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Linkage Map of *Salmonella typhimurium*, Edition IV

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INTRODUCTION

A linkage map showing 133 genes of *Salmonella typhimurium*, based on F-factor-mediated conjugation studies using five Hfr strains, was presented in 1965 (247). Many of the genes had previously been mapped by P22-mediated transduction by M. Demerec and his collaborators. Two revised editions of the linkage map have been presented subsequently (245, 246). In the period since the third edition was published, much new information has accumulated from many laboratories on genetic analysis of new genes and greater accuracy of map location of those formerly known, as well as further data on the functions of these genes. It is the purpose of this report to summarize the current genetic data and to present an up-to-date linkage map.

The results of many investigators, who have generously made available unpublished as well as published data, are incorporated into this map. In addition to this map, a brief survey of some of the methods of genetic analysis currently available in *Salmonella* is presented.

An accompanying paper describes F^+, Hfr, and F-prime strains of *S. typhimurium* and *S. abony* (249). Current linkage maps of *Escherichia coli* K-12 have been presented in the past (289, 291, 292), and an accompanying paper (290) presents an updated version of this map.

METHODS OF GENETIC ANALYSIS IN SALMONELLA

In the early 1940's, interest in the bacteria and in viruses as models for genetic systems arose through the work with bacteriophages by Delbrück and collaborators (reviewed in reference 52), and with bacteria by many investigators, including Demerec (57, 80, 81). The discovery of four means of genetic exchange, i.e., transformation (14), conjugation (167), phage recombination (78), and transduction (328) provided the spark which ignited a period of great activity in genetic analysis in these microorganisms, a period which is not yet over. In *S. typhimurium*, much of the genetic analysis in the 1950's and early 1960's was done in the laboratory of M. Demerec, and practically all the genetic mapping was based on the use of the transducing phage P22 (57). Later, a system of F-mediated conjugation was established in *S. typhimurium* (327) and *S. abony* (176), and subsequently more Hfr strains isolated (247). Up to now, the major advances in genetic analysis of *S. typhimurium* have been based on these two methods. However, at this

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time many new methods, as well as modifications of the old methods, are available. The following section is a brief review of these materials and methods of genetic analysis, as they apply to *S. typhimurium*.

Bacteriophage-Mediated Transduction

Generalized transduction. (i) *Phage P22 and related phages (167b).* Zinder and Lederberg (328) first demonstrated generalized transduction using phage P22 with *S. typhimurium*. Methods have been described for complete transduction with P22 (125) and for complementation analysis by abortive transduction (123). The advantages of P22 are its ease of handling and its relatively high frequency of transduction; in addition, a number of mutants of the phage are available which give infrequent lysogenization (P22L4, 270; P22*cly*, 132) and hence facilitate isolation of nonlysogenic transductants, or which display altered frequency of transduction (256a). Phage P22 has some disadvantages. Firstly, it carries a relatively small transducing fragment. The transducing and plaque-forming phages carry deoxyribonucleic acid (DNA) molecules of approximately equal length, with 90% of the DNA of transducing particles being bacterial DNA and 10% being phage DNA (256). Since the molecular weight of P22 DNA is 2.6 to 2.7 $\times$ 10^7 (237), and that of *S. typhimurium* DNA is about 3 $\times$ 10^9 (174), a single transducing phage would be estimated to carry about 1% of the bacterial chromosome. Interrupted conjugation crosses indicate that the genes on the same transducing fragment may be separated by a minute of transfer time; the total map distance of 138 min therefore suggests that each transducing fragment is somewhat less than 1% of the total chromosome. This small size, relative to P1, the commonly used transducing phage of *E. coli*, has slowed the preparation of a linkage map based entirely on joint transduction studies. Secondly, a considerable proportion of *Salmonella* isolates from nature and some of the laboratory lines of *S. typhimurium* LT2 and LT7 are lysogenic for P22, and hence their use in transduction, though not impossible, is more difficult. Thirdly, rough mutants of *S. typhimurium* are usually resistant to P22 because loss of the O-specific side chains of the LPS prevents phage adsorption (302).

Several transducing phages related to P22 have been reported: MG40 (116), P22al (316), P22-11 (318), PSA68 (99), and L (35). Serological cross-neutralization tests indicate that P22, L, and PSA68 are closely related, while MG40 is not inactivated by antiserum against P22 or PSA68 (99). L lysogens are immune to L, PSA68, and MG40 phages, but not to P22; MG40 and PSA68 lysogens are lysed by P22, whereas P22 lysogens are immune to all four (99). In addition to the immunity common to temperate bacteriophages, P22 exhibits an additional mechanism for excluding superinfecting phage which is not immunity specific, called superinfection exclusion; this exclusion results in significant reduction in transduction by P22 on a P22 lysogen (92a, 167a, 285a). The relative frequencies of transductants for several genes by the four phages were similar (99).

(ii) *ES18.* This bacteriophage is heat-labile, and differs serologically and morphologically from phage P22 (162). Unlike P22 it can attack all classes of smooth as well as of nonsmooth (rough) derivatives of *S. typhimurium* LT2 lines which have been cured of prophage Fels 2; a host range mutant, ES18*h1*, overcomes the resistance conferred by lysogeny for Fels 2. ES18 is a general transducing phage; complete and abortive transduction occurs at rates less than P22 by one or two orders of magnitude. Transductional analysis of many rough mutants, not normally possible with P22 because of failure of P22 to absorb to these mutants, has been done with ES18 (163). From genetic data, it is apparent that the size of the transducing fragment is similar to that of P22.

(iii) *KB1.* This phage differs from P22 in plaque morphology, frequency of transductant formation, superinfection immunity properties, antiserum specificity, and heat sensitivity (43). Transduction of genes in different parts of the linkage map is obtained at higher frequencies per plaque-forming unit than with P22. KB1 belongs to the heat-sensitive B phage group of Boyd and Bidwell, whereas P22 and related phages are heat-resistant type A phages.

(iv) *P1.* This generalized transducing phage of *E. coli* K-12 will not adsorb to normal, smooth strains of *Salmonella*, but will adsorb to certain classes of rough mutants (218), such as *galE* and *rfaG* mutants in which the LPS is galactose deficient (222); the techniques for isolation of such mutants have been described (302). P1 has already been used in genetic analysis of *S. typhimurium* (26; Roth, *personal communication*, *Stocker, personal communication*). The P1 transducing fragment is larger than that of P22, so joint transduction is obtained between genes not jointly transduced by P22.

Specialized transduction. Phage P22 is known best as a generalized transducing phage but, in addition, P22 can undertake specialized

transduction of the genes near to its attachment site on the chromosome, close to *pro* (133, 241a, 270, 272). Since P22 has secondary attachment sites in addition to *ataA* (234), it is likely that it will also be possible for P22 to pick up chromosomal genes from these secondary sites. Specialized P22 transducing phages have been constructed which carry various regions of the *E. coli* chromosome, including *lac pro argF* (241a); this is possible because P22 can lysogenize by insertion into *E. coli* F' *pro lac* episomes carried by *S. typhimurium*. The episomal insertion site opens the possibility of constructing other P22 specialized phages after episome fusion by the method of Maas (233a). Smith (266) succeeded in analyzing the *hut* genes of *S. typhimurium* 15–59, a wild-type strain able to utilize histidine, by using the specialized transducing phage λ. The *hut* genes were transferred to a λ-sensitive *E. coli* K-12 by conjugation with a *S. typhimurium* Hfr strain; since the *hut* genes are located close to *gal*, they were frequently carried in λ-transducing particles.

Genetic mapping by abortive transduction. Enomoto (98) devised a method for mapping *mot* and *fla* mutants of *S. typhimurium*. This was based on the frequency of abortive transductants formed when double mutants for flagellar genes were transduced with P22 phage grown on wild-type cells; only if the wild-type alleles of the two mutant genes are carried on the same transducing fragment can an abortive motile transductant be formed. Such transductants of nonmotile, flagellar mutants are readily observed and enumerated as "trails" (motile abortive transductants) on a semisolid medium. However, the method may be difficult to extend to other genes for which abortive transductants are less readily observed.

Bacterial Conjugation

Conjugation, involving genetic exchange after cell-to-cell contact, and mediated by a sex factor, was first observed and exploited in *E. coli* (167, 290). A system of *col*-factor-mediated conjugation was established in *S. typhimurium* (278). Subsequently, the F-factor of *E. coli* K-12 was transferred into *S. typhimurium* (327) and *S. abony* (176), and Hfr strains were isolated and used in genetic studies (176, 247, 327), including interrupted mating analysis yielding a linkage map based on times of entry of Hfr chromosomal genes (247). An accompanying article describes the materials and methods available for conjugation analysis in *Salmonella* (249); at this time a considerable number of Hfr strains have been isolated, and F-prime factors representing a part of the linkage map of *Salmonella* are available.

Hybrid Analysis

Studies on hybrids of *E. coli* and *S. typhimurium* are reviewed by Baron et al. (20) and Middleton and Mojica-a (195). Transduction analysis of hybrids has been done to determine genetic homology (13, 82, 86, 87, 164, 326). Some of the hybrids isolated are unstable heterogenotes (20, 200a), whereas some are stable with the genetic material of the donor apparently integrated stably into the recipient chromosome (195, 200a). Hybrids permit the study of a number of interesting problems (195): the synaptic homology of genetic material from different organisms; the response of structural genes from one organism to regulatory genes from another; the properties of hybrid genes and hybrid gene products; and, in some situations, the determination of gene linkages (87, 164).

