) and the locus for a glutamyl-tRNA synthetase (*gltE*) with unknown orientation with respect to *lct* (E. J. Murgola, *personal communication*). The *uhp* (126) and *cysE* (120) loci both map near *pyrE* at minute 72, but their orientation with respect to each other and to *pyrE* is uncertain. The *gltC* locus is now placed at a corrected position between *pyrE* and *tna* (136). A new gene, *asn*, maps at minute 73.5 midway between *bgl* and *ilv* and appears to be the earliest marker known to be transferred by Hfr strain AB313 (34). Two unusual nonsense suppressor alleles, *su7* and *su8* (symbolized here as *supU* and *supV*, respectively), have been mapped recently at a point close to the *ilv* operon at minute 74 (208). There have been at least four separate reports on mutations that map near *metE* at minute 74.5

and which cause altered sensitivity to ultraviolet or X-irradiation. The genes involved in these various mutations have been called *rad* (14), *dar-2* (222), *uvrD* (157), and most recently *rep* (32). It may be that some or even all of these mutations will turn out to be different alleles of a single locus, but current data are too fragmentary to permit such an interpretation at this time. A fifth gene (*polA*) that also maps near *metE* and which also affects sensitivity to ultraviolet light has been identified as the probable structural gene for DNA polymerase (50, 92). These workers conclude that *polA* and *uvrD* are likely to be separate genes because of phenotypic differences between the two types of mutants. It is also clear that *polA* and *rep* must be separate genes because the former locus maps clockwise to *metE* (92), whereas the latter maps anticlockwise to *metE* (32). Two genes concerned with ubiquinone biosynthesis, *ubiB* and *ubiD*, have been mapped within 0.5 min of the clockwise side of *metE* (43). The *old-30* and *old-64* alleles described by Overath et al. (162) are designated here as *oldA* and *oldB*, respectively; both genes are located between *metE* and *rha*, but their orientation relative to other nearby markers is not known.

**Region from 75 to 90 min.** Morrissey and Fraenkel (146) showed that *pfk* maps between *rha* and *glpK* at minute 76 in a detailed report that lists the cotransduction frequencies of several other standard markers in the 76- to 78-min region. Recent studies on antibiotic-resistant mutants of *E. coli* that possess altered deoxyribonucleic acid (DNA)-dependent RNA polymerases seem to indicate that mutations affecting this enzyme can occur in two distinct regions. On the one hand, rifampicin-resistant mutants, designated *rif*, cotransduce with the *argH* locus at high frequencies of 65 to 77%, and preliminary evidence favors placing *rif* at minute 77 between *argH* and *metB* (16, 55; W. F. Doolittle, *personal communication*). On the other hand, streptovaricin-resistant mutants, designated *stv* (241), and streptolydigin-resistant mutants (193) cotransduce with *argH* at lower frequencies of 20 to 35%, and the first indication is that *stv* maps clockwise to *argH*, away from *rif* (241). The *ace* operon, together with a closely linked regulatory gene for the operon (*iclR*, also known as *aceD*), is placed near minute 78 between *metA* and *pgi* on the basis of transduction data (26, 223). The structural gene for aspartase, *aspA*, has been mapped near minute 82 by time of entry, but its position relative to other nearby markers is not known (138). Both the position and orientation of the *ampA* and *purA* genes near minute 82 have now been corrected with the aid of new transduc-

tion data reported by Eriksson-Grenberg (70). A third gene concerned with ubiquinone formation, *ubiA*, maps near minute 83 by time-of-entry determinations, but its exact position relative to standard markers such as *purA* or *fdp* is not known (42). The *mel-7* and *mel-4* alleles described by Schmitt (195) are symbolized here as *melA* and *melB*, respectively; time-of-entry experiments indicate that both genes map near minute 84, but precise ordering of the *mel* loci with respect to other nearby markers has not been done (195). The *melB* locus is of particular interest, as it specifies the hitherto unmapped second thio-methylgalactoside permease described by Prestidge and Pardee (173). Preliminary genetic studies of Holland and Threlfall (105) showed that certain pleiotropic mutations (*ref*), which result in both increased sensitivity to ultraviolet irradiation and specific tolerance to colicin E2, map in the general vicinity of minutes 88 to 89. A gene responsible for specific host restriction of phage P1 (*por*) has been mapped at minute 89 (236), very close to the site of other restriction markers (23, 39, 128, 235) which are collectively identified here by the tentative symbol *hsp*.

Lomax and Greenberg (131) proposed the term *deo* operon to denote a cluster of three closely linked genes that code for enzymes involved in deoxyribonucleoside catabolism. The genes were mapped with the following orientation at a point about 0.5 min anticlockwise to *thr* at minute 90/0: *deoA*-(*deoB*)-*deoC*-*thr* (131). Subsequently, Ahmad and Pritchard (4) presented a more detailed genetic analysis of the region in which these three genes plus a fourth related gene, *pup*, were sequenced with respect to each other and also in relation to the nearest known outside markers, *hsp* and *serB*. The *deoA*, *B*, and *C* loci were termed *tpp*, *drm*, and *dra*, respectively, and the order deduced from many crosses was *hsp-dra-tpp-drm-pup-serB-thr*. Both reports (4, 131) concur that *dra*(*deoC*) and *tpp*(*deoA*) probably belong to a common operon; it is not certain, however, that *drm*(*deoB*) and *pup* also belong to this operon and there is some evidence to support a two-operon model (4). In view of these uncertainties, the relatively noncommittal gene symbols of Ahmad and Pritchard (4) have been adopted here in preference to the earlier *deo* convention (131). As the final entry in this survey, it should be noted that unpublished data from this laboratory show that the position of the *trpR* locus (37, 114) is anticlockwise to *thr* at minute 90/0.

#### CONCLUDING REMARKS

The circular genome of *E. coli* has a contour length of about 1,000  $\mu\text{m}$  (31), which is equivalent

to about  $3 \times 10^6$  nucleotide pairs per chromosome or approximately 3,000 genes, assuming the average gene contains 1,000 nucleotide pairs. If one ascribes an informational role to all of the DNA in the bacterial genome, it follows that the 310 genes identified thus far account for roughly 10% of the potential information content of *E. coli*.

As Fig. 1 shows, the distribution of genes on the map is now fairly uniform so that few genetically silent regions remain. The most conspicuous quiet region is the segment from minutes 3 to 9, and there are smaller silent areas at minutes 27-31, 39-42, and 85-88. For the most part, attempts to locate new markers in these regions will have to rely upon the time of entry approach to mapping. The rest of the map, however, now seems to contain a sufficient number of markers per minute of map length to assure the feasibility of mapping most new markers by transduction methods.

Although the present genetic map must inevitably contain some errors and imperfections, it is nevertheless substantially more accurate than previous maps. Much of the high precision of gene placement in Fig. 1 can be attributed to the widespread use of multifactor transduction crosses and overlapping deletion analyses in current genetic investigations. Continuing efforts along these lines will unquestionably lead to further clarification of the overall genetic structure of *E. coli*.

