BACTERIOLOGICAL REVIEWS, Dec. 1976, p. 847-868
Vol. 40, No. 4

Printed in U.S.A.

The Genome of Bacteriophage T4

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INTRODUCTION

Over the past three decades, bacteriophage T4 has been genetically, biochemically, and structurally characterized to the point where it is now one of the best understood biological systems. T4 is a complex deoxyribonucleic acid (DNA) virus with a genome large enough to accommodate between 160 and 170 "average-size" genes of 1,000 nucleotide pairs. About 140 T4 genes now have been identified genetically and, to some extent, characterized functionally. The resulting information provides a fairly complete picture of how such a genome is organized and how it programs the process of viral multiplication in a host bacterial cell.

This article provides an overview of the organization and function of the T4 genome, as well as a current reference source of information on the individual genes of T4. The number of essential genes defined by amber (*am*) and temperature-sensitive (*ts*) mutations has not changed appreciably from the 65 identified in the early studies of Epstein, Edgar, and their collaborators (67), although the functions of these genes continue to become more completely understood (34, 58, 214). However, a considerable number of new so-called nonessential genes has been identified and characterized in the past few years. A review prepared in 1973 (214) included 30 of these genes, and the total now has increased to over 70.

We have summarized current knowledge on the locations, sizes, and functions of T4 genes in the form of a detailed linkage map, tables of gene functions, and a chart showing classes of gene functions. To keep the bibliography to a reasonable length, we have not attempted to reference all of the papers from which information has been taken. Instead, wherever possible, we have cited recent research publications or review articles that in our judgment provide the most convenient access to earlier literature. Additional references to original work may be found in several other recent compilations of information on the T4 genome (34, 58, 62, 140, 155a, 214).

GENE CLASSES AND GENE NAMES

Laboratory growth conditions for T4 most commonly employ *Escherichia coli* B as the host bacterium and Hershey broth or agar as the growth medium. These conditions were used in the early isolation of mutants that carry conditionally lethal *am* and *ts* mutations (67). Consequently, the genes defined by these mutations have been termed "essential genes." As a matter of historical practice, essential genes in T4 have been designated by numbers or, in three cases, by single lowercase letters (*e*, *t*, and *y*). In general, the numbering of these genes is in map order, but since not all were discovered when the original set was numbered, map order and numerical order do not correspond strictly.

More recently, increasing numbers of nonessential genes have been discovered. These genes are defined by mutations that alter or prevent phage growth under some conditions, but do not prevent plaque formation on *E. coli* B grown in Hershey medium. The nonessential genes are designated by two-letter or three-letter mnemonic symbols for the corresponding gene functions or defective phenotypes. Exceptions are the classically described *r* genes, of which five are now known (*r*I-*r*V), defined by mutations that cause rapid (premature) lysis. We have replaced four other previously used one-letter symbols with more descriptive three-letter designations. The three genes *v*, *w*, and *x*, defined by mutations to increased ultraviolet (UV) sensitivity, have been redesignated as *den*V, *uvs*W, and *uvs*X, respectively. Likewise, gene *m*, defined by a suppressor of gene 30 mutations, has been redesignated *sum*. Otherwise, we have used the gene designations of the original authors.

There are a number of possible confusions concerning gene nomenclature in phage. The designation of a gene as nonessential is necessarily arbitrary; many of the known nonessential genes clearly are essential under certain conditions (see, for example [48]). Nevertheless, we have elected to retain this distinc-

tion, since the standard growth conditions are widely used, and since renaming all the known essential genes would cause much unnecessary confusion. It should be kept in mind, however, that outside of the laboratory the essentiality or nonessentiality of phage genes is a relative rather than an absolute distinction.

Another confusion can arise between nonessential gene names and the generic names for classes of mutants obtained by a particular selection procedure, such as *am, ts, cs* (cold sensitive), *hus* (hydroxyurea sensitive), *far* (folate analogue resistant), and so on (see Tables 1 and 2). For example, there are three mutations to hydroxyurea sensitivity, *hus*-1, *hus*-3, and *hus*-7, that have been shown to fall into three different genes with different names: 49, *dex*A, and 39, respectively (78). To minimize such confusion in the future, we strongly recommend that new phenotypic classes of mutants be given *two-letter* designations, and that, in accord with the accepted conventions for bacterial gene nomenclature (46), new nonessential genes be given *three-letter* designations.

GENE LOCATIONS AND GENE SIZES

Early maps of T4 (63, 185) were constructed from frequencies of recombination between conditionally lethal mutations, using an empirically derived mathematical mapping function to correct for systematic effects of high negative interference and thereby obtain internally consistent map distances (185). By this approach the genetic map first was shown to be circular (188). The total map length was estimated to be about 2,500 map units, where a map unit corresponds to a recombination frequency of 1% as determined by measurements made in the range of 0.01 to 1%, over which recombination distances are generally additive (185). (There is some confusion in the literature on this point. It is incorrect to estimate distance in map units directly from uncorrected recombination frequencies in the nonadditive range of greater than 1%. For example, the distance between two markers that recombine with a frequency of 4% is 9.5 map units, as calculated using the four-parameter switch function of Stahl et al. [185; Fig. 6], but assuming a total map length of 2,500 map units.)