Localized Mutagenesis

The procedure of localized mutagenesis is used to introduce mutations into specific regions of the chromosome by means of transduction (131). The method had previously been shown to be effective in introducing new mutations after the treatment of transforming DNA in *Bacillus* (7, 153). A transducing phage preparation is mutagenized and then used to transduce a gene in the region of the bacterial chromosome in which mutations are desired. The transductants for the known gene are selected under conditions which will not discriminate against the desired mutations. Temperature-sensitive mutations in specific regions of the chromosome can be obtained, for example, by transducing an auxotrophic marker in the desired region with mutagenized phage and selecting for prototrophic transductants at low temperature, and then testing them for ability to grow at high temperatures. In this manner, temperature-sensitive mutations in either the transduced gene or in adjacent genes may be isolated (131).

Deletion Analysis

Deletions were used in *Drosophila* in classical proofs that genes are located at specific points on chromosomes, and by Benzer (30) in T4 phage as an aid to the analysis of fine structure of the rII genes. Mutants selected for

phage T1 resistance in *E. coli* may have deleted the *trp* genes (8), and this fact has been used for genetic analysis of the *trp* region (313). Several investigators have used deletions of *S. typhimurium* encompassing parts of the chromosome to locate the positions of genes (55, 201a, 202, 212, 295), or in analysis of fine structure of genes (84, 142, 171). Deletions joining two separate operons have been used to reveal the nature of genetic control (144, 168). Selective methods for obtaining deletions were first used in *Salmonella* in the *supX* system (201a). A deletion system has recently been developed which readily permits comparative genetic studies on a range of organisms. Chlorate is toxic under anaerobiosis to many bacteria, including *S. typhimurium*, due to an enzyme system which reduces nitrate as well as chlorate. Mutants that are resistant to chlorate and which are also defective in nitrate reductase activity can be readily isolated by a positive selection system (2), and many of those mutants are deletions. Mutations in at least four separate genetic regions are involved. Analysis of deletions leading to chlorate resistance in *S. typhimurium* (280–282, Ames and Alper, *personal communication*) and in *E. coli* (2) has located a considerable number of genes which are adjacent to the *chl* genes. These types of deletion analysis have the advantage that a means of genetic exchange and the prior isolation of many mutants are not necessary, so the system can be used to study a wide range of unrelated organisms.

Chromosome Replication Analysis

Replication of the chromosome of *E. coli* appeared to be a semiconservative process, beginning from a fixed point-of-origin and proceeding around the closed circular chromosome (51). This implies that in cell populations synchronized for cell division, replication of a given portion of the chromosome tends to occur at a specific time, depending on the distance of this region from the origin of replication. A linkage map of *Bacillus subtilis* was constructed, based on the greater concentration of transforming principle in replicated than in nonreplicated portions of the chromosome (315). A variety of procedures based on measures of transduction frequencies or of mutation frequencies have been used to determine the sequences of replication of the chromosome of *E. coli*. The same type of tests with *S. typhimurium* (214) indicated that replication proceeds in both directions from a fixed point-of-origin.

NOMENCLATURE

With few exceptions, the nomenclature recommended by Demerec et al. (83) is employed. Each gene is given a three-letter designation which may be followed by a capital letter. These gene symbols are listed in Table 1.

In most laboratories, each new mutant at the time of isolation is given a gene designation and an allele number (83). Therefore, a newly isolated mutant with a requirement for histidine would be given the gene designation *his*. The allele number must be chosen so as not to overlap with those already used. Therefore, the *Salmonella* Genetic Stock Centre, at the University of Calgary, has as one of its functions the assignment of blocks of allele numbers to investigators using *Salmonella*. It is recommended that gene designations allele numbers, and stock designations be cleared through the Stock Centre.

THE LINKAGE MAP

In the map presented in Fig. 1, the position of some of the genes and the basic time intervals between the genes are based on interrupted conjugation analysis presented earlier (249). Increased accuracy of gene location is based primarily on transduction analysis, and also, to some degree, on deletion analysis and other methods described earlier. Due to the importance of transduction analysis, the linkage map (Fig. 1) illustrates transduction fragments as arcs of the circle. All those genes covered by a set of arcs and indicated to be at a specific point of the map form a transduction linkage group. The genes at the extremities of the group, for example *argI* and *thrB*, at 0 min, may not themselves be jointly transduced, but they can be shown to be linked through other genes which are jointly transduced. Thus, a transduction linkage group may be made up of one, two, or more transduction fragments, depending on how well analyzed a specific region may be. The lengths of transduction fragments are illustrated by the solid arcs under the transduction linkage groups; the exact lengths shown for these transduction fragments are sometimes estimates based on partial data. Most transduction linkage groups displayed are those of phage P22, though a few are of phage ES18, which appears to be a similar size. Transduction linkage groups of phage P1 are denoted by a cross-hatched line.

Each transduction fragment is presented as though it occupies a point on the linkage map. A summary of interrupted conjugation experiments and joint transduction studies suggests

TABLE 1.

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
ah1		*Ah1A; Ah1C;* H1 activity controller		65	138, 231
ah2		*Ah2;* H2 activity controller		82	136, 137, 138
amtA		Resistance in 40 mM 3-amino-1,2,4-triazole in presence of histidine		52	A
ape		Deficient in enzyme for hydrolysis of *N*-acetyl-phenylalanine-β-naphthyl ester		19	AA
araB	Arabinose	Utilization	*ara*	3	36, 185
argA	Arginine	Acetyl ornithinase	*argE*	128	247, 298[c]
argB	Arginine	*N*-acetylglutamate synthetase	*argA*	95	247, 298[c]
argC	Arginine	*N*-acetyl-γ-glutamokinase	*argB*	128	247, 298[c]
argE	Arginine	Argininosuccinic acid synthetase	*argG*	102	26, 247, 298[c]
argF	Arginine	Argininosuccinase	*argH*	128	247, 298[c]
argG	Arginine	Acetyl ornithine-δ-transaminase	*argD*	108	161, 247, 298,[c] 308
argH	Arginine	*N*-acetylglutamic-γ-semialdehyde dehydrogenase	*argC*	128	247, 298[c]
argI	Arginine	*argK, argD;* Ornithine transcarbamylase	*argI*	0	287
aroA	Aromatic	3-Enolpyruvylshikimate 5-phosphate synthetase	*aroA*	45	110, 213
aroB	Aromatic	5-Dehydroquinate synthetase	*aroB*	108	110, 213, 308
aroC	Aromatic	5-Dehydroshikimate reductase	*aroE*	108	110, 213, 214
aroD	Aromatic	Chorismate synthetase	*aroC*	75	110, 213
aroE	Aromatic	5-Dehydroquinase	*aroD*	46	110, 213
aroF	Aromatic	Tyrosine-repressible $DAHP^+$ synthetase	*aroF*	87	B
aroG	Aromatic	Phenylalanine-repressible DAHP	*aroG*	33	280, B
aroH	Aromatic	Tryptophan-repressible DAHP synthetase	*aroH*	46	B
aroP	Aromatic	Permease	*aroP*	8	6
aroT		Ability to transport tryptophan, phenylalanine, tyrosine		52	295, FF
asd		Aspartic semialdehyde dehydrogenase	*asd*	112	BB
attA		*attP22* I; attachment site for prophage P22		10	17
attB		*attP27* I; attachment site for prophage P27		19	17
attB		*attP27* II; second attachment site for prophage P27		10	17
attC		*attP221;* attachment site for prophage P221		40	317
attD		$attP6_1$, *attP14;* attachment site for prophages $P6_1$ or P14 in group C *Salmonella*		85	17, 181
aziA	Azide	Resistant to 3 mM sodium azide on L-methionine	*azi*	3	67
aziB	Azide	Resistant to 3.4 mM sodium azide		125	C
bac		Osmotic-sensitive mutant		110	10, Q
bioA	Biotin	Requirement	*bioA*	33	46, 280
cdd		Cytidine deaminase		67	25
che		Chemotaxis	*che*	65	140
chlA	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase	*chlA*	33	280
chlB	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, and hydrogen lyase	*chlB*	122	59

[a] Abbreviations: AICAR, aminoimidazolecarboxamide ribotide; AIR, aminoimidazole ribotide; AMP, adenosine-5′-phosphate; ATP, adenosine triphosphate; C-AIR, phosphoribosyl(PR)-aminoimidazole carboxylate; CTP, cytosine diphosphate; DAHP, 3-deoxy-D-arabinoheptulosonic acid-7-phosphate; F-AICAR, PR-formamidoimidazole carboxamide; GAR, glycinamide ribotide; GDP, guanosine diphosphate; F-GAM, formylglycinamidine ribotide; F-GAR, formyl-glycinamide ribotide; GMP, guanosine-5′-phosphate; IMP, inosine-5′-phosphate; PRA, 5-phosphoribosylamine; PRPP, 5-phosphoribosylpyrophosphate; S-AICAR, succinyl-aminoimidazole-carboxamide ribotide; S-AMP, adenylosuccinate; TDP, thymidine diphosphate; UDP, uridine diphosphate; XMP, xanthosine-5′-phosphate.