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#### LITERATURE CITED

- Adhya, S., P. Cleary, and A. Campbell. 1968. A deletion analysis of prophage lambda and adjacent genetic regions. *Proc. Nat. Acad. Sci. U.S.A.* 61:956-962.
- Adler, H. I., and A. A. Hardigree. 1964. Analysis of a gene controlling cell division and sensitivity to radiation in *Escherichia coli*. *J. Bacteriol.* 87:720-726.
- Adler, J., and A. D. Kaiser. 1963. Mapping of the galactose genes of *Escherichia coli* by transduction with phage P1. *Virology* 19:117-126.
- Ahmad, S. I., and R. H. Pritchard. 1969. A map of four genes specifying enzymes involved in catabolism of nucleosides and deoxynucleosides in *Escherichia coli*. *Mol. Gen. Genet.* 104:351-359.
- Alföldi, L., G. S. Stent, and R. C. Clowes. 1962. The chromosomal site of the RNA control (RC) locus in *Escherichia coli*. *J. Mol. Biol.* 5:348-355.
- Alikhanian, S. I., T. S. Iljina, E. S. Kaliaeva, S. V. Kaliaeva, S. V. Kameneva, and V. V. Sukhodolec. 1965. Mutants of *Escherichia coli* K12 lacking thymine. *Nature* 206:848-849.
- Anderson, P., Jr. 1969. Sensitivity and resistance to spectinomycin in *Escherichia coli*. *J. Bacteriol.* 100:939-947.
- Aono, H., and N. Otsuji. 1968. Genetic mapping of regulator gene *phoS* for alkaline phosphatase in *Escherichia coli*. *J. Bacteriol.* 95:1182-1183.
- Apirion, D. 1967. Three genes that affect *Escherichia coli* ribosomes. *J. Mol. Biol.* 30:255-275.
- Apirion, D., and D. Schlessinger. 1968. Mapping and complementation of three genes specifying 30S ribosomal components in *Escherichia coli*. *J. Bacteriol.* 96:1431-1432.
- Armstrong, J. B., and J. Adler. 1967. Genetics of motility in *Escherichia coli*: complementation of paralyzed mutants. *Genetics* 56:363-373.
- Armstrong, J. B., and J. Adler. 1969. Location of genes for motility and chemotaxis on the *Escherichia coli* genetic map. *J. Bacteriol.* 97:156-161.
- Ashworth, J. M., H. L. Kornberg, and D. L. Nothmann. 1965. Location of the structural gene for citrate synthase on the chromosome of *Escherichia coli* K12. *J. Mol. Biol.* 11:654-657.
- Axelrod, D. E., and H. I. Adler. 1967. Genetic factors that increase radiation resistance of *Escherichia coli*. *Bacteriol. Proc.*, p. 48.
- Azulay, E., and J. Puig. 1968. Reconstitution of enzymatically active particles from inactive soluble elements in *Escherichia coli* K12. *Biochem. Biophys. Res. Commun.* 33:1019-1024.
- Babinet, C., and H. Condamine. 1968. Mutants résistants a la rifampicine, modifiés dans leur DNA-RNA-polymérase. *C. R. Acad. Sci. Paris* 267:231-232.
- Beckwith, J. R., A. B. Pardee, R. Austrian, and F. Jacob. 1962. Coordination of the synthesis of the enzymes in the pyrimidine pathway of *E. coli*. *J. Mol. Biol.* 5:618-634.
- Beckwith, J. R., E. R. Signer, and W. Epstein. 1966. Transposition of the *lac* region of *E. coli*. *Cold Spring Harbor Symp. Quant. Biol.* 31:393-401.
- Böck, A., L. E. Faiman, and F. C. Neidhardt. 1966. Biochemical and genetic characterization of a mutant of *Escherichia coli* with a temperature-sensitive valyl ribonucleic acid synthetase. *J. Bacteriol.* 92:1076-1082.
- Böck, A., and F. C. Neidhardt. 1966. Isolation of a mutant of *Escherichia coli* with a temperature-sensitive fructose-1,6-diphosphate aldolase activity. *J. Bacteriol.* 92:464-469.
- Böck, A., and F. C. Neidhardt. 1966. Location of the structural gene for glycyl ribonucleic acid synthetase by means of a strain of *Escherichia coli* possessing an unusual enzyme. *Z. Vererbungsl.* 98:187-192.
- Böck, A., and F. C. Neidhardt. 1967. Genetic mapping of phenylalanyl-sRNA synthetase in *Escherichia coli*. *Science* 157:78-79.
- Boyer, H. 1964. Genetic control of restriction and modification in *Escherichia coli*. *J. Bacteriol.* 88:1652-1660.
- Brenner, S., and J. R. Beckwith. 1965. *Ochre* mutants, a new class of suppressible nonsense mutants. *J. Mol. Biol.* 13:629-637.
- Brice, C. B., and H. L. Kornberg. 1967. Location on the chromosome of *Escherichia coli* of a gene specifying phosphopyruvate synthase activity. *Biochim. Biophys. Acta* 136:412-414.
- Brice, C. B., and H. L. Kornberg. 1968. Genetic control of isocitrate lyase activity in *Escherichia coli*. *J. Bacteriol.* 96:2185-2186.
- Brown, K. D. 1968. Regulation of aromatic amino acid biosynthesis in *Escherichia coli* K12. *Genetics* 60:31-48.
- Burmeister, M. 1969. Chromosomal location of a gene involved in potassium ion uptake in *Escherichia coli* B. *J. Bacteriol.* 100:796-802.
- Buttin, G. 1962. Sur la structure de l'operon galactose chez *Escherichia coli* K12. *C. R. Acad. Sci. Paris* 255:1233-1235.
- Buttin, G. 1963. Mécanismes régulateurs dans la biosynthèse des enzymes du métabolisme du galactose chez *Escherichia coli* K12. II. Le déterminisme génétique de la régulation. *J. Mol. Biol.* 7:183-205.
- Cairns, J. 1963. The bacterial chromosome and its manner