Genetic distances determined from recombination frequencies using the mapping function can be related to physical distances only by assuming that the physical length of a map unit is constant over all intervals of the genome. There is now considerable evidence that this assumption cannot be made. The ingenious marker-rescue test of Mosig using incomplete T4 genomes (138, 139) (formally analogous to cotransduction mapping in bacteria) permitted the first estimates of physical distances between markers by a method independent of recombination frequencies. The results showed that the recombinational map was distorted in some regions. This conclusion has been supported by subsequent, more direct measurements of physical map distances, made from electron micrographs of heteroduplex DNA molecules containing one strand from each of two deletion mutants (106). In addition, estimates of intragenic physical distances have been made by comparing the electrophoretically determined molecular weights of polypeptide fragments resulting from different *am* mutations in a given gene (8, 30), and have shown that in at least one gene, recombination frequencies per nucleotide pair differ drastically in two adjacent intervals (8). Since the polypeptide products of many T4 genes now have been identified as bands of approximately known molecular weight by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (120, 147, 198), this method now provides a general means for estimating minimum physical gene sizes, and thus minimum distances between adjacent genes.

The most reliable estimate of T4 genome size is now 1.66×10^5 nucleotide pairs (106), or 166 kilobase pairs (kb). Thus, the *average* physical length of a genetic map unit can be estimated as 1.66×10^5 divided by 2,500, or about 70 nucleotide pairs. Denatured T4 DNA fragments exhibit the renaturation kinetics of a single frequency class, with a $C_0t_{1/2}$ value of about 0.3 mol-s/liter under standard conditions, indicating that the genome contains less than about 3% repetitive sequences (24).

In apparent disagreement with the genome size, the DNA molecules in T4 phage particles are 170 kb in length. Moreover, they are linear, despite the circularity of the genetic map. These discrepancies are explained by the arrangement of the nucleotide sequences in T4 DNA molecules. In a population of T4 genomes, the sequences are circularly permuted with respect to one another. That is, different molecules begin and end at different points in the sequence, and this property accounts for the observed circularity of genetic linkage (194, 195). In addition, each molecule is terminally redundant; that is, the sequence at one end is repeated at the other (126, 189). Each molecule therefore contains somewhat more than one genome-equivalent of DNA. The extent of the redundancy is about 2% of the genome size (106).

TABLE 1. *Bacteriophage T4 genes*[a]

Gene[b]	Map position[c] (kb)	Mutations isolated[d]	Promoter class[e]	Gene product, function, or defective phenotype[f]	Molecular weight of gene product[g]
Essential genes					
1	74	*am*	Q	Deoxyribonucleotide kinase (56)	22,000 (147)
2	75	*am*	[L]	Head completion (61); *d*: filled inactive heads and killing noninfectious phage particles (107), incomplete processing (cleavage) of head proteins (123, 198); may function to protect phage DNA following injection (52, 174, 175)	
3	76	*am, ts*	L	Tail tube, sheath stabilizing component (107, 108, 110)	29,000 (105)
4	79	*am*	[L]	Head completion (61); *d*: filled inactive heads and empty capsids (107)	
5	81	*am, ts*	L	Baseplate (107), central plug component (105)	37,000 (198)
6	82	*am, h, ts*	L	Baseplate (107), outer wedge component (105)	78,000 (198)
7	85	*am, h, ts*	L	Baseplate (107), outer wedge component (105)	127,000 (198)
8	87	*am, h, ts*	L	Baseplate (107), outer wedge component (105)	39,000 (198)
9	88	*am, ts*	L	Baseplate completion (105, 107)	30,000 (198)
10	90	*am, h, ts*	L	Baseplate (107), outer wedge component (105)	90,000 (198)
11	92	*am, ts*	L	Baseplate (61, 177), outer wedge component (103, 105)	25,000 (198)
12	93	*am, ts*	L	Baseplate completion (61, 105, 177), subunit of short tail fibers (101)	57,000 (198)
13	95	*am, ts*	L	Head completion (61), neck assembly, probably structural (40)	33,000 (198)
14	96	*am, ts*	L	Head completion (61), neck assembly, possibly structural (40)	30,000 (198)
15	97	*am, ts*	L	Tail completion, connector to collar (107, 110)	32,000 (198)
16	98	*am*	[L]	Head completion (61), required for DNA packaging into capsid (107)	
17	98	*ac, am, ts*	L	Head completion (61), quinacrine resistance (*q*; 154), required for DNA packaging into capsid (107), probably nonstructural (198)	69,000 (198)
18	100	*am, CBW, ts*	L	Tail, sheath subunit (107, 109)	70,000 (198)
19	101	*am, ts*	L	Tail, central tube subunit (107, 108, 109, 110)	20,000 (198)
20	102	*am, ts*	L	Head component; *d*: polyhead (61, 122)	65,000 (198)
21	103	*am, ts*	L	Head assembly (61, 122), protease for cleavage of head structural proteins (123, 151, 196)	25,000[h]
22	104	*am, ts*	L	Head assembly (61, 122), major protein of assembly core in τ particle; degraded (120, 121, 123, 151, 170, 196)	32,000 (198) ↓ Small peptides
23	105	*am, mi, ts*	L	Head component (61, 122), major capsid subunit; cleaved during assembly (30, 120, 196); defects can alter head size (54)	55,000 (198) ↓ 47,000
24	107	*am, os, ts*	L	Head component (61, 100, 122), minor capsid subunit; cleaved during assembly (120, 196); defects can alter head size (17)	47,000 (198) ↓ 45,000
25	112	*am*	L	Baseplate (107), outer wedge component (105)	15,000 (104)
26	113	*am, ts*	[L]	Baseplate (107), central plug formation, probably nonstructural (105, 114)	
27	115	*am, ts*	L	Baseplate (107), central plug component (105)	48,000 (12)
28	116	*am, ts*	L	Baseplate (107), central plug formation, probably nonstructural (105, 114)	
29	117	*am, ts*	L	Baseplate (107), central plug component (105)	77,000 (198)