[b] Numbers refer to the Literature Cited section; letters refer to personal communication from the following investigators. A, A. Wiater and T. Kłopotowski; B, A. DeLeo and D. B. Sprinson; C, Z. Ciésla and T. Kłopotowski; D, M. Alper and B. Ames; E, J. R. Stern; F, D. Berkowitz; G, J. Calvo; H, J. Kemper and P. Margolin; I, J. L. Ingraham; J, R. G. Martin; K, J. Gots; L, W. Ferron and A. Eisenstark; M, D. Berkowitz, J. Hushon, and B. H. Ames; N, K. Sanderson; O, D. Hulanicka and T. Kłopotowski; P, R. Eisenstark; Q, M. R. Ferrari, J. J. Figuero, and D. N. Antón; R, A. M. Palermo and D. N. Antón; S, W. Brill; T, D. Friedberg and J. Calvo; U, F. Casse; V, M. C. Pascal; W, E. Gollub and D. B. Sprinson; X, P. Jargiello and P. Hoffee; Y, W. Walczak, J. Wild, and T. Kłopotowski; Z, J. Brenchley; AA, C. Miller; BB, C. W. Shuster and J. Betts; CC, Krishnapillai and B. A. D. Stocker; DD, Ortega; EE, J. Roth and M. Grabnar; FF, L. Corwin; GG, Nygaard and J. Neuhard; HH, G. Levine and H. Whitfield; II, J. Roth; JJ, B. A. D. Stocker; KK, C. Colson

[c] These studies were done with the related organism, *E. coli*.

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
chlC	Chlorate	Resistance; affects nitrate reductase	*chlC*	52	U, V
chlD	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase		33	280
chlE	Chlorate	Resistance		33	281
chlF	Chlorate	Resistance		33	281
chlG	Chlorate	Resistance; affects nitrate reductase, tetrathionate reductase, chlorate reductase, and hydrogen lyase		82	282
chr	Chromium	*car*; chromium sensitivity		52	75
clb	Cellobiose	Utilization		104	278
cod		Cytosine deaminase		105	26
crp		Cyclic AMP receptor protein	*crp*	108	132
crr		Constitutive factor III for sugar transport		76	74
cya		*ple*; adenyl cyclase (cyclic-AMP requirement)	*cya*	122	132, 314
cysA	Cysteine	Sulfate-thiosulfate permease	*cysA*	76	89, 90, 200, 217, 227-229
cysBabc	Cysteine	Cysteine regulation	*cysB*	52	200
cysC	Cysteine	Adenylyl sulfate kinase	*cysC*	90	84, 142
cysD	Cysteine	Adenosine-5′-triphosphate sulfurylase		90	84, 142
cysEa, b	Cysteine	Cysteine synthetase; serine transacetylase	*cysE*	116	28, 159, 160, 200
cysG, I	Cysteine	Sulfite reductase (2nd step)	*cysG*	108, 90	84, 127, 142, 259
cysH	Cysteine	Adenylyl sulfate-3′-phosphate reductase	*cysH*	90	84, 142, 230
cysJ	Cysteine	Sulfite reductase (1st step)		90	84, 142, 230
cytR		Control of activity of *udp* and *cdd*		128	GG
dad		D-Amino acid dehydrogenase		50	BB
dapA		Dihydropicolinic acid synthetase	*dapA*	77	BB
dapB		Dihydropicolinic acid reductase	*dapB*	2	BB
dapC		Tetrahydrodipicolinic acid succinylase	*dapC*	7	BB
dapD		*N*-succinyl diaminopimelic acid transaminase	*dapD*	7	BB
dapF		Diaminopimelic acid racemase		7	BB
dct		Transport of dicarboxylic acids	*dct*	116	DD
deoA	Deoxyribose	*tpp*; thymidine phosphorylase	*tpp*	0	37, 128, 145, 239a
deoB	Deoxyribose	*drm*; phosphodeoxyribose mutase	*drm*	0	24, 128, 130, 145
deoC	Deoxyribose	*dra*; deoxyribose-5-phosphate aldolase, type II	*dra*	0	24, 128, 129, 145
deoD	Deoxyribose	*pup*; purine nucleoside phosphorylase	*pup*	0	128, 145
deoK	Deoxyribose	Deoxyribose kinase		37	128
deoP	Deoxyribose	Deoxyribose permease		37	128
deoR	Deoxyribose	Constitutive for enzymes of *deoA, B, C,* and *D*		22	36a, X
dhuA	D-Histidine	Utilization; increased activity of histidine-binding protein J		75	5, 158
divA	Division	*wrkA*; septum-initiation defect		128	67
divC	Division	*smoA*; cell envelope defect	*envA*	3	67
dml	D-Malate	Utilization		120	274, E
dnaC		DNA synthesis initiation and cell division uncoupling	*dnaC*	2	257, 273
dsd		D-Serine sensitivity	*dsdCA*	70	F
enb		*asc*; enterobactin (dihydroxybenzolyserine trimer)		20	106, 232
fabB		Fatty acid biosynthesis; cells lyse at high temperature in absence of oleic acid	*fabB*	75	131
fdhA		Formate hydrogenlyase complex; formate dehydrogenase		116	66, U
fdp		Fructose-1,6-diphosphatase	*fdp*	1	X
flaAI,AII,AIII, B,C,D,E,K, L,N,P,Q		No flagellin detected		65	98, 138, 139, 149, 286, 297, 309, 310, 311, 190
flaF	Flagella	No flagellin detected		47	98, 138, 139, 149, 286, 297, 309, 311

(*Continued*)

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
flaG	Flagella	Flagellin but no flagella			138, 286
flaM		No flagellin detected		65	311
flrB	Fluoroleucine resistance	Leucine or isoleucine regulation, or both		26	3, 53, G
fol		Dihydrofolate reductase (trimethoprim resistance)		3	32, H
galE	Galactose	*galD*;epimerase	*galE*	33	105, 181, 211, 258
galF	Galactose	*gal*; modifier of UDP-glucose pyrophosphorylase		65	206, 211, 212, 207
galK	Galactose	*galA*; galactokinase	*galK*	33	105, 181, 211, 258
galO	Galactose	Operator	*galO*	33	105, 258
galR	Galactose	Regulation	*galR*	95	244
galU	Galactose	UDP-glucose pyrophosphorphorylase	*galU*	52	CC, 207
gas		Gas production during carbohydrate fermentation		133	278
gdh		Glutamic dehydrogenase		129	DD
glpD		α-Glycerol phosphate dehydrogenase		112	DD
glpK	Glycerol	Glycerol kinase	*glpK*	120	278
glpT	Glycerol	L-α-Glycerol phosphate transport	*glpT*	74	239
glt	Glutamate	Requirement	*glt*	34	G
glyA	Glycine	Serine hydroxymethylase	*glyA*	78	240
gnd		Gluconate-6-phosphate dehydrogenase	*gnd*	65	202
guaA	Guanine	XMP aminase (XMP to GMP)	*guaB*	78	111, 210, 240
guaB	Guanine	IMP dehydrogenase (IMP to XMP)	*guaA*	78	111, 210, 240
gxu		Guanine-xanthine phosphoribosyl-transferase		10	112
H1	H antigen	Phase-one flagellar antigen (flagellin)	*hag*	65	100, 138, 177, 191 231, 310
H2	H antigen	Phase-two flagellar antigen (flagellin)	None	82	100, 138, 177, 191, 231, 310
hemA	Heme	Heme deficient; responds to δ-amino levulinate	*hemA*	50	253
hemB	Heme	Heme deficient	*hemB*	13	253
hisA	Histidine	Isomerase		65	124, 171, 183, 184, 187
hisB	Histidine	Imidazole glycerol phosphate dehydrase and histidinol phosphatase (bifunctional)		65	124, 171, 187, 284
hisC	Histidine	Imidazole acetol phosphate transaminase	*hisC*	65	124, 171, 187, 188, 189, 235
hisD	Histidine	Histidinol dehydrogenase	*hisD*	65	118, 319–323, 305, 235
hisE	Histidine	Phosphoribosyl-ATP-pyrophosphohydrolase	*hisE*	65	124, 171, 187
hisF	Histidine	Cyclase	*hisF*	65	124, 171, 187
hisG	Histidine	Phosphoribosyl-ATP-pyrophosphorylase	*hisG*	65	38, 39, 124, 171, 187, 242, 301
hisH	Histidine	Amido transferase	*hisH*	65	124, 171, 187
hisI	Histidine	Phosphoribosyl-AMP-hydrolase	*hisI*	65	124, 171, 187
hisJ	Histidine	Histidine-binding protein J for histidine permeation		75	5, 5a, 167a
hisO	Histidine	Operator	*hisO*	65	101, 102, 240, 299
hisP	Histidine	Permease		75	5, 6, 44, 240
hisR	Histidine	Regulation		122	44, 240, 260, 241
hisS	Histidine	Histidyl tRNA synthetase		78	44, 45, 79, 170, 192, 193, 240, 262, 306, 307
hisT	Histidine	Histidine derepression		75	44, 61, 240
hisU	Histidine	Histidine derepression		120	9, 44, 240
hisW	Histidine	Histidine derepression		73	9, 44, 240
hspLT	Host specificity	Restriction-modification system		12	31, 69–72, 219
hspS	Host specificity	Restriction-modification system	*hsm,hsr*	135	31, 69–72, 219
hutC	Histidine	Utilization; repressor		33	46, 194, 266–269
hutG	Histidine	Utilization; formininoglutamate hydrolase		33	46, 194, 266–269
hutH	Histidine	Utilization; histidase		33	46, 194, 266–269
hutI	Histidine	Utilization; imidazolonepropionate hydrolase		33	46, 194, 266–269