- of replication as seen by autoradiography. *J. Mol. Biol.* 6:208-213.
32. Calendar, R., B. Lindqvist, G. Sironi, and A. J. Clark. 1970. Characterization of REP<sup>-</sup> mutants and their interaction with P2 phage. *Virology* 40:72-83.
  33. Cattaneo, J., M. Damotte, N. Sigal, F. Sanchez-Medina, and J. Puig. 1969. Genetic studies of *Escherichia coli* K12 mutants with alterations in glycogenesis and properties of an altered adenosine diphosphate glucose pyrophosphorylase. *Biochem. Biophys. Res. Commun.* 34:694-701.
  34. Cedar, H., and J. H. Schwartz. 1969. The asparagine synthetase of *Escherichia coli*. I. Biosynthetic role of the enzyme, purification and characterization of the reaction products. *J. Biol. Chem.* 244:4112-4121.
  35. Clowes, R. C. 1965. Transmission and elimination of colicin factors and some aspects of immunity to colicin E1 in *Escherichia coli*. *Zentralbl. Bakteriol. Parasitenk. Abt. I. Orig.* 196:152-160.
  36. Cohen, A., D. P. Allison, H. I. Adler, and R. Curtiss, III. 1967. Genetic transfer to mini-cells of *Escherichia coli* K-12. *Genetics* 56:550-551.
  37. Cohen, G., and F. Jacob. 1959. Sur la répression de la synthèse des enzymes intervenant dans la formation du tryptophane chez *Escherichia coli*. *C. R. Acad. Sci. Paris* 248:3490-3492.
  38. Cohen, G. N., J. C. Patte, P. Truffa-Bachi, C. Sawas, and M. Doudoroff. 1965. Repression and end-product inhibition in a branched biosynthetic pathway, p. 243-253. In *Mécanismes de régulation des activités cellulaires chez les micro-organismes*. Centre National de la Recherche Scientifique, Paris.
  39. Colson, C., S. W. Glover, N. Symonds, and K. A. Stacey. 1965. The location of the genes for host-controlled modification and restriction in *Escherichia coli* K-12. *Genetics* 52:1043-1050.
  40. Cooper, P. H., I. N. Hirshfield, and W. K. Maas. 1969. Map location of arginyl-tRNA synthetase mutations in *Escherichia coli* K-12. *Mol. Gen. Genet.* 104:383-390.
  41. Cox, E. C., and C. Yanofsky. 1969. Mutator gene studies in *Escherichia coli*. *J. Bacteriol.* 100:390-397.
  42. Cox, G. B., F. Gibson, and J. Pittard. 1968. Mutant strains of *Escherichia coli* K-12 unable to form ubiquinone. *J. Bacteriol.* 95:1591-1598.
  43. Cox, G. B., I. G. Young, L. M. McCann, and F. Gibson. 1969. Biosynthesis of ubiquinone in *Escherichia coli* K-12: location of genes affecting the metabolism of 3-oxotaprenyl-4-hydroxybenzoic acid and 2-octaprenyl-phenol. *J. Bacteriol.* 99:450-458.
  44. Cozzarelli, N. R., W. B. Freedberg, and E. C. C. Lin. 1968. Genetic control of the L- $\alpha$ -glycerophosphate system in *Escherichia coli*. *J. Mol. Biol.* 31:371-387.
  45. Cozzarelli, N. R., and E. C. C. Lin. 1966. Chromosomal location of the structural gene for glycerol kinase in *Escherichia coli*. *J. Bacteriol.* 91:1763-1766.
  46. Curtiss, R., III. 1965. Chromosomal aberrations associated with mutations to bacteriophage resistance in *Escherichia coli*. *J. Bacteriol.* 89:28-40.
  47. Curtiss, R., III, L. J. Charamella, C. M. Berg, and P. E. Harris. 1965. Kinetic and genetic analysis of D-cycloserine inhibition and resistance in *Escherichia coli*. *J. Bacteriol.* 90:1238-1250.
  48. Dalal, F. R., and J. S. Gots. 1965. Glycine auxotrophs of *Salmonella typhimurium*. *Bacteriol. Proc.*, p. 89.
  49. Davies, J., and F. Jacob. 1968. Genetic mapping of the regulator and operator genes of the *Lac* operon. *J. Mol. Biol.* 36:413-417.
  50. De Lucia, P., and J. Cairns. 1969. Isolation of an *E. coli* strain with a mutation affecting DNA polymerase. *Nature* 224:1164-1166.
  51. Del Campillo-Campbell, A., G. Kayajanian, A. Campbell, and S. Adhya. 1967. Biotin-requiring mutants of *Escherichia coli* K-12. *J. Bacteriol.* 94:2065-2066.
  52. 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.
  53. Demerec, M., E. L. Lahr, T. Miyake, I. Goldman, E. Balbinder, S. Banic, K. Hashimoto, E. V. Glanville, and J. D. Gross. 1958. Bacterial genetics. Carnegie Inst. Wash. Year Book 57:390-406.
  54. Dempsey, W. B. 1969. Characterization of pyridoxine auxotrophs of *Escherichia coli*: chromosomal position of linkage group I. *J. Bacteriol.* 100:295-300.
  55. di Mauro, E., L. Snyder, P. Marino, A. Lamberti, A. Coppo, and G. P. Tocchini-Valentini. 1969. Rifampicin sensitivity of the components of DNA-dependent RNA polymerase. *Nature* 222:533-537.
  56. Donch, J., and J. Greenberg. 1968. Genetic analysis of *lon* mutants of strain K-12 of *Escherichia coli*. *Mol. Gen. Genet.* 103:105-115.
  57. Doolittle, W. F., and C. Yanofsky. 1968. Mutants of *Escherichia coli* with an altered tryptophanyl-transfer ribonucleic acid synthetase. *J. Bacteriol.* 95:1283-1294.
  58. Doy, C. H., A. Rivera, Jr., and P. R. Srinivasan. 1961. Evidence for the enzymatic synthesis of N-(5'-phosphoribosyl) anthranilic acid, a new intermediate in tryptophan biosynthesis. *Biochem. Biophys. Res. Commun.* 4:83-88.
  59. Dürwald, H., and H. Hoffmann-Berling. 1968. Endonuclease I-deficient and ribonuclease I-deficient *Escherichia coli* mutants. *J. Mol. Biol.* 34:331-346.
  60. Echols, H., A. Garen, S. Garen, and A. Torriani. 1961. Genetic control of repression of alkaline phosphatase in *E. coli*. *J. Mol. Biol.* 3:425-438.
  61. Eggertsson, G. 1968. Suppressors causing temperature sensitivity of growth in *Escherichia coli*. *Genetics* 60:269-280.
  62. Eggertsson, G. 1968. Mapping of ochre suppressors in *Escherichia coli*. *Genet. Res.* 11:15-20.
  63. Eggertsson, G., and E. A. Adelberg. 1965. Map positions and specificities of suppressor mutations in *Escherichia coli* K-12. *Genetics* 52:319-340.
  64. Eisenberg, M. A., and K. Krell. 1969. Dethiobiotin synthesis from 7,8-diaminopelargonic acid in cell-free extracts of a biotin auxotroph of *Escherichia coli* K-12. *J. Biol. Chem.* 244:5503-5509.
  65. Eisenberg, M. A., and C. Star. 1968. Synthesis of 7-oxo-8-aminopelargonic acid, a biotin vitamer, in cell-free extracts of *Escherichia coli* biotin auxotrophs. *J. Bacteriol.* 96:1291-1297.
  66. Emmerson, P. T. 1968. Recombination deficient mutants of *Escherichia coli* K12 that map between *thyA* and *argA*. *Genetics* 60:19-30.
  67. Englesberg, E., J. Irr, J. Power, and N. Lee. 1965. Positive control of enzyme synthesis by gene C in the L-arabinose system. *J. Bacteriol.* 90:946-957.
  68. Epstein, W., and M. Davies. 1970. Potassium-dependent mutants of *Escherichia coli* K-12. *J. Bacteriol.* 101:836-843.
  69. Epstein, W., and C. F. Fox. 1970. Mapping of a locus for unsaturated fatty acid biosynthesis in *Escherichia coli*. *J. Bacteriol.* 103:274-275.
  70. Eriksson-Grennberg, K. G. 1968. Resistance of *Escherichia coli* to penicillins. II. An improved mapping of the *ampA* gene. *Genet. Res.* 12:147-156.
  71. Falkow, S., H. Schneider, L. S. Baron, and S. B. Formal. 1963. Virulence of *Escherichia-Shigella* genetic hybrids for the guinea pig. *J. Bacteriol.* 86:1251-1258.
  72. Fangman, W. L., and A. Novick. 1966. Mutant bacteria showing efficient utilization of thymidine. *J. Bacteriol.* 91:2390-2391.
  73. Farkas, W., and C. Gilvarg. 1965. The reduction step in diaminopimelic acid biosynthesis. *J. Biol. Chem.* 240:4717-4722.
  74. Fiil, N., and J. D. Friesen. 1968. Isolation of "relaxed" mutants of *Escherichia coli*. *J. Bacteriol.* 95:729-731.
  75. Flaks, J. G., P. S. Leboy, E. A. Birge, and C. G. Kurland.