TABLE 1—*Continued*

Gene[b]	Map position[c] (kb)	Mutations isolated[d]	Promoter class[e]	Gene product, function, or defective phenotype[f]	Molecular weight of gene product[g]
30	122	*am, ts*	E or Q	DNA ligase (*lig*) (69); *d*: arrested DNA synthesis; suppressed by *den*A, *sum* (see entry under this gene), and *r*II mutations (98)	68,000 (147)
31	127	*am, ts*	E or Q[h]	Head assembly (61, 122); interacts with host in organization of capsid subunits (41, 76, 77, 191, 192, 218)	16,000[h]
32	145	*am, ts*	Q	DNA-binding protein (3, 4); translational repressor of its own synthesis (163)	36,000 (147)
33	147	*am*	[Q]	Polypeptide associated with host RNA polymerase (157, 187); *d*: no synthesis of late proteins (23)	10,000 (187)
34	149	*am, c, ts*	L	Tail fiber (63, 111), subunit of proximal half; carries A antigens (18, 215)	145,000 (198)
35	152	*am, ts*	L	Tail fiber (63, 111), minor component of distal half (18, 215)	39,000 (198)
36	153	*am, CBW, ts*	L	Tail fiber (63, 111), minor component of distal half (18, 215)	24,000 (198)
37	155	*am, h, ts*	L	Tail fiber (63, 111), major component of distal half (18, 215); carries bacterial host range determinants (6, 7)	115,000 (198)
38	157	*am, ts*	L	Tail fiber (63, 111), assembly of distal half-fiber precursor; nonstructural (18, 19, 215)	28,000 (198)
39	4	*am, ts*	E	*d*: delayed DNA synthesis; leaky at 37°C (141, 229), no DNA synthesis at 25°C (141)	64,000 (147)
40	20	*am, ts*	[Q]	Head assembly; *d*: polyhead (122)[i]	13,000[h]
41	22	*am, ts*	Q	DNA replication, lagging strand chain initiation(?) (4); *d*: lack of or arrest of DNA synthesis, production of single-stranded DNA (149)	66,000 (147)
42	23	*am, ts*	E	Deoxycytidylate hydroxymethylase (206, 222); *d*: no DNA synthesis	25,000 (147)
43	27	*am, ts*	E, Q	DNA polymerase (*pol*) (4, 49, 201); *d*: no DNA synthesis	112,000 (147)
44	32	*am, ts*	[E]	Component of DNA replication complex (4); *d*: no DNA synthesis	35,000 (147)
45	33	*am, hus, ts*	[E]	Component of DNA replication complex (4), also associated with host RNA polymerase (183); participates in control of late transcription (224, 225); *d*: no DNA synthesis	24,000 (147)
46	36	*am, ts*	E	DNase(?) (25, 135, 155); *d*: arrest of DNA synthesis, decreased recombination, and impaired degradation of host DNA (16, 205); suppressed by *das* mutations (see *das* entry, below)	71,000 (147)
47	38	*am, ts*	E	DNase(?) (25, 135, 155); *d*: arrest of DNA synthesis, decreased recombination, and impaired degradation of host DNA (16, 205); suppressed by *das* mutations (see *das* entry, below)	37,000 (147)
48	118	*am*	L	Baseplate component (12, 107), probably on top surface (108, 110)	37,000 (198)
49	45	*am, hus, ts*	[E or Q]	DNase(?) (71); required for DNA packaging into capsid (125); defect suppressed by *fds*A, *fds*B mutations (see entries under these genes, below)	
50	78	*am*	[L]	Head completion (61); *d*: filled, inactive heads (107), incomplete processing (cleavage) of head proteins (123, 198)	
51	114	*am, ts*	L	Baseplate (107); plug formation; may be nonstructural (105, 114)	
52	161	*am*	E	*d*: delay in DNA synthesis at 37°C (141, 229), no DNA synthesis at 25°C (141)	51,000 (147)
53	80	*am, ts*	L	Baseplate (107), outer wedge component (105)	23,000 (104)