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
hutM	Histidine	Utilization; promoter for *hutIGC*		33	46, 194, 266–269
hutP	Histidine	Utilization; promoter for *hutUH*		33	46, 194, 266–269
hutQ	Histidine	Utilization; operator for *hutUH*		33	44, 194, 266–269
hutR	Histidine	Utilization; catabolite insensitivity of *hutUH*		33	44, 194, 266–269
hutU	Histidine	Utilization; urocanase		33	44, 194, 266–269
ilvA	Isoleucine	*ile*; threonine deaminase	*ilvA*	122	12, 50, 126, 164, 255, 325
ilvB	Isoleucine-valine	α-Acetohydroxyl acid synthetase (valine sensitivity)	*ilvB*		11, 47, 221
ilvC	Isoleucine-valine	*ilvA*; reductoisomerase	*ilvC*	122	12, 95, 107
ilvD	Isoleucine-valine	ilvB; dehydrase	*ilvD*	122	12, 107
ilvE	Isoleucine-valine	*ilvC*; transaminase	*ilvE*	122	12, 68, 107
ilvS	Isoleucine	Isoleucine tRNA synthetase		3	40
inlA	Inositol	Fermentation		135	147, 278
inlB	Inositol	Fermentation		82	282
leuA	Leucine	Condensing enzyme	*leuA*	3	48, 55, 185, 150
leuB	Leucine	Dehydrogenase	*leuB*	3	48, 49, 185
leuC,D	Leucine	Isomerase		3	48, 119, 154, 185
leuO	Leucine	Operator		3	48, 54, 117, 185
leuS	Leucine	Leucyl tRNA synthetase	*leuS*	26	4, 53, 196, 275
leuT		Leucine transport		52	FF
lip	Lipoic acid	Requirement		26	T
lys	Lysine	Requirement	*lysA*	95	93, 247
malB	Maltose	Utilization	*malB*	130	250
malQ		Amylomaltase	*malQ*	112	DD
meq	Menaquinone	Low-level resistance to aminoglycoside antibiotics; deficiency in menaquinone		122	254[a]
metA	Methionine	*metI*; homoserine O-transsuccinylase	*metA*	128	16, 64, 165, 264, 265
metB	Methionine	Cystathionine synthetase	*metB*	128	16, 122, 152, 264, 265
metC	Methionine	Cystathionase	*metC*	100	77, 201, 265
metE	Methionine	N^5-methyltetrahydropteroyl triglutamate-homocysteine methylase	*metE*	122	60, 264, 265, 254a
metF	Methionine	N^5N^{10}-methylenetetrahydrofolate reductase	*metF*	128	16, 264, 265
metG	Methionine	Methionyl-transfer RNA synthetase		67	16, 63, 120, 121, 264, 265
metH	Methionine	Cobalamine-dependent homocysteine-N^5-methyltetrahydrofolate transmethylase		128	16, 65, 293
metJ	Methionine	Methionine-analogue resistant	*metJ*	128	16, 62, 63, 166, 243, 254a
metK	Methionine	Methionine-analogue resistant	*metK*	95	62, 63, 165, 166, 198, 243, 254a
metP	Methionine	Methionine permease (α-methyl methionine resistant)		5	15
motA,B	Motility	Reduced motility	*mot*	65	96, 97, 98, 138, 311
mtlA	Mannitol	Mannitol dehydrogenase	*mtlD*	116	33, 251
mtlB	Mannitol	Mannitol permease		116	33, 251
mut	Mutator	Increases frequency of mutation		136	156, 157
nicA	Nicotinic acid	Requirement	*nadA*	33	280, D
nicB	Nicotinamide	Requirement		82	282
nml	*N*-methyl-lysine	*N*-methyl-lysine in flagellar protein		65	155, 277
oafA	O antigen factor	*ofi*; O-5; O-factor 5 (acetyl group)			181, 276
oafC	O antigen factor	Determines factor 1 in group *E. Salmonella*		22	181, 276
oafR	O antigen factor	Synthesis of O antigen 12_2		20	180, 181, 276
panA	Pantothenic acid	(?) to α-Ketoisovaleric acid	*pan*	7	85, 232
panB	Pantothenic acid	α-Ketoisovaleric acid to ketopantoic acid		7	85, 232
panC	Pantothenic acid	Pantoic acid to pantothenic acid		7	85, 232
pck		Phosphoenolpyruvate carboxykinase		22	56
pdxB		Pyridoxine requirement	*pdxB*	75	131
pfk		Phosphofructokinase	*pfk*	124	DD
pgi		Phosphoglucoisomerase	*pgi*	130	103, 271

[a] Note added in proof. The designation *meq* has been shown to be inappropriate for the mutants so named, because menaquinone is formed in some growth conditions, and the levels of cytochromes are altered (A. Sasarman, *personal communication*).

(*Continued*)

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
pheA	Phenylalanine	Prephenate dehydratase-chorismate mutase P	*pheA*	87	110, 213, W
pheR	Phenylalanine	Regulator gene for *pheA*		96	W
pig	Pigment	Brownish colonies		81	278
pil		*fim*; pili (fimbriae)	*pil*	23	278
pmi	Mannose	Phosphomannoisomerase	*man*	48	181, 276, 303
polA		DNA polymerase I	*polA*		HH
ppc		*asp*; phosphoenolpyruvic carboxylase (aspartate or glutamate requirement)	*ppc*	128	16, 175, 294
proA	Proline	Glutamate to glutamic-γ-semialdehyde	*proA*	10	143, 199, 270, 234, 272
proB	Proline	Glutamate to glutamic-γ-seminaldehyde	*proB*	10	143, 199, 234, 270, 272
proC	Proline	Glutamic-γ-semialdehyde to proline	*proC*	10	143, 199, 270, 272
ptdD		Deficient in dipeptidase		10	AA
ptdN		Deficient in enzyme for hydrolysis of amino acid B-naphthyl amides, di- and tripeptides, and amino acid amides.		40	AA
ptdP		Deficient in aminopeptidase P		122	AA
ptsH		*carB*; histidine protein of the phosphotransferase system	*ptsH*	76	74, 244a
ptsI		*carA*; enzyme I of the phosphotransferase system	*ptsI*	76	74, 244a
purA	Purine	S-AMP synthetase (IMP to S-AMP)	*purA*	135	115, 247
purB	Purine	Adenylosuccinate lyase (S-AMP to AMP)	*purB*	43	113
purC	Purine	S-AICAR synthetase (C-AIR to S-AICAR)	*purC*	78	240, K
purD	Purine	GAR synthetase (PRA to GAR)	*purD*	128	114, 300
purE	Purine	AIR carboxylase (AIR to C-AIR)	*purE*	19	247, K
purF	Purine	PRPP amidotransferase (PRPP to PRA)	*purF*	75	300
purG	Purine	F-GAR amidotransferase (F-GAR to F-GAM)	*purG*	78	240
purH	Purine	AICAR formyltransferase (AICAR to F-AICAR)	*purH*	128	114
purI	Purine	AIR synthetase (F-GAM to AIR)	*purI*	78	76, 240
purJ	Purine	IMP cyclohydrolase (F-AICAR to IMP)	*purJ*	128	114, 115
putA	Proline	Utilization; proline oxidase (proline utilization)		42	88, S, EE
putB	Proline	Utilization; pyrroline-5-carboxylate dehydrogenase (proline utilization)		42	88, S, EE
putC	Proline	Utilization; constitutive synthesis of *putA* and *putB* enzymes		42	88, S, EE
putR	Proline	Utilization; catabolite repression insensitivity of *putA* and *putB* enzymes		42	88, S, EE, 209
pyrA	Pyrimidine	*argD*; *ars*; *aus*; carbamate kinase (arginine - uracil requirement)	*pyrA*	3	1, 216, 287, 312
pyrB	Pyrimidine	Aspartate transcarbamylase	*pyrB*	0	29, 312
pyrC	Pyrimidine	Dihydroorotase	*pyrC*	42	29, 312
pyrD	Pyrimidine	Dihydroorotic acid dehydrogenase	*pyrD*	42	29, 312
pyrE	Pyrimidine	Orotidylic acid phosphorylase	*pyrE*	116	29, 163, 251, 312
pyrF	Pyrimidine	Orotidylic acid decarboxylase	*pyrF*	52	29, 248, 312
pyrG		Cytidine triphosphate synthetase		90	25, 208
pyrH		Uridine monophosphate kinase		7	141
pyrR	Pyrimidine	Regulation; derepression of aspartate transcarbamylase and dihydroorotate dehydrogenase		7	215
recA		Recombination deficient; degrades DNA	*recA*	92	94, 304, L
rec		Recombination deficient; reduced DNA degradation		91	94, L
rel	RNA relaxed	RC:regulation of RNA synthesis	*rel*	91	186, J
rfaE	Rough	Lipopolysaccharide core defect; proximal heptose deficient	*rfa*	106	172, 173, 181, 223-226, 276, 279
rfaF	Rough	Lipopolysaccharide core defect; distal heptose deficient		116	172, 173, 181, 223-226, 276
rfaG	Rough	Lipopolysaccharide core defect; glucose I transferase		116	172, 173, 181, 223-226, 276, 303