1966. Mutations and genetics concerned with the ribosome. Cold Spring Harbor Symp. Quant. Biol. 31:623-631.
76. Fox, C. F., J. R. Carter, and E. P. Kennedy. 1967. Genetic control of the membrane protein component of the lactose transport system of *Escherichia coli*. Proc. Nat. Acad. Sci. U.S.A. 57:698-705.
77. Fraenkel, D. G. 1967. Genetic mapping of mutations affecting phosphoglucose isomerase and fructose diphosphatase in *Escherichia coli*. J. Bacteriol. 93:1582-1587.
78. Fraenkel, D. G., and B. L. Horecker. 1965. Fructose-1,6-diphosphatase and acid hexose phosphatase of *Escherichia coli*. J. Bacteriol. 90:837-842.
79. Frédéricq, P., and M. Betz-Bareau. 1952. Recombinants génétiques de souches marquées par résistance aux colicines et aux bactériophages. Ann. Inst. Pasteur 83: 283-294.
80. Fulton, C. 1965. Continuous chromosome transfer in *Escherichia coli*. Genetics 52:55-74.
81. Gallucci, E., and A. Garen. 1966. Suppressor genes for nonsense mutations. II. The *Su-4* and *Su-5* suppressor genes of *Escherichia coli*. J. Mol. Biol. 15:193-200.
82. Ganesan, A. K., and B. Rotman. 1966. Transport systems for galactose and galactosides in *Escherichia coli*. I. Genetic determination and regulation of the methylgalactoside permease. J. Mol. Biol. 16:42-50.
83. Garen, A., S. Garen, and R. C. Wilhelm. 1965. Suppressor genes for nonsense mutations. I. The *Su-1*, *Su-2*, and *Su-3* genes of *Escherichia coli*. J. Mol. Biol. 14:167-178.
84. Gartner, T. K., and M. Riley. 1964. Genetic studies on tryptophanase mutants of *Escherichia coli* K12. Bacteriol. Proc., p. 18.
85. Gholson, R. K., G. J. Tritz, T. S. Matney, and A. J. Andreoli. 1969. Mode of nicotinamide adenine dinucleotide utilization by *Escherichia coli*. J. Bacteriol. 99:895-896.
86. Gibson, F., and J. Pittard. 1968. Pathways of biosynthesis of aromatic amino acids and vitamins and their control in microorganisms. Bacteriol. Rev. 32:465-492.
87. Glandsdorff, N., 1965. Topography of cotransducible arginine mutations in *Escherichia coli* K-12. Genetics 51:167-179.
88. Glandsdorff, N. 1967. Pseudoinversions in the chromosome of *Escherichia coli* K-12. Genetics 55:49-61.
89. Glanville, E. V., and M. Demerec. 1960. Threonine, isoleucine, and isoleucine-valine mutants of *Salmonella typhimurium*. Genetics 45:1359-1374.
90. Gorini, L., W. Gunderson, and M. Burger. 1961. Genetics of regulation of enzyme synthesis in the arginine biosynthetic pathway of *Escherichia coli*. Cold Spring Harbor Symp. Quant. Biol. 26:173-182.
91. Gratia, J. P. 1966. Studies on defective lysogeny due to chromosomal deletion in *Escherichia coli*. Biken J. 9: 77-87.
92. Gross, J., and M. Gross. 1969. Genetic analysis of an *E. coli* strain with a mutation affecting DNA polymerase. Nature 224:1166-1168.
93. Guest, J. R. 1969. Biochemical and genetic studies with nitrate reductase C-gene mutants of *Escherichia coli*. Mol. Gen. Genet. 105:285-297.
94. Hane, M. W., and T. H. Wood. 1969. *Escherichia coli* K-12 mutants resistant to nalidixic acid: genetic mapping and dominance studies. J. Bacteriol. 99:239-241.
95. Hatfield, D., M. Hofnung, and M. Schwartz. 1969. Genetic analysis of the maltose A region in *Escherichia coli*. J. Bacteriol. 98:559-567.
96. Hatfield, D., M. Hofnung, and M. Schwartz. 1969. Nonsense mutations in the maltose A region of the genetic map of *Escherichia coli*. J. Bacteriol. 100:1311-1315.
97. Helling, R. B. 1968. Selection of a mutant of *Escherichia coli* which has high mutation rates. J. Bacteriol. 96:975-980.
98. Henning, U., G. Dennert, R. Hertel, and W. S. Shipp. 1966. Translation of the structural genes of the *E. coli* pyruvate dehydrogenase complex. Cold Spring Harbor Symp. Quant. Biol. 31:227-234.
99. Henning, U., and C. Herz. 1964. Ein Strukturgen-Komplex für den Pyruvat-Dehydrogenase-Komplex von *Escherichia coli* K12. Z. Vererbungsl. 95:260-275.
100. Herbert, A. A., and J. R. Guest. 1968. Biochemical and genetic studies with lysine + methionine mutants of *Escherichia coli*: lipoic acid and  $\alpha$ -ketoglutarate dehydrogenase-less mutants. J. Gen. Microbiol. 53:363-381.
101. Herbert, A. A., and J. R. Guest. 1969. Studies with  $\alpha$ -ketoglutarate dehydrogenase mutants of *Escherichia coli*. Mol. Gen. Genet. 105:182-190.
102. Hiraga, S., K. Igarashi, and T. Yura. 1967. A deoxythymidine kinase-deficient mutant of *Escherichia coli*. Biochim. Biophys. Acta 145:41-51.
103. Hiraga, S., K. Ito, T. Matsuyama, H. Ozaki, and T. Yura. 1968. 5-Methyltryptophan-resistant mutations linked with the arginine G marker in *Escherichia coli*. J. Bacteriol. 96:1880-1881.
104. Hoffman, E., R. Wilhelm, W. Konigsberg, and J. Katze. 1970. A structural gene for seryl-tRNA synthetase in *Escherichia coli*. J. Mol. Biol. 46:619-625.
105. Holland, I. B., and E. J. Threlfall. 1969. Identification of closely linked loci controlling ultraviolet sensitivity and refractivity to colicin E2 in *Escherichia coli*. J. Bacteriol. 97:91-96.
106. Howard-Flanders, P., and R. P. Boyce. 1966. DNA repair and genetic recombination: studies on mutants of *Escherichia coli* defective in these processes. Radiat. Res. 6 (Suppl.):156-184.
107. Howard-Flanders, P., R. P. Boyce, and L. Theriot. 1966. Three loci in *Escherichia coli* K-12 that control the excision of pyrimidine dimers and certain other mutagen products from DNA. Genetics 53:1119-1136.
108. Howard-Flanders, P., E. Simson, and L. Theriot. 1964. A locus that controls filament formation and sensitivity to radiation in *Escherichia coli* K-12. Genetics 49:237-246.
109. Huang, M., and J. Pittard. 1967. Genetic analysis of mutant strains of *Escherichia coli* requiring *p*-aminobenzoic acid for growth. J. Bacteriol. 93:1938-1942.
110. Imae, Y. 1968. Mitomycin C-sensitive mutant of *Escherichia coli* K-12. J. Bacteriol. 95:1191-1192.
111. Ishibashi, M., Y. Sugino, and Y. Hirota. 1964. Chromosomal location of thymine and arginine genes in *Escherichia coli* and an F' incorporating them. J. Bacteriol. 87:554-561.
112. Itikawa, H., S. Baumberg, and H. J. Vogel. 1968. Enzymic basis for a genetic suppression: Accumulation and deacylation of *N*-acetylglutamic  $\gamma$ -semialdehyde in enterobacterial mutants. Biochim. Biophys. Acta 159:547-550.
113. Ito, J., and I. P. Crawford. 1965. Regulation of the enzymes of the tryptophan pathway in *Escherichia coli*. Genetics 52:1303-1316.
114. Ito, K., S. Hiraga, and T. Yura. 1969. Temperature-sensitive repression of the tryptophan operon in *Escherichia coli*. J. Bacteriol. 99:279-286.
115. Ito, K., S. Hiraga, and T. Yura. 1969. Tryptophanyl transfer RNA synthetase and expression of the tryptophan operon in the *trpS* mutants of *Escherichia coli*. Genetics 61:521-538.
116. Jacob, F., A. Ullman, and J. Monod. 1964. Le promoteur, élément génétique nécessaire à l'expression d'un opéron. C. R. Acad. Sci. Paris 258:3125-3128.
117. Jacob, F., and E. L. Wollman. 1958. Genetic and physical determinations of chromosomal segments in *Escherichia coli*. Symp. Soc. Exp. Biol. 12:75-92.
118. Jacob, F., and E. L. Wollman. 1961. Sexuality and the genetics of bacteria. Academic Press Inc., New York.
119. Jacoby, G. A., and L. Gorini. 1967. Genetics of control of the arginine pathway in *Escherichia coli* B and K. J. Mol. Biol. 24:41-50.
120. Jones-Mortimer, M. C. 1968. Positive control of sulphate reduction in *Escherichia coli*. Isolation, characterization and mapping of cysteineless mutants of *E. coli* K12. Biochem. J. 110:589-595.