TABLE 1—*Continued*

Gene[b]	Map position[c] (kb)	Mutations isolated[d]	Promoter class[e]	Gene product, function, or defective phenotype[f]	Molecular weight of gene product[g]
54	120	*am*	L	Baseplate component (12, 107); initiation site for central tube polymerization(?) (108, 110)	36,000 (104)
55	41	*am, ts*	Q	Polypeptide associated with host RNA polymerase (157, 187); *d*: lack of late protein synthesis (23)	17,000 (147)
56	18	*am, ts*	[E or Q]	Deoxycytidine-deoxyuridine di- and triphosphatase (dCTPase-dUTPase); *d*: no DNA synthesis (201, 206)	15,000 (147)
57	73	*am*, other	Q	Assembly of long (18, 215) and short (101) tail fibers, nonstructural (53); may act in conjunction with a host component (160)	18,000 or 6,000[j]
58–61	19	*am*	[E or Q]	*d*: delay in DNA synthesis at 37°C, decreased recombination, increased UV sensitivity, leaky (141, 229); no DNA synthesis at 25°C (141)	
59	146	*am*	[E or Q]	*d*: arrest of DNA synthesis (167, 226); suppressed by *dar* mutations (see *dar* entry, below)	
60	3	*am*	E	*d*: delay in DNA synthesis, leaky at 37°C (141, 229), no DNA synthesis at 25°C (141)	
61				See 58-61 (229)	
62	31	*am*	[E]	Component of DNA replication complex (4); *d*: no DNA synthesis	18,000 (147)
63	133	*am, mi*	Q	Catalyzes tail fiber attachment to baseplate, nonstructural (215, 217, 220)	42,000 (198)
64	77	*am*	[L]	Head completion (61); *d*: filled inactive heads (107), incomplete processing (cleavage) of head proteins (123, 198)	
65	78	*am*	[L]	Head completion (61); *d*: filled inactive heads and empty capsids (107)	
e	66	*am, del, ts*	E, Q	Endolysin (phage lysozyme) (190); *d*: no cell lysis at end of normal infectious cycle (142); suppressed by *r*IV mutations (see *r*IV entry)	18,000 (198)
t	158	*am*	[L]	Lysis function, possibly a phospholipase (144); *d*: impairment of cell lysis, extended infectious cycle (97); suppressed by *r*II mutations (97); T4B mutant *st*II has same phenotype and locus (117)	
y	[110]	*am, uvs*		*d*: lethality, uncharacterized (131)	
Nonessential genes					
ac	162	*ac, del*	[E or Q]	Acriflavin uptake, acriflavin resistance (173)	
alc	[130]	–	[E or Q]	Allows late transcription of cytosine-containing phage DNA (182); probably polypeptide 2 associated with host RNA polymerase (187)[k]	
alt	–	–		Injected T4 capsid protein, alters host RNA polymerase α subunit; cleaved during head assembly (93)	79,000 (93) ↓ 61,000
ama	[164]	*ac*	[E or Q]	Resistant to aminoacridine (156)	
cd	[130]	–	[E or Q]	Deoxycytidylate deaminase (84, 85)	
cef	7	*del, pha*	[E or Q]	*d*: failure to grow on AR-8 (92)[m]	
dam	[53]	–	[E or Q]	*D*NA *a*denine *m*ethylase; mapped in phage T2 (27); *d*: hypermethylation or failure to methylate T2 or T4 phage DNA (88, 159)	
dar	[110]	*hus*	[L]	*D*NA *ar*rest *r*eversal; suppresses gene 59 mutations (223)	
das	[147]	–	[E or Q]	*D*NA *ar*rest *s*uppression; suppresses gene 46 and 47 mutations (91); same as *sua* (116)	
[D1]	165	*del*	[E or Q]	Nonessential region defined by deletions (48)	
dda	11	*del*, other	[E or Q]	*D*NA-*d*ependent *A*TPase (9, 92)	15,000 (45)