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
rfaH	Rough	Lipopolysaccharide core defect; galactose I transferase		122	172, 173, 181, 223–226, 276, 303
rfaJ	Rough	Lipopolysaccharide core defect; glucose II transferase		116	172, 173, 181, 223–226, 276, 303
rfaK	Rough	Lipopolysaccharide core defect; acetylglucosamine deficient		116	172, 173, 181, 223–226
rfaL	Rough	Lipopolysaccharide core defect; O-translocase		116	172, 173, 181, 223–226, 276, 302
rfaP	Rough	Lipopolysaccharide core defect; phosphorylation of heptose		115	91, 201, 181, 276
rfbA	Rough	TDP-glucose pyrophosphorylase		65	173, 181, 212, 276
rfbB	Rough	TDP-glucose oxidoreductase	*rfbB*	65	168, 178, 181, 212, 276
rfbD	Rough	TDP-rhamnose synthetase		65	173, 181, 212, 276
rfbF	Rough	CDP-glucose pyrophosphorylase		65	173, 181, 212, 276, 324
rfbG	Rough	CDP-glucose oxidoreductase		65	173, 181, 212, 276, 324
rfbH	Rough	CDP-abequose synthetase		65	173, 181, 212, 276, 324
rfbK	Rough	Phosphomannomutase (man-2)	*som*	65	173, 181, 212, 276, 324
rfbL	Rough	Phosphomannomutase B		65	173, 181, 212, 276, 324
rfbM	Rough	GDP-mannose pyrophosphorylase (man-3)		65	173, 181, 212, 276, 324
rfbT	Rough	O-translocase		66	173, 181, 212, 276, 302, 324
rfc	Rough	*rouC*; O-repeat unit not polymerized		51	173, 181, 212, 205, 276, 324
rfe	Rough	Defect in side chain synthesis in *S. minnesota* and *S. montivideo*		122	179
rft	Rough	"Transient" T1 forms		25	34, 181, 252
rha	Rhamnose	Utilization	*rha*	127	247
rif	Rifampin	Resistance to rifampin	*rif*	128	131
serA	Serine or glycine	3-Phosphoglycerate dehydrogenase	*serA*	95	296
serB	Serine or glycine	Phosphoserine phosphatase	*serB*	0	296
serC	Serine	Requirement for pyridoxine plus L-serine or glycine		73	C, Z
sid		Siderochrome (ferrichrome transport; albomycin resistance)		7	232
smoA		Smooth colony morphology in histidine constitutive mutants		3	C
smoB		Smooth colony morphology in histidine constitutive mutants		0	C
spcA	Spectinomycin	Resistance; ribosomal		108	308
spcB	Spectinomycin	Resistance; nonribosomal		108	308
strA	Streptomycin	High-level resistance; ribosomal	*strA*	108	214, 220, 288, 308
strB	Streptomycin	Low-level resistance plus auxotrophy; nonribosomal		78	240, 308
sufA		Frameshift suppressor affecting a proline tRNA		116	238, 239
sufB		Frameshift suppressor affecting a proline tRNA		73	238, 239
sufC		Frameshift suppressor		45	238, 239
sufD		Frameshift suppressor affecting a glycine tRNA		95	238, 239
sulE		Frameshift suppressor		128	238, 239
sufF		Recessive frameshift suppressor		19	238, 239
supC		Ochre suppressor	*supC*	51	M
supD		*supW*; suppressor	*supD*	65	101a
supK		*supT*; recessive UGA suppressor		95	236
supM		Ochre suppressor	*supM*	128	M
supQ		Suppressor of nonsense and deletion mutations of leuD		10	154

(*Continued*)

TABLE 1—*Continued*

Genetic symbol	Mnemonic	Former or alternate gene symbol, enzyme deficiency or other phenotype[a]	Homologous locus in *E. coli*	Map position (min)	References[b]
supR		Amber suppressor		122	197
supS		UGA suppressor		122	197
supX		*su-leu*-500; suppressor of *leu*-500		52	92, 104, 201a
supY		Amber suppressor	*supE*	33	M
supZ		Amber suppressor	*supF*	54	M
suc	Succinate + NH_4	Utilization		33	D
tdk		Thymidine kinase	*tdk*	52	26
thiA	Thiamine	Thiamine or pyrimidine moiety	*thiA,B*	128	131, K
thiB	Thiamine	Requirement		82	282
thiC	Thiamine	Thiazole type		17	K
thiD	Thiamine	Thiamine only		74	K
thiE	Thiamine	Thiazole type		85	K
thiF	Thiamine	Thiazole type		85	K
thiG	Thiamine	Thiamine or thiazole moiety	*thiA,B*	128	K
thrA	Threonine	Homoserine to homoserine phosphate (homoserine kinase)?	*thr*	0	107, 285
thrB	Threonine	Homoserine phosphate to?	*thr*	0	107, 285
thrC	Threonine	β-Aspartyl phosphate to homoserine	*thr*	0	107, 285
thrD	Threonine	Aspartic acid to β-aspartyl phosphate		0	107, 285
thy	Thymine	Requirement	*thyA*	95	93
tre	Trehalose	Utilization		58	283
trpA	Tryptophan	Anthranilate synthetase	*trpE*	52	21, 19, 42, 134, 182, 203, 204, 263
trpB	Tryptophan	Phosphoribosylpyrophosphate transferase	*trpD*	52	21, 19, 42, 134, 182, 203, 204, 263
trpC	Tryptophan	Tryptophan synthetase, component A	*trpA*	52	18, 22, 41
trpD	Tryptophan	Tryptophan synthetase, component B	*trpB*	52	18, 22, 41
trpE	Tryptophan	Phosphoribosyl anthranilic acid isomerase; indole glycerol phosphate synthetase	*trpC*	52	18, 22, 41
trpO	Tryptophan	Operator	*trpO*	52	19, 73
trpP	Tryptophan	Promoter		52	19, 22, 23
trpR	Tryptophan	Resistance to 5-methyltryptophan; derepression of tryptophan enzymes		0	19, 285
trzA		Resistance to 1,2,4-triazole		76	74, O
ttr		Tetrathionite reductase		46	58
tyrA	Tyrosine	Requirement	*tyrA*	87	213
tyrO	Tyrosine	*fpr*; operator for *aroF* and *tyrA*	*aroK*	87	108, W
tyrR	Tyrosine	Regulator gene for *aroF* and *tyrA*	*tyrR*	52	109
udk		Uridine kinase		67	26
udp		Uridine phosphorylase	*udp*	122	25, 208
upp		Uridine monophosphate pyrophosphorylase		78	25
uvrA		Resistance to ultraviolet light	*uvrA*	130	8a
uvrB		Ultraviolet light sensitivity	*uvrB*	33	280, D
uvr		Ultraviolet light sensitivity		125	P
vh2		*Vh2*; control of rate of phase variation		82	135, 138
viaA		*ViA*; Vi antigen		69	146, 148
viaB		*ViB*; Vi antigen (in *S. typhosa*)		137	146, 147
woi		Increased yield of *his01242* transductants by *hisW1824* recipients		125	R
xhc		Temperature-sensitive mutant linked to *purF* and *hisT*		75	131
xmi		Temperature-sensitive mutant linked to *argA* and *rif*		128	131
xyl	Xylose	Utilization	*xyl*	116	250, 251

that the length of a transduction fragment is slightly less than 1 min. It is possible, in principle, to estimate the length of each transduction linkage group in minutes, based on the number of transduction fragments and an estimate of the time required for transfer of a fragment, but this cannot be done accurately at this time. Therefore, the length of the gene interval between the closest genes in two adjacent linkage groups will be less than the interval between the points at which the two groups are shown on the map, but the actual distance is not usually clear. For example, transduction groups are shown at 0 min and 3

min, with *thrB* and *ilvS* the closest genes; the exact distance between the two genes is less than 3 min, and the two genes might be jointly transduced by P1 or even by P22 phage.

SUMMARY OF THE ORDER OF GENES ON THE LINKAGE MAP

To clarify the basis for the location of the genes on the map shown in Fig. 1 and to indicate the assurance with which gene positions are assigned, a discussion of the gene order is presented below, starting at 0 min. Conjugation refers to F-factor-mediated conjugation; in those cases where *col*-factor-mediated conjugation is used, this will be stated. Transduction normally refers to P22-mediated transduction; in those cases where ES18, P1, or other phages are used, this also will be stated. In addition, analysis is sometimes based on the isolation of deletion mutants. It is important that the reader understand clearly that the assignment of gene position is based in many cases on firm evidence by several groups, whereas in other cases only approximate mapping has been done by conjugation or a similar method which permits analysis of large areas of the chromosome, with joint transduction studies either not attempted or not successful. Only investigations essential to the present conclusions on the genetic location are cited in the text. Other references, dealing with the genetic fine structure of the genes and with their function, are cited in Table 1.

In some cases, the joint transduction data on which the map sequences shown in Fig. 1 are based are summarized in tables which accompany the text. In other cases, the reader must find the data in the original references. A degree of selection of data has been necessary in the tables because a single report may present more than one linkage value for a particular gene interval, due to random variation, and to selective differences in different experiments. In some cases, an average has been used; in others, the range of values observed is reported. Joint transduction data appear not to be greatly affected by the environmental conditions of a cross, and hence the data from experiments done at different times by different workers can be compared.

A similar discussion of gene order was presented in an earlier edition of the linkage map (246). In this edition, genes will be mentioned in most cases only if they are newly described or if their location has been corrected.