121. Josephson, B. L., and D. G. Fraenkel. 1969. Transketolase mutants of *Escherichia coli*. *J. Bacteriol.* **100**:1289-1295.
122. Kawasaki, T., T. Nakata, and Y. Nose. 1968. Genetic mapping with a thiamine-requiring auxotroph of *Escherichia coli* K-12 defective in thiamine phosphate pyrophosphorylase. *J. Bacteriol.* **95**:1483-1485.
123. Kawasaki, T., and Y. Nose. 1969. Thiamine regulatory mutants in *Escherichia coli*. *J. Biochem. (Tokyo)* **65**:417-425.
124. Kay, W. W., and H. L. Kornberg. 1969. Genetic control of the uptake of C<sub>4</sub>-dicarboxylic acids by *Escherichia coli*. *Fed. Eur. Biochem. Soc. Letters* **3**:93-96.
125. Kessler, D. P., and E. Englesberg. 1969. Arabinose-leucine deletion mutants of *Escherichia coli* B/r. *J. Bacteriol.* **98**:1159-1169.
126. Kornberg, H. L., and J. Smith. 1969. Genetic control of hexose phosphate uptake by *Escherichia coli*. *Nature* **224**:1261-1262.
127. Kupor, S. R., and D. G. Fraenkel. 1969. 6-Phosphogluconolactonase mutants of *Escherichia coli* and a maltose blue gene. *J. Bacteriol.* **100**:1296-1301.
128. Lederberg, S. 1966. Genetics of host-controlled restriction and modification of deoxyribonucleic acid in *Escherichia coli*. *J. Bacteriol.* **91**:1029-1036.
129. Lee, N., and E. Englesberg. 1962. Dual effects of structural genes in *Escherichia coli*. *Proc. Nat. Acad. Sci. U.S.A.* **48**:335-348.
130. Lennox, E. S. 1955. Transduction of linked genetic characters of the host by bacteriophage P1. *Virology* **1**:190-206.
131. Lomax, M. S., and G. R. Greenberg. 1968. Characteristics of the *deo* operon: role in thymine utilization and sensitivity to deoxyribonucleosides. *J. Bacteriol.* **96**:501-514.
132. Maas, W. K. 1965. Genetic defects affecting an arginine permease and repression of arginine synthesis in *Escherichia coli*. *Fed. Proc.* **24**:1239-1242.
133. Maas, W. K., R. Maas, J. M. Wiame, and N. Glansdorff. 1964. Studies on the mechanism of repression of arginine biosynthesis in *Escherichia coli*. I. Dominance of repressibility in zygotes. *J. Mol. Biol.* **8**:359-364.
134. Maccacaro, G. A., and W. Hayes. 1961. Pairing interaction as a basis for negative interference. *Genet. Res.* **2**:406-413.
135. Marcus, M., and Y. S. Halpern. 1967. Genetic analysis of glutamate transport and glutamate decarboxylase in *Escherichia coli*. *J. Bacteriol.* **93**:1409-1415.
136. Marcus, M., and Y. S. Halpern. 1969. Genetic analysis of the glutamate permease in *Escherichia coli* K-12. *J. Bacteriol.* **97**:1118-1128.
137. Marcus, M., and Y. S. Halpern. 1969. Genetic and physiological analysis of glutamate decarboxylase in *Escherichia coli* K-12. *J. Bacteriol.* **97**:1509-1510.
138. Marcus, M., and Y. S. Halpern. 1969. Mapping of the aspartase gene in *Escherichia coli* K-12. *Israel J. Med. Sci.* **5**:413-415.
139. Markovitz, A., M. M. Lieberman, and N. Rosenbaum. 1967. Derepression of phosphomannose isomerase by regulator gene mutations involved in capsular polysaccharide synthesis in *Escherichia coli* K-12. *J. Bacteriol.* **94**:1497-1501.
140. Markovitz, A., N. Rosenbaum, and B. Baker. 1968. P1-mediated transduction of a gene that controls radiation sensitivity and capsular polysaccharide synthesis from *Shigella dysenteriae* to *Escherichia coli*. *J. Bacteriol.* **96**:221-226.
141. Markovitz, A., R. J. Sydiskis, and M. M. Lieberman. 1967. Genetic and biochemical studies on mannose-negative mutants that are deficient in phosphomannose isomerase in *Escherichia coli* K-12. *J. Bacteriol.* **94**:1492-1496.
142. Matsushiro, A. 1965. On the transcription of the tryptophan operon in *Escherichia coli*. I. The tryptophan operator. *J. Mol. Biol.* **11**:54-63.
143. Matsuzawa, H., M. Matsuhashi, A. Oka, and Y. Sugino. 1969. Genetic and biochemical studies on cell wall peptidoglycan synthesis in *Escherichia coli* K-12. *Biochem. Biophys. Res. Commun.* **36**:682-689.