TABLE 1—*Continued*

Gene[b]	Map position[c] (kb)	Mutations isolated[d]	Promoter class[e]	Gene product, function, or defective phenotype[f]	Molecular weight of gene product[g]
*den*A	136	*del, hus*	[E or Q]	DNA endonuclease II (92, 158, 166); *d*: inability to degrade host DNA (89, 202), suppression of ligase$^-$ (gene 30) mutations in ligase$^+$ host (200)	
*den*B	164	*del*, other	[E or Q]	DNA endonuclease IV (48, 199); *d*: fails to degrade cytosine-containing T4 DNA (119), impaired breakdown of host DNA (119)	
*den*V	62	*del, uvs*	[E or Q]	DNA endonuclease V (227, 228); formerly *v* (87); injected with phage DNA (168); *d*: increased UV sensitivity	18,000 (137)
*dex*A	[9]	*del, hus*	[E or Q]	DNA exonuclease A (92, 203); *d*: impaired breakdown of host DNA	
frd	142	*am, del, far*, other	[E or Q]	Dihydrofolate reductase (83, 92, 230); nonessential component of baseplate (113)	29,000 (68)
*fds*A	[22]	–	[E or Q]	Suppresses gene 49 mutations (50); may be the same as gene x^l	
*fds*B	[110]	–	[L]	Suppresses gene 49 mutations (50); may be the same as gene y^l	
*gor*1	–			See *βgt*	
*gor*2	40	–	[E or Q]	Grows on *rif*^{R} host restrictive for T4; probably affects host RNA polymerase function (183)	
αgt	39	*am*, other	[E or Q]	α-Glucosyl transferase (75, 94, 161); *d*: failure to glucosylate phage DNA	
βgt	22	*am, gor*, other	[E or Q]	β-glucosyl transferase (74, 75, 161); *d*: failure to glucosylate phage DNA; same as *gor*1 (183)	46,000 (95)
hoc	–	–		*h*ighly antigenic *o*uter *c*apsid protein (96)	40,000 (96)
[*hm*]	[73]	–	[E or Q]	*d*: increased mutation frequency (55, 79)	
imm	24	–	[E or Q]	*d*: lack of immunity to superinfection (42, 197)	
*ip*I	73	*am, del, pla*	E	Internal protein I (20, 22); cleaved during assembly (196)	10,000→8,900 (196)
*ip*II	65	*am, del*	E	Internal protein II (20, 22); cleaved during assembly (196)	11,700→10,000 (196)
*ip*III	66	*am, del*	E, Q	Internal protein III (20, 22); cleaved during assembly (120, 196)	21,200→18,300 (196)
m				See *sum*	
mb	–	–	[E or Q]	Modifier of phage tRNA's (209)	
[*mms*]	–	–		Sensitive to methylmethanesulfonate; *d*: defective DNA repair (57)	
mod	12	*del*, other	[E or Q]	Modifies host RNA polymerase α subunit (92, 93)	
mot	159	*far, ts*	[E]	*m*oderation *o*f *t*ranscription of some early enzymes; *d*: failure to activate Q promoters (130); same as *far* P85 (33)	
ndd	163	*del, pla*, other	[E or Q]	*n*uclear *d*isruption *d*eficient (48, 179, 180); *d*: impaired host nuclear breakdown	
*nrd*A	140	*del*, other	[E or Q]	Ribonucleoside diphosphate reductase subunit (92, 230)	[85,000 or 35,000][h]
*nrd*B	138	*del*, other	[E or Q]	Ribonucleoside diphosphate reductase subunit (92, 230)	[85,000 or 35,000][h]
*nrd*C	49	*del*, other	[E or Q]	Thioredoxin (193)	10,400 (15)
p12,000	7	*del*	[E or Q]	Polypeptide of unknown function missing from electropherograms of deletion mutants (92)	12,000 (147)
p50,000	[9]	*del*	[E or Q]	Polypeptide of unknown function missing from electropherograms of deletion mutants (92)	50,000 (147)
*pla*CTr5X	6	*del, pla*	[E or Q]	*d*: failure to grow on CTr5X (92)	
*pla*262	164	*del, pla*	[E or Q]	*d*: failure to grow on CT262 (48)	
*pse*F	[9]	*del*, other	[E or Q]	*d*: lack of a deoxyribonucleotide-5′-phosphatase activity (92)[o]	
*pse*T	[130]	*pla*, other	[E or Q]	*d*: lack of a deoxyribonucleotide-3′-phosphatase activity (47)	