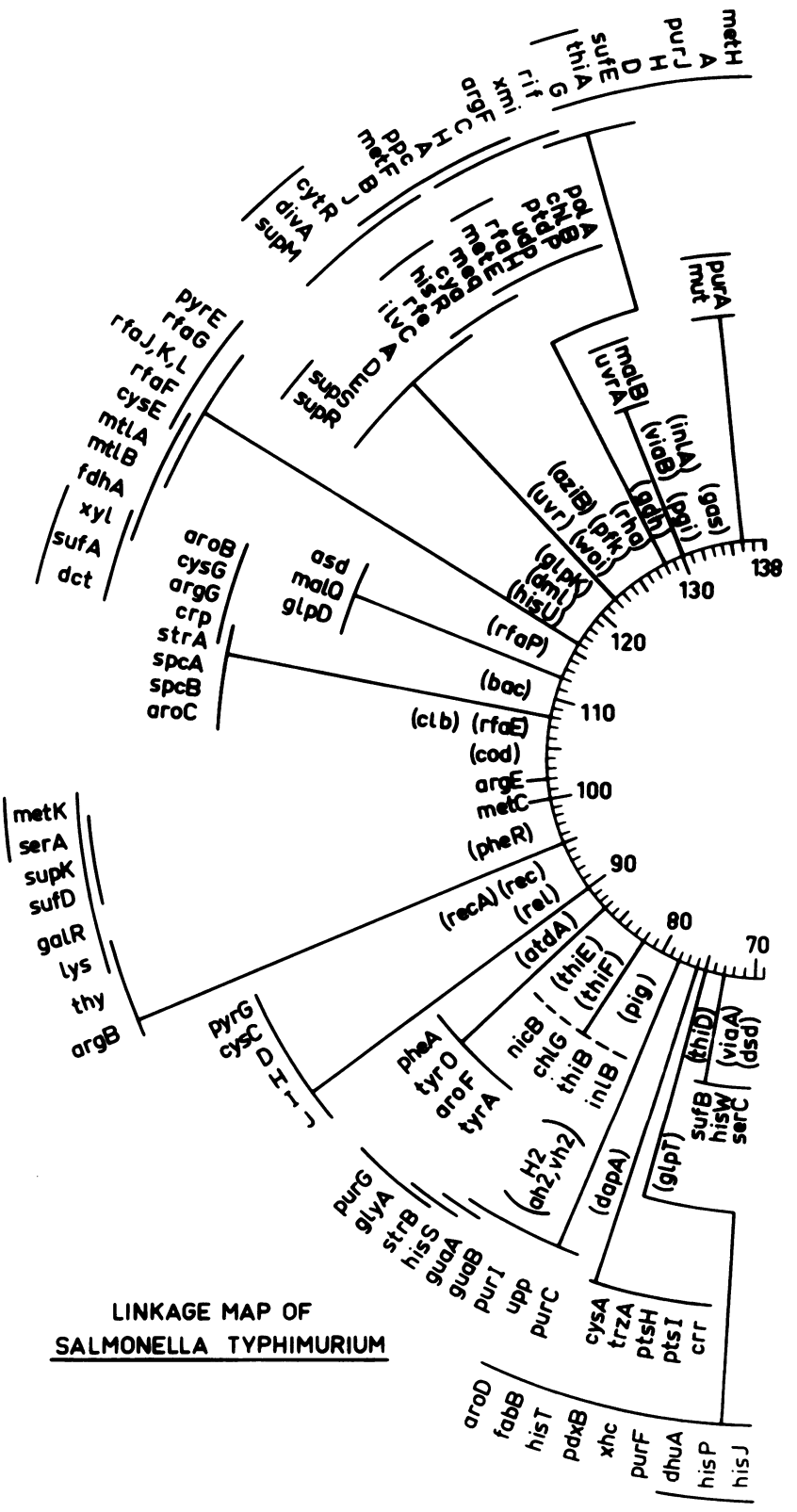
Region from 0 to 32 min

At 0 min is placed a transduction linkage group encompassing genes from *argI* to *thrB*. The P22 transduction data for this group are summarized (Table 2). Alleles formerly called *argD* (for ornithine transcarbamylase), and placed at 8 min (246), have been shown to be alleles of *pyrA*, at min 3, lacking carbamate kinase (287). Mutants lacking ornithine transcarbamylase, now named *argI*, have been isolated and found to be linked to *pyrB* (Table 2) (287). The position of *trpR*, a gene giving resistance to 5-methyl tryptophan and derepression of tryptophan enzymes, has been refined (285). The gene *fdp* is mapped approximately by Jargiello and Hoffee (*personal communication*).

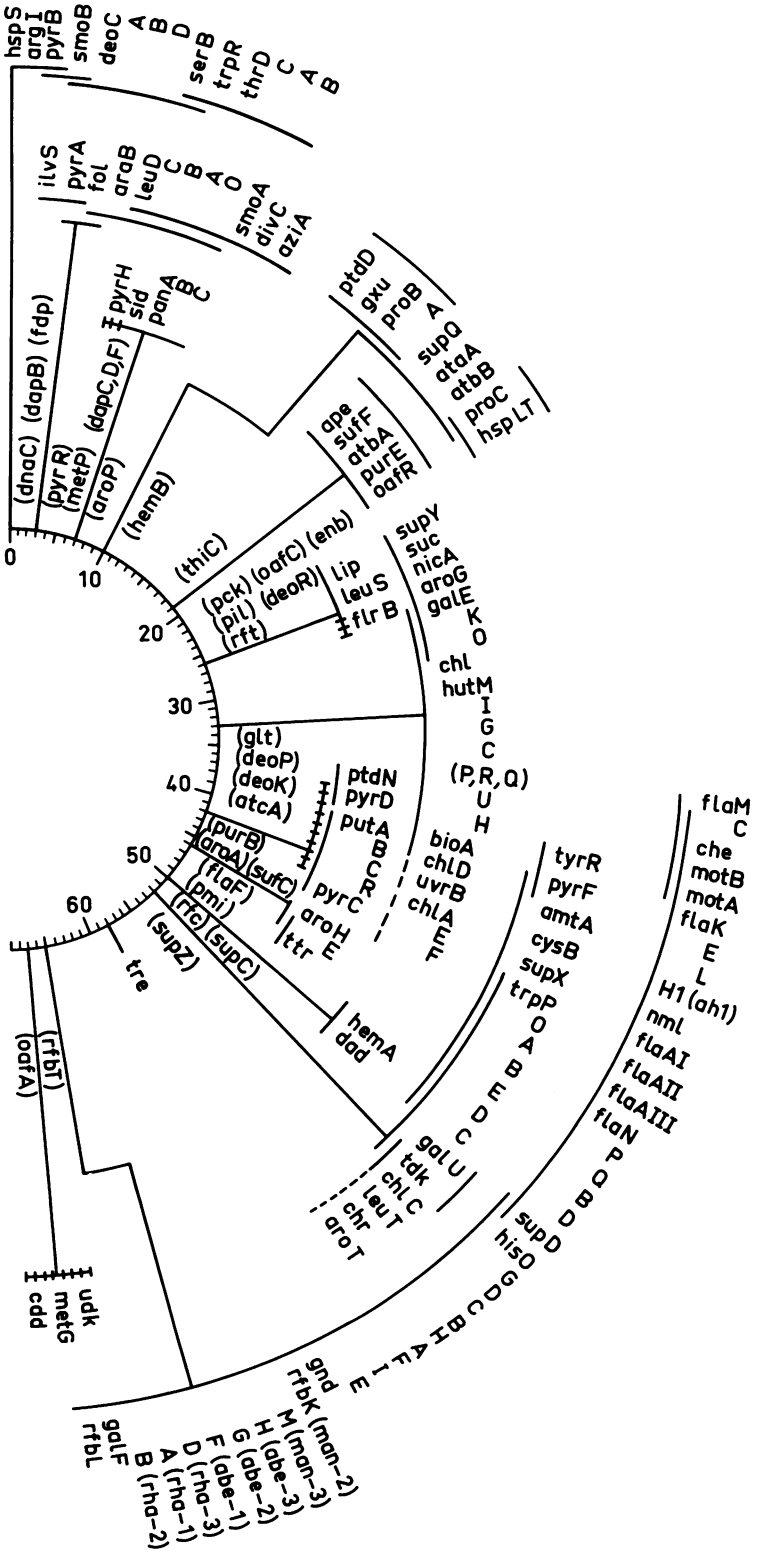
The map sequences *ilvS-pyrA-fol* (40) and *fol-araB-leuD* (Margolin, *personal communication*) are based on joint transduction frequencies. The order *araB-leuDCBAO* was determined by transduction crosses (185). The order *leu-* (*smoA-divC*)-*aziA* is based on joint transduction crosses with *leu* (67; Kłopotowski, *personal communication*). The mutant *divC* has a defect in cell division (67). A mutant with a defect in DNA synthesis and in cell division is called *dnaC* and is placed between *leu* and *thr*, based on conjugation data; joint transduction to nearby genes could not be demonstrated (273). The genes *ars* and *aus* are removed from the map, as they are considered to be alleles of *pyrA* (111a, 216; Ingraham, *personal communication*).

Between 3 and 10 min, several genes have been placed by conjugation, their relative order unknown: *metP* (15); *pyrR* (215); *dapC, D, F* (Shuster and Betts, *personal communication*); and *aroP* (6) (formerly placed incorrectly at 30 min [246]). In this region is another group; one of the known classes of *sid* mutants is jointly transduced with *pan* (Pollack and Neilands, *personal communication*), and *pyrH* is transduced with *panABC* by P1 phage (Ingraham, *personal communication*). There is some evidence that *pyrR* is an allele of *pyrH*, but whereas *pyrH* is jointly transduced with *pan* by P1, *pyrR* is not jointly transduced; however, the latter allele was tested with P22, which is a smaller phage. The gene *carA*, now called *ptsI*, was incorrectly placed in this region, and is now moved to min 76 (169; Levinthal, *personal communication*).