144. McFall, E. 1967. Mapping of the D-serine deaminase region in *Escherichia coli* K-12. *Genetics* **55**:91-99.
145. Miller, J. H., K. Ippen, J. Scaife, and J. Beckwith. 1968. The promoter-operator region of the *lac* operon of *Escherichia coli*. *J. Mol. Biol.* **38**:413-420.
146. Morrissey, A. T. E., and D. G. Fraenkel. 1969. Chromosomal location of a gene for fructose 6-phosphate kinase in *Escherichia coli*. *J. Bacteriol.* **100**:1108-1109.
147. Nagel de Zwaig, R., and S. E. Luria. 1967. Colicin-tolerant mutants of *Escherichia coli*. *Bacteriol. Proc.*, p. 155.
148. Nagel de Zwaig R., and S. E. Luria. 1967. Genetics and physiology of colicin-tolerant mutants of *Escherichia coli*. *J. Bacteriol.* **94**:1112-1123.
149. Nakamura, H. 1968. Genetic determination of resistance to acriflavine, phenethyl alcohol, and sodium dodecyl sulfate in *Escherichia coli*. *J. Bacteriol.* **96**:987-996.
150. Neidhardt, F. C. 1966. Roles of amino acid activating enzymes in cellular physiology. *Bacteriol. Rev.* **30**:701-719.
151. Nijkamp, H. J. J., and P. G. de Haan. 1967. Genetic and biochemical studies of the guanosine 5'-monophosphate pathway in *Escherichia coli*. *Biochim. Biophys. Acta* **145**:31-40.
152. Nijkamp, H. J. J., and A. A. G. Oskamp. 1968. Regulation of the biosynthesis of guanosine 5'-monophosphate: evidence for one operon. *J. Mol. Biol.* **35**:103-109.
153. Nomura, M., and C. Witten. 1967. Interaction of colicins with bacterial cells. III. Colicin-tolerant mutations in *Escherichia coli*. *J. Bacteriol.* **94**:1093-1111.
154. Normark, S. 1969. Mutation in *Escherichia coli* K-12 mediating spherulike envelopes and changed tolerance to ultraviolet irradiation and some antibiotics. *J. Bacteriol.* **98**:1274-1277.
155. Normark, S., H. G. Boman, and E. Mattsson. 1969. Mutant of *Escherichia coli* with anomalous cell division and ability to decrease episomally and chromosomally mediated resistance to ampicillin and several other antibiotics. *J. Bacteriol.* **97**:1334-1342.
156. Novotny, C. P., and E. Englesberg. 1966. The L-arabinose permease system in *Escherichia coli* B/r. *Biochim. Biophys. Acta* **117**:217-230.
157. Ogawa, H., K. Shimada, and J. Tomizawa. 1968. Studies on radiation-sensitive mutants of *E. coli*. I. Mutants defective in the repair synthesis. *Mol. Gen. Genet.* **101**:227-244.
158. Oishi, M. 1969. An ATP-dependent deoxyribonuclease from *Escherichia coli* with a possible role in genetic recombination. *Proc. Nat. Acad. Sci. U.S.A.* **64**:1292-1299.
159. Okada, T. 1966. Mutational site of the gene controlling quantitative thymine requirement in *Escherichia coli* K-12. *Genetics* **54**:1329-1336.
160. Ørskov, F., and I. Ørskov. 1962. Behavior of *Escherichia coli* antigens in sexual recombination. *Acta Pathol. Microbiol. Scand.* **55**:99-109.
161. Otsuji, N. 1968. Properties of mitomycin C-sensitive mutants of *Escherichia coli* K-12. *J. Bacteriol.* **95**:540-545.
162. Overath, P., G. Pauli, and H. U. Schairer. 1969. Fatty acid degradation in *Escherichia coli*, an inducible acyl-CoA synthetase, the mapping of *old*-mutations, and the isolation of regulatory mutants. *Eur. J. Biochem.* **7**:559-574.
163. Pascal, M., J. Puig, and M. Lepelletier. 1969. Étude génétique d'une mutation affectant l'activité L-lactate-déshydrogénase chez *Escherichia coli* K12. *C. R. Acad. Sci. Paris* **268**:737-739.
164. Patte, J. C., and G. N. Cohen. 1965. Isolement et propriétés d'un mutant d'*Escherichia coli* despourvu d'aspartokinase sensible à la lysine. *Biochim. Biophys. Acta* **99**:561-563.
165. Patte, J., G. LeBras, and G. N. Cohen. 1967. Regulation by methionine of the synthesis of a third aspartokinase and of a second homoserine dehydrogenase in *Escherichia coli* K12. *Biochim. Biophys. Acta* **136**:245-257.
166. Patte, J., P. Truffa-Bachi, and G. N. Cohen. 1966. The