TABLE 1—*Continued*

Gene[b]	Map position[c] (kb)	Mutations isolated[d]	Promoter class[e]	Gene product, function, or defective phenotype[f]	Molecular weight of gene product[g]
[*psu*⁺SB]	–	*psu*	[E or Q]	Apparent general nonsense suppression (162)	
q				See gene 17	
*r*I	55	*del, r*	[E or Q]	*d*: rapid lysis (59, 60)	
*r*IIA	2	*del, pla, r*	E	Membrane protein (66); *d*: rapid lysis, inability to multiply on λ lysogens (59, 60); suppression of gene 30 (ligase) mutations (98)	95,000 (147)
*r*IIB	0	*del, pla, r*	E, Q	Membrane protein (204); *d*: rapid lysis, inability to multiply on λ lysogens (59, 60); suppression of gene 30 (ligase) mutations (98)	33,000 (147)
*r*III	129	*r*	[E or Q]	*d*: rapid lysis (59, 60)[p]	
*r*IV	20	*r*	[E or Q]	"Spackle"; *d*: rapid lysis, suppression of gene *e* mutations (65, 150)	
*r*V	[160]	*r, ts*	[E or Q]	*d*: temperature-dependent rapid lysis (118)	
rc	[7]	*ac*	[E or Q]	*r*apid clock (156); acriflavin resistance	
*reg*A	29	*hus, ts*	[E or Q]	Regulation of translation of several early enzymes (99, 207)	
*reg*B	61	*del, far*	[E or Q]	Regulation of translation of several early enzymes (33)	
rs	[164]	*ac*	[E or Q]	*r* suppression in host strain S/6/5 but not in host strain B/5 (156); acriflavin resistance	
sip	[160]	–	[E or Q]	Suppression of *r*II mutations (72)[q]	
soc	–	–		*s*mall outer capsid protein (96)	10,000 (96)
sp				"Spackle"; see *r*IV	
*st*I	[63]	*r*	[E or Q]	*d*: rapid lysis; isolated in T4B (117)	
*st*II				See essential gene *t*	
*st*III	[63]	*r*	[E or Q]	Suppression of *st*II (*t*) and *e* mutations in T4B (117)	
stp	163	*del*, other	[E or Q]	Suppression of *pse*T mutations (47, 48)	
suα				See *das*	
*su*30	[53]	–	[E or Q]	Enhances *r*II suppression of ligase mutations (115)	
sud	[9]	*del, hus*, other	[E or Q]	Suppression of gene 32 (DNA unwinding protein) defects (92, 124)	
sum	[3]	–	[E or Q]	Suppression of gene 30 (ligase) mutations (35)	
td	141	*del*, other	[E or Q]	Thymidylate synthetase (169, 176); nonessential baseplate component (112)	29,000 (28)
tk	56	*am, BrdU, del*		Thymidine kinase (31–33)	
tRNA^{arg}	70–72[r]	*del*	Q	Arginine tRNA[s]	
tRNA^{gln}	70–72[r]	*del, psu*	Q	Glutamine tRNA (1, 82, 132, 209); mutates to psu_2^+ (38)	
tRNA^{gly}	70–72[r]	*del*	Q	Glycine tRNA (1, 82, 132, 209)	
tRNA^{ile}	70–72[r]	*del*	Q	Isoleucine tRNA (1, 82, 132, 209)	
tRNA^{leu}	70–72[r]	*del*	Q	Leucine tRNA (1, 82, 132, 209)	
tRNA^{pro}	70–72[r]	*del*	Q	Proline tRNA (1, 82, 132, 209)	
tRNA^{ser}	70–72[r]	*del, psu*	Q	Serine tRNA (1, 82, 132, 209); mutates to psu_1^+, psu_a^+ and psu_b^+ (133, 210, 211)	
tRNA^{thr}	70–72[r]	*del*	Q	Threonine tRNA[s]	
unf	[130]	–	[E or Q]	*d*: impaired unfolding of host DNA (181)	
*uvs*W	[110]	*hus, uvs*	[L]	*d*: increased UV sensitivity, decreased recombination (86)	
*uvs*X	25	*uvs*	[E or Q]	*d*: increased UV sensitivity (55, 87)	
v				See *den*V	
vs	58	*am, del* other	E	Valyl-tRNA synthetase-modifying peptide (134, 143)	10,500 (127)
w				See *uvs*W	
wac	94	*am, CBW*	[L]	*w*hisker *a*ntigen *c*ontrol (51, 70); whisker subunit (40, 70); *d*: impaired tail fiber attachment (18, 216), altered tail fiber retraction in mature phage (39)	53,700 (196)
wh				See *frd*	
x				See *uvs*X	
y				See part A, Essential genes	

TABLE 1—*Continued*

[a] The following general articles, *not* referred to in the table, are useful sources of additional information and references as follows: for the original assignment of essential gene functions (67); for early functions of phage-coded enzymes (128, 129); for the original classification of gene functions in assembly of phage particles using in vitro complementation (63, 219); for recent work on phage assembly (29, 64, 76, 171, 212, 221); for gene functions in general (26, 34, 58, 136, 140, 155a, 178).

[b] Genes are listed in numerical order for essential genes and in alphabetical order for lettered essential genes and nonessential genes. Bracketed symbols indicate that the gene designation and the corresponding defective phenotype refer to only a single mutation or to a region that cannot yet be identified as a gene.

[c] Numbers indicate approximate positions of the promoter-proximal end of the gene to the nearest whole number of kilobase pairs on the scale in Fig. 1. Bracketed numbers indicate uncertain map position; unmapped genes or mutations are indicated by (–).

[d] See Table 2 for explanations of symbols and additional references. –, Mutations are of unknown type.

[e] Assignments of early (E) and quasilate (Q) promoters are based primarily on references 145 and 146 or references given in column 5. Assignments of late (L) promoters are from several studies that have identified products of assembly genes; see references in column 5. Brackets indicate presumed promoter classes based on map position and/or defective phenotype only. E, Q indicates that gene has both an early and a quasilate promoter.

[f] Name or function of gene product is given if known. If not, apparent gene product function based on defective phenotype is listed. For unidentified gene products whose function is unclear, the defective phenotype resulting from mutations in the gene is indicated as "*d*:".

[g] All figures given represent polypeptide molecular weights estimated from calibrated polyacrylamide gel electropherograms. Values differing by up to 20% have been published for some of these polypeptides; where available, we have listed values obtained in the laboratories of L. Gold and C. Yegian at the University of Colorado, Boulder, since these molecular weights represent the largest sets determined under identical conditions and therefore seem most likely to be internally consistent.

[h] C. Castillo, C-L. Hsiao, P. Coon, and L. W. Black, to be published.

[i] S. Brown, unpublished experiments with *am* mutants.

[j] R. Herrmann, unpublished data, cited in reference 160.

[k] L. Snyder, to be published.

[l] F. R. Frankel, personal communication.

[m] A. Rodriguez, unpublished data, cited in reference 92.

[n] It is not known which molecular weight corresponds to *nrd*A and which to *nrd*B (13, 14).

[o] A. R. Depew and N. R. Cozzarelli, unpublished observations cited in reference 92.