The genes for proline biosynthesis are jointly transduced (199); analysis of proline deletions lacking the attachment site I for phage P22 (*attA*) reveals the order *proBA-attA-proC*



LINKAGE MAP OF
SALMONELLA TYPHIMURIUM


hspS
argI
pyrB
smoB
deoC
A
B
D
serB
trpR
thrD
C
A
B
ilvS
pyrA
fol
araB
leuD
C
B
A
O
smoA
divC
aziA
pyrH
sid
panA
B
C
(dapC,D,F)
(dnaC)
(dapB)
(fdp)
(pyrR)
(metP)
(aroP)
(hemB)
(thiC)
ptdD
gxu
proB
A
supQ
ataA
atbB
proC
hspLT
ape
sufF
atbA
purE
oafR
0
10
20
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50
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(pck)
(oafC)
(enb)
(pil)
(deoR)
(rft)
Lip
leuS
flrB
supY
suc
nicA
araG
galE
K
O
chl
hutM
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(P,R,Q)
U
H
(glt)
deoP)
deoK)
(atcA)
ptdN
pyrD
putA
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(araA)
(sufC)
(flaF)
(pmi)
(rfc)
(supC)
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tre
pyrC
aroH
E
ttr
R
bioA
chlD
uvrB
chlA
E
F
tyrR
pyrF
amtA
cysB
supX
trpP
O
A
B
E
D
C
hemA
dad
galU
tdk
chlC
leuT
chr
aroT
flaM
C
che
motB
motA
flaK
E
L
H1 (ah1)
nml
flaAI
flaAII
flaAIII
flaN
P
O
D
B
supD
hisO
G
F
E
I
A
L
gnd
rfbK (man-2)
M (man-3)
H (abe-3)
G (abe-2)
F (abe-1)
D (rha-3)
A (rha-1)
B (rha-2)
galF
rfbL
(rfbT)
(oafA)
udk
metG
cdd

TABLE 2. *Percentages of P22-mediated joint transduction in the linkage group at min 0*[a]

hspS	*argI*	*pyrB*	*smoB*	*deoC*	*A*	*B*	*D*	*serB*	*trpR*	*thrD*
		0.5 (C)		2 (C)				75 (285)	50 (285)	
								10–40 (285)		
					0 (239a)					
	70 (287)				3 (145)			20 (239a)		
	3 (KK)						9 (145)			
			0 (KK)							

[a] The first number, outside the brackets, is the percentage of joint transduction of the two genes at the ends of the lines. The number shown in brackets is a published reference, found in Literature Cited. The letter in brackets is a reference to a personal communication, listed in a footnote to Table 1.

(270); *supQ* is close to *proA* and may be between *proA* and *ataA* (154). The genes *gxu* (112) and *ptdD* (Miller, *personal communication*) are jointly transduced with *proAB*, *gxu* giving 30 to 40% joint transduction, but their relative locations are not known. In addition to *ataA*, the attachment site II for prophage P27, *atbB*, maps close to *pro* (17). Early reports indicated that *proAB* and *proC* are jointly transduced (199), though some workers have been unable to confirm this. The gene *hspLT* is jointly transduced with *proC* (Colson, *personal communication*). The gene *thiC* has been added to the region between *proAB* and *purE* by conjugation crosses. The genes *sufF* (238) and *ape* (Miller, *personal communication*) are jointly transduced with *purE* by using P22 phage, whereas *atbA* and *oafR* are jointly transduced with *purE* by using ES18 phage (17). In the *purE-gal* region, placed arbitrarily at 26 min, are the genes *lipS* and *leuS*, jointly transduced by P22 phage, and *flrB*, linked to *lip* and *leuS* by P1 phage; based on conjugation studies, they are arranged in the order *purE-lip-leuS-flrB-gal* (Calvo, *personal communication*). Mutants in *deoR*, which are constitutive for enzymes determined by *deoA*, *B*, *C*, and *D*, are mapped in the same region by interrupted conjugation crosses (36a; Hoffee, *personal communication*).

Region from 33 to 55 min

The following order of genes has been revealed by transduction studies by Magasanik and co-workers (46, 194, 267, 269): *aroG-(galEKO)-hutIGC(PRQ)UH-bio*, with orientation of the group not known. Deletions obtained through selection of chlorate-resistant mutants have revealed the order *gal-bio-chlD-uvr-chlA-chlE-chlF* (281); other studies with the same type of system have shown the order *nic-aroG-gal-chl-hut(IG) CPUH-bio-uvrB* (Alper and Ames, *personal communication*; Presslitz and Magasanik, *personal communication*). This suggests an inversion between *E. coli* and *S. typhimurium*, for the map order in *E. coli* is *gal-chlD-hut-bio-uvr-chlA* (2), assuming that the homology of the *chlD* gene is established in the two genera.

The gene *pyrC* is placed at 42 min, based on interrupted conjugation crosses (247); *putABCR* are all jointly transduced, though the order is not known (88, 209; Brill, *personal communication*). The *put* genes are jointly transduced with *pyrC* by P22 (1 to 10%) and by P1 (50%) (Roth and Grabnar, *personal communication;* Brill, *personal communication*). The gene *ptdN* is jointly transduced with *pyrD* (Miller, *personal communication*),

FIG. 1. *Linkage map of Salmonella typhimurium. This figure represents a closed circular linkage map, though for convenience it is shown in two halves. The numbers on the inner circle represent the time-of-entry intervals, in minutes, for Hfr strains (247). Many genes have been added to this map, based on evidence referred to in Table 1, and in the text. All the genes covered by a set of arcs and shown at a specific point on the chromosome are part of a transduction linkage group. Those genes shown over a single arc are carried by a single transducing fragment. Smooth lines represent P22 or ES18 transduction fragments, cross-hatched lines represent P1 transduction fragments. Genes shown over a broken arc are mapped by deletion studies, and are not necessarily part of the transduction fragment to which they are attached. An arc outside a group of genes, e.g., dhuA, hisP, and hisJ at 75 min, indicates that their order is uncertain. The position of genes shown in brackets is known only approximately, usually from conjugation studies.*

but the relative order of these genes is not known. The gene *dadA* is jointly transduced with *hemA* (Walczak, Wild, and Kłopotowski, *personal communication*).

In *S. typhimurium*, the order *pro-gal-pyrF-cysB-trp-his* is based on the interrupted conjugation crosses (247) and the isolation of F*trp* factors (248). Gollub and Sprinson (*personal communication*) find *tyrR* 14% jointly transduced with *pyrF*, but not transduced with *trpA*, whereas *amtA* is mapped by joint transduction into the *cysB-pyrF* region (Wiater and Kłopotowski, *personal communication*). Deletion mutants reveal the position of *supX* (201a), of *chr* (75), and of *aroT* and *leuT* (295; Corwin and Thorne, *personal communication*). The gene *tdk* is jointly transduced 4% with *trp* by using phage P22 (26), and *galU* is jointly transduced 10% or less with *trp* by ES18 (Krishnapillai and Stocker, *personal communication*); both show no transduction with *pyrF*, but their relative order is not known. The gene *chlC* is jointly transduced with *tdk* but not with *trp*; it is transferred as a proximal gene by SU418 (Hfr B2) (249; Casse, *personal communication*), whereas *tdk* is transferred as a distal gene (26). Thus in *S. typhimurium* the gene order is *purB--pyrF-cysB-trp-galU-tdk-chlC--his*; the *pyrF-cysB-trp-galU-tdk-chlC* region (292) of *E. coli* is inverted in *S. typhimurium* (248).

Region from 56 to 105 min

The order for the *his* loci (171) and for *gnd* (202) was determined by transduction and overlapping deletions; data from deletions support the order *rfbL-B-A-D-(F-G)-H-N-M-K* (212). The gene *supD* is jointly transduced with *flaABD* but not with *flaC*; earlier linkage to *his* has not been confirmed (101a). The order of *fla* and *mot* genes has been modified from that reported earlier (246), based primarily on work by Yamaguchi et al. (311). These workers isolated mutants of *S. abortus-equi*, determined their complementation groups based on crosses to known *S. typhimurium* mutants, and determined the map order by using joint transduction frequency, deletion mutants, and three-point reciprocal crosses. Combining these results with other reports (97, 140, 278) indicates the order *his-supD-flaD-B-Q-P-N-AIII-AII-AI-nml-H1(ah1)-flaL-E-K-motA-motB-cheflaC-M*.

The genes *hisE-udk-metG-cdd* are jointly transduced in that order by phage P1, but not by P22 (26). Phage P22 gives 60% joint transduction of *hisW* and *sufB*, and the order (*sufB-hisW*)-*glpT-purF-aroD* is established by conjugation crosses (238). The order *hisP-purF-xhc-pdxB-hisT-fabB-aroD* is established by joint transduction data (131, 240). The position of *dhuA* is fixed by joint transduction with *purF* (158), and *serC* is jointly transduced with *hisW* (Ciésla and Kłopotowski, *personal communication*). The gene *thiD* is in the *purF* region, for it is complemented by the factor F32, which carries the *purF* region, but it is not jointly transduced with *purF* (Gots, *personal communication*). The genes *crr-ptsI-ptsH-trzA-cysA* are arranged in this order, based on three-point tests and deletion mapping (74, 244a). The order *purG-glyA-strB-hisS-guaA-purI-upp-purC* is based on joint transduction studies (25, 240). The attachment site for prophages $P6_1$ and P14, *atdA*, is close to Hl (181).