- threonine-sensitive homoserine dehydrogenase and aspartokinase activities of *Escherichia coli*. I. Evidence that the two activities are carried by a single protein. *Biochim. Biophys. Acta* 128:426-439.
167. Peyru, G., and D. G. Fraenkel. 1968. Genetic mapping of loci for glucose-6-phosphate dehydrogenase, gluconate-6-phosphate dehydrogenase, and gluconate-6-phosphate dehydrase in *Escherichia coli*. *J. Bacteriol.* 95:1272-1278.
  168. Pittard, J. 1965. Effect of integrated sex factor on transduction of chromosomal genes in *Escherichia coli*. *J. Bacteriol.* 89:680-686.
  169. Pittard, J., J. S. Loutit, and E. A. Adelberg. 1963. Gene transfer by F' strains of *Escherichia coli* K12. I. Delay in initiation of chromosome transfer. *J. Bacteriol.* 85:1394-1401.
  170. Pittard, J., and B. J. Wallace. 1966. Distribution and function of genes concerned with aromatic biosynthesis in *Escherichia coli*. *J. Bacteriol.* 91:1494-1508.
  171. Pittard, J., and B. J. Wallace. 1966. Gene controlling the uptake of shikimic acid by *Escherichia coli*. *J. Bacteriol.* 92:1070-1075.
  172. Power, J. 1967. The L-rhamnose genetic system in *Escherichia coli* K-12. *Genetics* 55:557-568.
  173. Prestidge, L. S., and A. B. Pardee. 1965. A second permease for methyl-thio- $\beta$ -D-galactoside in *Escherichia coli*. *Biochim. Biophys. Acta* 100:591-593.
  174. Puig, J., and E. Azoulay. 1967. Étude génétique et biochimique des mutants résistants au ClO<sub>2</sub>-(gènes *chlA*, *chlB*, *chlC*). *C. R. Acad. Sci. Paris* 264:1916-1918.
  175. Puig, J., E. Azoulay, J. Gendre, and E. Richard. 1969. Étude génétique des mutants de la région *chlA* chez l'*Escherichia coli*. *C. R. Acad. Sci. Paris* 268:183-184.
  176. Ramakrishnan, T., and E. A. Adelberg. 1965. Regulatory mechanisms in the biosynthesis of isoleucine and valine. II. Identification of two operator genes. *J. Bacteriol.* 89:654-660.
  177. Ramakrishnan, T., and E. A. Adelberg. 1965. Regulatory mechanisms in the biosynthesis of isoleucine and valine. III. Map order of the structural genes and operator genes. *J. Bacteriol.* 89:661-664.
  178. Reeve, E. C. R., and P. Doherty. 1968. Linkage relationships of two genes causing partial resistance to chloramphenicol in *Escherichia coli*. *J. Bacteriol.* 96:1450-1451.
  179. Reeves, P. 1966. Mutants resistant to colicin CA42-E<sub>2</sub>: cross resistance and genetic mapping of a special class of mutants. *Aust. J. Exp. Biol. Med. Sci.* 44:301-316.
  180. Reiner, A. M. 1969. Isolation and mapping of polynucleotide phosphorylase mutants of *Escherichia coli*. *J. Bacteriol.* 97:1431-1436.
  181. Reiner, A. M. 1969. Genetic locus for ribonuclease I in *Escherichia coli*. *J. Bacteriol.* 97:1522-1523.
  182. Rolfe, B., and M. A. Eisenberg. 1968. Genetic and biochemical analysis of the biotin loci of *Escherichia coli* K-12. *J. Bacteriol.* 96:515-524.
  183. Rosset, R., and L. Gorini. 1969. A ribosomal ambiguity mutation. *J. Mol. Biol.* 39:95-112.
  184. Rothman, J. L. 1965. Transduction studies on the relation between prophage and host chromosome. *J. Mol. Biol.* 12:892-912.
  185. Rotman, B., A. K. Ganesan, and R. Guzman. 1968. Transport systems for galactose and galactosides in *Escherichia coli*. II. Substrate and inducer specificities. *J. Mol. Biol.* 36:247-260.
  186. Rowbury, R. J., and D. D. Woods. 1964. O-succinyl-homoserine as an intermediate in the synthesis of cystathionine by *Escherichia coli*. *J. Gen. Microbiol.* 36:341-358.
  187. Ruiz-Herrera, J., M. K. Showe, and J. A. DeMoss. 1969. Nitrate reductase complex of *Escherichia coli* K-12: isolation and characterization of mutants unable to reduce nitrate. *J. Bacteriol.* 97:1291-1297.
  188. Saedler, H., A. Gullon, L. Fiethen, and P. Starlinger. 1968. Negative control of the galactose operon in *E. coli*. *Mol. Gen. Genet.* 102:79-88.
  189. Sanderson, K. E. 1970. Current linkage map of *Salmonella typhimurium*. *Bacteriol. Rev.* 34:176-193.
  190. Sásárman, A., M. Surdeanu, and T. Horodniceanu. 1968. Locus determining the synthesis of  $\delta$ -aminolevulinic acid in *Escherichia coli* K-12. *J. Bacteriol.* 96:1822-1884.
  191. Sásárman, A., M. Surdeanu, G. Szégli, T. Horodniceanu, V. Greceanu, and A. Dumitrescu. 1968. Hemineficient mutants of *Escherichia coli* K-12. *J. Bacteriol.* 96:570-572.
  192. Schaefer, S., and W. K. Maas. 1967. Inducible system for the utilization of  $\beta$ -glucosides in *Escherichia coli*. *J. Bacteriol.* 93:264-272.
  193. Schleif, R. 1969. Isolation and characterization of a streptolydigin resistant RNA polymerase. *Nature* 223:1068-1069.
  194. Schlesinger, S., and E. W. Nester. 1969. Mutants of *Escherichia coli* with an altered tyrosyl-transfer ribonucleic acid synthetase. *J. Bacteriol.* 100:167-175.
  195. Schmitt, R. 1968. Analysis of melibiose mutants deficient in  $\alpha$ -galactosidase and thiomethylgalactoside permease II in *Escherichia coli* K-12. *J. Bacteriol.* 96:462-471.
  196. Schwartz, M. 1966. Location of the maltose A and B loci on the genetic map of *Escherichia coli*. *J. Bacteriol.* 92:1083-1089.
  197. Schwartz, M. 1967. Sur l'existence chez *Escherichia coli* K12 d'une regulation commune à la biosynthèse des recepteurs du bactériophage  $\lambda$  et au métabolisme du maltose. *Ann. Inst. Pasteur* 113:685-704.
  198. Shapiro, J. A. 1966. Chromosomal location of the gene determining uridine diphosphoglucose formation in *Escherichia coli* K-12. *J. Bacteriol.* 92:518-520.
  199. Shapiro, J. A., and S. L. Adhya. 1969. The galactose operon of *E. coli* K-12. II. A deletion analysis of operon structure and polarity. *Genetics* 62:249-264.
  200. Sheppard, D., and E. Englesberg. 1966. Positive control in the L-arabinose gene-enzyme complex of *Escherichia coli* B/r as exhibited with stable merodiploids. *Cold Spring Harbor Symp. Quant. Biol.* 31:345-347.
  201. Sheppard, D. E., and E. Englesberg. 1967. Further evidence for positive control of the L-arabinose system by gene *araC*. *J. Mol. Biol.* 25:443-454.
  202. Siegel, E. C., and V. Bryson. 1967. Mutator gene of *Escherichia coli* B. *J. Bacteriol.* 94:38-47.
  203. Sigal, N., and J. M. Puig. 1968. Étude génétique des mutants du système de la glycogénèse chez *Escherichia coli* K 12. *C. R. Acad. Sci. Paris* 267:1223-1226.
  204. Signer, E. R. 1966. Interaction of prophages at the *attB* site with the chromosome of *Escherichia coli*. *J. Mol. Biol.* 15:243-255.
  205. Signer, E. R., J. R. Beckwith, and S. Brenner. 1965. Mapping of suppressor loci in *Escherichia coli*. *J. Mol. Biol.* 14:153-166.
  206. Skaar, P. D. 1956. A binary mutability system in *Escherichia coli*. *Proc. Nat. Acad. Sci. U.S.A.* 42:245-249.
  207. Smith, D. A., and J. D. Childs. 1966. Methionine genes and enzymes of *Salmonella typhimurium*. *Heredity* 21:265-286.
  208. Soll, L., and P. Berg. 1969. Recessive lethals: a new class of nonsense suppressors in *Escherichia coli*. *Proc. Nat. Acad. Sci. U.S.A.* 63:392-399.
  209. Somerville, R. L., and C. Yanofsky. 1965. Studies on the regulation of tryptophan biosynthesis in *Escherichia coli*. *J. Mol. Biol.* 11:747-759.
  210. Sparling, P. F. 1970. Kasugamycin resistance: 30S ribosomal mutation with an unusual location on the *Escherichia coli* chromosome. *Science* 167:56-58.
  211. Stouthamer, A. H., P. G. de Haan, and H. J. J. Nijkamp. 1965. Mapping of purine markers in *Escherichia coli* K12. *Genet. Res.* 6:442-453.
  212. Stretton, A. O. W., S. Kaplan, and S. Brenner. 1966. Nonsense codons. *Cold Spring Harbor Symp. Quant. Biol.* 31:173-179.