[p] Several recent observations suggest that *r*III may be located slightly clockwise from gene 31, rather than counterclockwise as shown in Fig. 1 (77, 176a; L. Black, personal communication[h]; H. R. Revel, unpublished observations).

[q] T. Homyk, Jr., A. Rodriguez, and J. Weil, unpublished observations cited in reference 92.

[r] See Fig. 2.

[s] J. Abelson, unpublished data, cited in reference 155a and personal communication.

Current knowledge of gene locations and sizes is summarized in the linkage map of Fig. 1. The map has been modified and updated from a previous version (214), and was constructed as follows. The map length of 166 kb was assumed, and a zero point was arbitrarily placed at the divide between the *r*IIA and *r*IIB cistrons, a point which has been well defined genetically (5, 11) and physically by deletion heteroduplex mapping (106). Loci whose physical distances from the zero point have been determined by heteroduplex mapping of deletion mutations then were positioned. These points are indicated by heavy radial lines on the inside of the map circle. As many additional genes as possible were placed relative to these points based on the positions of markers estimated by the Mosig method (140). Where necessary these positions have been changed to accommodate minimum gene sizes derived from estimated molecular weights of identified polypeptide gene products. Positions of the remaining genes relative to the physically mapped loci were estimated from recombination frequencies using the mapping function (185).

Genes of unknown size are represented by radial lines. Genes whose polypeptide products have been identified are represented by stippled bars indicating minimum gene length based on polypeptide molecular weights estimated from sodium dodecyl sulfate-gel electrophoresis. The mean value of known polypeptide gene product sizes in T4 is 43,000 daltons, corresponding to a gene size of about 1.2 kb. An expansion of the transfer ribonucleic acid (tRNA) region is shown in Fig. 2.

Regions of the genome in which nonlethal deletions have been demonstrated are indicated by dashed circular segments inside the map circle. Such deletions now have been obtained in five regions of the genome previously designated as largely "silent": the regions flanked by essential genes 39 and 56 (92), 49 and *e* (33, 211), *e* and 57 (211), 63 and 32 (92), and 52 and 60 (48). The dashed segments represent maximum lengths of nonessential sequences as defined by overlapping deletions. Study of these deletion mutations has led to more precise physical mapping of the genome, as well as definition of many new nonessential genes.

Regions of homology and nonhomology between T4 and the closely related phage T2, as determined by electron microscopy of hybrid duplex DNA molecules, are shown on the innermost circle in Fig. 1 (106). Deletion loops are indicated as sectors, and substitution loops are represented by truncated sectors whose inner and outer arc lengths indicate the lengths of the

TABLE 2. *Phenotypic classes of T4 mutants*

Symbol	Phenotype/selection method	Genes or loci of occurrence	References[a]
ac	Acridine resistant	*ac*, *ama*, *rc*, *rs*, 17	
am	Amber; UAG nonsense mutation, conditionally defective: mutant gene functions in UAG su^+ host strains; does not function in su^- host strains	All known essential genes and some nonessential genes	67
BrdU	Bromo-deoxyuridine resistant	*tk*	
c	Cofactor requirement	34	59, 111
CBW	Carbowax resistant	18, 36, *wac*	70
cs	cold sensitive, conditionally lethal: grows at 37°C but not at 17°C	Some essential genes	166a
del	*deletion*		
	del(*r*II): nonreverting *r*II mutant	163–3kb	10, 152
	del(39–56): tandem *r*II duplication with compensating deletion in gene 39–56 region	6–16 kb	92
	del(tk): *tk* mutant selected for bromodeoxyuridine resistance and *r* plaque morphology	50–61 kb	33
	del(far): selected for folate analogue resistance and *r* plaque morphology	48–64 kb	33
	del(e): nonreverting lysozyme mutant	60–73 kb	190, 211
	del(psu_b^-): found among psu_b^- derivatives selected from psu_b^+	67–73 kb	211
	del(63-32): tandem *r*II duplication with compensating deletion in gene 63-32 region	136–144 kb	92
	del(sa): selected for acridine resistance and suppression of *pse*T	162–165 kb	48
eph	Electrophoretic variant; mutation causes change in electrophoretic mobility of mature phage particles		36
ex	Mutant shows decreased exclusion of phage T2 in mixed infections	5 sites adjacent to genes 32, 42, 56, 60, *αgt*	153
far	Folate analogue resistant	*frd*, *mot*, 48–64 kb	33
gor	Grows on rif^R bacteria restrictive for T4 wild type	*βgt* (*gor*-1), *gor*-2	183
h	Altered host range	6, 7, 8, 10, 37	6, 44
hus	Hydroxyurea sensitive	*dar*, *den*A, *dex*A, *sud*, 45, 49, 5–10 kb, 157–160 kb	78
mi	Minute plaques	23, 63, other genes	
oc	Ochre; UAA nonsense mutation, conditionally defective: mutant gene functions in UAA su^+ host strains; does not function in su^- host strains	Several essential genes	23b
op	Opal; UGA nonsense mutation, conditionally defective: mutant gene functions in UGA su^+ host strains; does not function in su^- host strains	Several essential genes	23b
os	Osmotic shock resistant	24	100
pla	Fails to grow (make plaques) on specific *E. coli* host strains:		
	pla(λ): fails to grow on (λ) lysogens	*r*IIA, *r*IIB	
	pla196: fails to grow on CT196	*pse*T	
	pla262: fails to grow on CT262	164 kb	
	pla439: fails to grow on CT439	Some tRNA	81, 208
	pla447: fails to grow on CT447	*ndd*	
	pla596: fails to grow on CT596	*ip*I	21
	plaCTr5X: fails to grow on CTr5X[b]	6 kb, *pse*T	
	plaAR-8: fails to grow on AR-8	*cef*	
psu	Phage-coded suppressor of nonsense mutations	$tRNA^{gln}$, $tRNA^{ser}$, psu^+SB	
r	Rapid lysis, characteristic large, clear plaques	*r*I, *r*II, *r*III, *r*IV, *r*V, *st*I, *st*III	
ts	Temperature sensitive; conditionally defective: mutant gene product functions at 25°C; does not function at 42°C	Many essential genes	67
uvs	Sensitive to UV light	*den*V, *uvs*W, *uvs*X, *y*	