The linkage group *inlB-thiB-chlG-nicB* is determined by chlorate-resistant deletions (282). Some of the *thiE, F* mutants mapped in this general region (Gots, *personal communication*) may be homologous to *thiB*. A single transducing fragment carries *phe* and *tyr* (213), as well as *aroF* and *tyrO* (formerly *fpr*) (109). The gene *pyrG* is jointly transduced with *cysCD* (25). Joint transduction crosses, summarized in Table 3, indicate the map order *argB-thy-lys-galR-sufD-supK-serA-metK*. Analysis of conjugation crosses indicates the order *serA-metC-argE-cod-strA*, with none of these genes jointly transduced (26). The gene *pheR* is complemented by the *E. coli* episome F116, and is placed in the 96-min region (Gollub and Sprinson, *personal communication*).

Region from 106 to 138 min

A summary of analysis of joint transduction established the gene order *aroC-spcB-spcA-strA-(crp-argG)-cysG-aroB* (132, 214, 308). Conjugation analysis shows *bac* linked to *strA* and *cysG*, but joint transduction with *cysG*, *strA*, *malA*, *aroB*, *aroC*, *metC*, or *argG* was not detected (10).

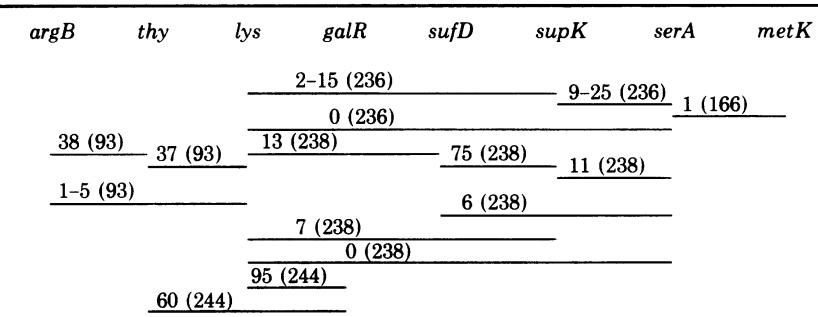
A maltose allele is located by interrupted conjugation at min 111 (251). Aceves, Ortega, and Artis (*personal communication*) have mapped a mutant lacking amylomaltase, *malQ*, linked by ES18 transduction to *glpD*, in this region. Shuster and Betts (*personal communication*) find a *glpD* allele, jointly transduced with *asd*, in this general region by using conjugation. Joint transduction analysis (163, 251) determined the map order *xyl-mtlA-mtlB-cysE-rfaF-(rfaJ,K,L)-rfaG-pyrE* (163, 251). The genes *sufA* (238), *dct* (Parada and Ortega, *personal communication*), and

fdhA (66) are jointly transduced with *xyl*, with *fdhA* between *xyl* and *mtl*, but the relative order of the genes is not known. The gene *glpK* (formerly *glk*) was placed in this region by *col*-factor-mediated conjugation studies (279). Close to *xyl* the gene *rfaP* has been mapped by conjugation crosses (179).

The sequence of the genes shown at 122 min, from *supR* to *chlB*, is based on joint transduction data summarized in Table 4; the order is indicated in Fig. 1. This group includes *chlB*, *ptdP*, *udp*, *rfaH*, and *meq*, not shown on the earlier map (246). The following genes have been added by conjugation to the 122- to 128-min region of the map, their relative order unknown: *woi* (Palermo and Antón, *personal communication*); *pfk* (C. Calvo and Ortega, *personal communication*). A mutant similar to the *ilvB* mutation of *E. coli*, for acetohydroxy acid synthetase, has been isolated in *S. typhimurium*, but since it is not precisely mapped, it is not shown in Fig. 1; there seem to be two forms of acetohydroxy acid synthetase in *S. typhimurium*, with loss of one of the two forms leading to valine sensitivity (11, 47, 221; Umbarger, *personal communication*). A mutation leading to a defect in DNA polymerase I (*polA*) is 0.3% co-transducible with *metE* (Levine and Whitfield, *personal communication*).

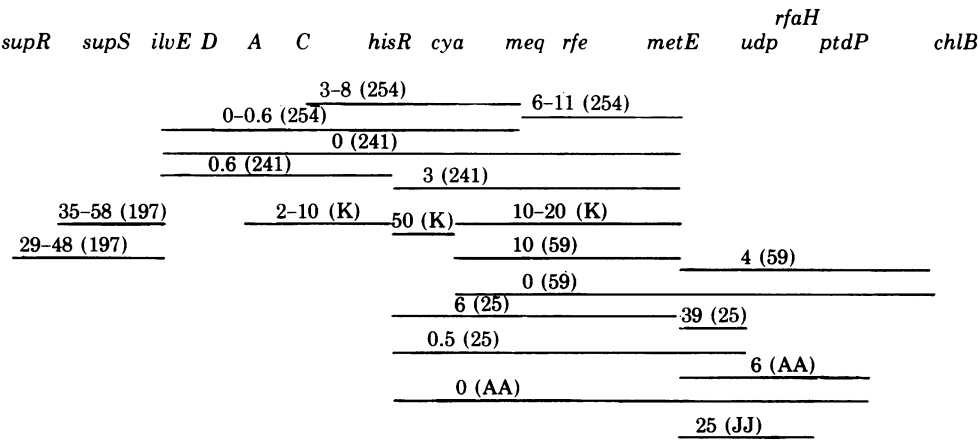
A transduction linkage group, placed at min 128, from *divA* to *metH*, is shown in Fig. 1. The joint transduction data on which the order is partially based are summarized in Table 5. Two separate transduction linkage groups shown earlier (246) have been linked through *rif*; *xmi*, a temperature-sensitive mutant of unknown function, has been mapped after localized mutagenesis (131). *DivA* mutants result in a septum-initiation defect; they were formerly *wrkA* (67). The orientation of the group is known from conjugation crosses (16, 247) and from Hfr strains, with point of origin

TABLE 3. *Percentages of P22-mediated joint transduction in the linkage group at min 95*[a]



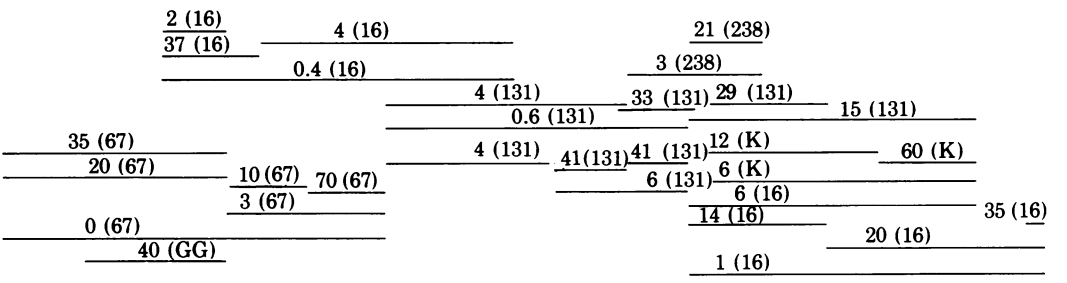
[a] Symbols in the table are described in the footnote to Table 2.

TABLE 4. *Percentages of P22-mediated joint transduction in the linkage group at min 122*[a]



[a] Symbols in the table are described in the footnote to Table 2.

TABLE 5. *Percentages of P22-mediated joint transduction in the linkage group at min 128*[a]



[a] Symbols in the table are described in the footnote to Table 2.

inside the group (249). The gene *malB* is 1 to 2 min clockwise from *metA* by interrupted conjugation crosses (250), and *uvrA* is jointly transduced 26% with *malB* (8a). Ortega and Aguilar (*personal communication*) located *gdk* at approximately 128 min by conjugation crosses. Genes for host restriction and modification, allelic to the *E. coli* KB system, are mapped by conjugation in the following order: *hspS-thr-leu* (72), and are jointly transduced with *pyrB* (Colson, *personal communication*).

DISCUSSION

The increased precision in genetic analysis of *S. typhimurium* may be measured by the progress in joint transduction studies, primarily with bacteriophage P22, which locate more than one gene in a transduction linkage group. In 1965, 133 genes were known on the linkage map. Of these, 80 were jointly transduced with another known gene, but in most cases these other genes were for a related function and were often contiguous; there were only 15 known instances of transduction linkage of genes for unrelated functions (247). Now, in 1972, of 323 genes placed on the linkage map (Fig. 1), there are 113 cases in which joint transduction is detected between genes for unrelated functions. There is a high probability that a newly identified gene, once located approximately by conjugation analysis, can be shown to be jointly transduced with a known gene in that region. Because of its larger size, the use of the bacteriophage P1, though awkward because of the need to isolate P1-sensitive mutants (218, 222), may assist greatly in locating genes in transduction fragments. Thus it may soon be possible to construct a linkage map encompassing the entire chromosome, based entirely on overlapping transduction fragments.

Further genetic analysis may be assisted by new methods. For example, the localized mutagenesis method (131) may be used to map new genes close to a known auxotrophic gene. Further systems permitting direct selection of mutants, many of which are deletions, as in mutants resistant to chlorate (2, 280), may be possible.

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