213. Sugino, Y. 1966. Mutants of *Escherichia coli* sensitive to methylene blue and acridines. *Genet. Res.* 7:1-11.
214. Taylor, A. L., and M. S. Thoman. 1964. The genetic map of *Escherichia coli* K-12. *Genetics* 50:659-677.
215. Taylor, A. L., and C. D. Trotter. 1967. Revised linkage map of *Escherichia coli*. *Bacteriol. Rev.* 31:332-353.
216. Tingle, M. A., and F. C. Neidhardt. 1969. Mapping of a structural gene for valyl-transfer ribonucleic acid synthetase in *Escherichia coli* by transduction. *J. Bacteriol.* 98:837-839.
217. Tritz, G. J., T. S. Matney, J. L. R. Chandler, and R. K. Gholson. 1970. Identification of the *purI* locus in *Escherichia coli* K-12. *J. Bacteriol.* 102:881-883.
218. Tritz, G. J., T. S. Matney, and R. K. Gholson. 1970. Mapping of the *nadB* locus adjacent to a previously undescribed purine locus in *Escherichia coli*. *J. Bacteriol.* 102:377-381.
219. Tyler, B., R. Wishnow, W. F. Loomis, Jr., and B. Magasanik. 1969. Catabolite repression gene of *Escherichia coli*. *J. Bacteriol.* 100:809-816.
220. Umbarger, H. E., M. A. Umbarger, and P. M. L. Siu. 1963. Biosynthesis of serine in *Escherichia coli* and *Salmonella typhimurium*. *J. Bacteriol.* 85:1431-1439.
221. Van de Putte, P., J. Van Dillewijn, and A. Rörsch. 1964. The selection of mutants of *Escherichia coli* with impaired cell division at elevated temperature. *Mutation Res.* 1:121-128.
222. Van de Putte, P., C. A. Van Sluis, J. Van Dillewijn, and A. Rörsch. 1965. The location of genes controlling radiation sensitivity in *Escherichia coli*. *Mutat. Res.* 2:97-110.
223. Vanderwinkel, E., and M. De Vlieghe. 1968. Physiologie et génétique de l'isocitritase et des malate synthases chez *Escherichia coli*. *Eur. J. Biochem.* 5:81-90.
224. Venables, W. A., and J. R. Guest. 1968. Transduction of nitrate reductase loci of *Escherichia coli* by phages P1 and  $\lambda$ . *Mol. Gen. Genet.* 103:127-140.
225. Vise, A. B., and J. Lascelles. 1967. Some properties of a mutant strain of *Escherichia coli* which requires lysine and methionine or lipoic acid for growth. *J. Gen. Microbiol.* 48:87-93.
226. Vogel, H. J., D. F. Bacon, and A. Baich. 1963. Induction of acetylornithine  $\delta$ -transaminase during pathway-wide repression, p. 293-300. In H. J. Vogel, V. Pryson, and J. O. Lampen (ed.), *Informational macromolecules*. Academic Press Inc., New York.
227. Walker, J. R. 1969. *Escherichia coli* *ras* locus: its involvement in radiation repair. *J. Bacteriol.* 99:713-719.
228. Wallace, B. J., and J. Pittard. 1967. Genetic and biochemical analysis of the isoenzymes concerned in the first reaction of aromatic biosynthesis in *Escherichia coli*. *J. Bacteriol.* 93:237-244.
229. Wallace, B. J., and J. Pittard. 1969. Regulator gene controlling enzymes concerned in tyrosine biosynthesis in *Escherichia coli*. *J. Bacteriol.* 97:1234-1241.
230. Wang, C. C., and A. Newton. 1969. Iron transport in *Escherichia coli*: relationship between chromium sensitivity and high iron requirement in mutants of *Escherichia coli*. *J. Bacteriol.* 98:1135-1141.
231. Wang, R. J., H. G. Morse, and M. L. Morse. 1969. Carbohydrate accumulation and metabolism in *Escherichia coli*: the close linkage and chromosomal location of *ctr* mutations. *J. Bacteriol.* 98:605-610.
232. Weisblum, B., and J. Davies. 1968. Antibiotic inhibitors of the bacterial ribosome. *Bacteriol. Rev.* 32:493-528.
233. Willetts, N. S., A. J. Clark, and B. Low. 1969. Genetic location of certain mutations conferring recombination deficiency in *Escherichia coli*. *J. Bacteriol.* 97:244-249.
234. Willetts, N. S., and D. W. Mount. 1969. Genetic analysis of recombination-deficient mutants of *Escherichia coli* K-12 carrying *rec* mutations cotransducible with *thyA*. *J. Bacteriol.* 100:923-934.
235. Wood, W. B. 1966. Host specificity of DNA produced by *Escherichia coli*: bacterial mutations affecting the restriction and modification of DNA. *J. Mol. Biol.* 16:118-133.
236. Wu, T. T. 1969. Locus determining P1 phage restriction in *Escherichia coli*. *J. Bacteriol.* 98:314.
237. Wu, T. T., T. M. Chused, and E. C. C. Lin. 1967. A dehydrogenase enabling mutants of *Escherichia coli* to grow on 1,2-propanediol. *Bacteriol. Proc.*, p. 52.
238. Yanofsky, C., and J. Ito. 1966. Nonsense codons and polarity in the tryptophan operon. *J. Mol. Biol.* 21:313-334.
239. Yanofsky, C., and E. S. Lennox. 1959. Transduction and recombination study of linkage relationships among the genes controlling tryptophan synthesis in *Escherichia coli*. *Virology* 8:425-447.
240. Yu, M. T., A. R. Kaney, and K. C. Atwood. 1965. Genetic mapping of fructose-1, 6-diphosphatase in *Escherichia coli*. *J. Bacteriol.* 90:1150-1152.
241. Yura, T., and K. Igarashi. 1968. RNA polymerase mutants of *Escherichia coli*. I. Mutants resistant to streptovaricin. *Proc. Nat. Acad. Sci. U.S.A.* 61:1313-1319.
242. Yura, T., and C. Wada. 1968. Phenethyl alcohol resistance in *Escherichia coli*. I. Resistance of strain C600 and its relation to azide resistance. *Genetics* 59:177-190.
243. Zabin, I. 1963. Proteins of the lactose system. Cold Spring Harbor Symp. Quant. Biol. 28:431-435.
244. Zamenhof, P. J. 1966. A genetic locus responsible for generalized high mutability in *Escherichia coli*. *Proc. Nat. Acad. Sci. U.S.A.* 56:845-852.
245. Zamenhof, P. J. 1969. On the identity of two bacterial mutator genes: effect of antimutagens. *Mutat. Res.* 7:463-465.