[a] Additional references may be found in Table 1 under the appropriate gene entries.
[b] Strain CTr5X is a derivative of CT196 (47).

TABLE 3. *T4-induced enzymes*

Enzyme	Gene[a]	Reference[a]
DNA adenine methylase	*dam*	
DNA-dependent adenosine triphosphatase	*dda*	
DNA endonuclease I[b]	–	2
DNA endonuclease II	*den*A	
DNA endonuclease III	–	165
DNA endonuclease IV	*den*B	
DNA endonuclease V	*den*V	
DNA endonuclease VI	–	102
DNA exonuclease A	*dex*A	
DNA exonuclease B	–	73, 148
DNA ligase	30	
DNA polymerase-exonuclease	43	
Deoxycytidylate deaminase	*cd*[c]	
Deoxycytidylate hydroxymethylase	42	
Deoxycytidine-deoxyuridine di- and triphosphatase	56	
Deoxyribonucleotide kinase	1	
Deoxyribonucleotide-3'-phosphatase	*pse*T[c]	
Deoxyribonucleotide-5'-phosphatase	*pse*F[c]	
Dihydrofolate reductase	*frd*	
Endolysin (see lysozyme)		
α-Glucosyl transferase	*αgt*	
β-Glucosyl transferase	*βgt*	
Ligase (see DNA and RNA ligases)		
Lysozyme (endolysin)	*e*	
Phospholipase	*t*[c]	
Protease, specific for capsid protein cleavage in assembly	21	
Ribonucleotide reductase subunit	*nrd*A	
Ribonucleotide reductase subunit	*nrd*B	
RNA ligase	–	43, 172
Thioredoxin	*ndr*C	
Thymidine kinase	*tk*	
Thymidylate synthetase	*td*	

[a] The symbol (–) in the Gene column indicates that a gene for the enzyme has not yet been mutationally identified. References are given only for these enzymes. References for enzymes coded by identified genes may be found in Table 1.

[b] Designated simply as DNA endonuclease by the discoverers (2).

[c] Enzyme activity is missing from cells infected with mutants defective in the indicated gene, but the enzyme has not been shown directly to be the gene product.

nonhomologous sequences in the two phages. Genetic studies have shown that T2 and T4 are almost completely homologous with regard to the locations of essential genes (164). Therefore, to the extent that T4 genes are correctly placed on the genetic map circle in Fig. 1, the homology map can be interpreted to indicate the genes or regions in which divergence has taken place between T2 and T4. In theory it should be possible to use this information for physical mapping of additional genetic markers, by matching regions of reduced recombination frequency in T2–T4 crosses with substitution loops on the homology map. This approach was exploited by Beckendorf to position the large substitution loop at 155 kb relative to markers in genes 37 and 38 (7).

GENE FUNCTIONS AND GENOME ORGANIZATION

The promoters of T4 genes fall into three categories. These categories are recognized at different times during the infectious cycle, and have been designated early (E), quasilate (Q) and late (L) (145, 146). Directions of transcription, now known for 38 genes from genetic evidence, are indicated in Fig. 1 by arrows inside the map circle. Transcription directions for the single genes 43, 23, 32, *r*IIA, and *r*IIB have been determined from the relative sizes of polypeptide fragments corresponding to *am* mutations of known map order. Arrows that extend over more than one gene indicate cotranscription; their directions and extents have been inferred from polar effects of nonsense mutations and UV irradiation on expression of neighboring genes (90, 184, 198). Evidence from messenger RNA hybridzation experiments using separated single strands of T4 DNA indicates that probably all early and quasilate genes are transcribed in a counter clockwise direction on the map as represented in Fig. 1, whereas late genes are transcribed in a clockwise direction (80). In general, early and quasilate genes are segregated from late genes in the genome. "Switch" regions, where transcription changes from one direction to another, are presumed to occur at only four points on the map, at approximately 75 kb (between genes 1 and 2), 121 kb (between genes 54 and 30), 147 kb (between genes 33 and 34), and 159 kb (between genes *t* and 52). Thus, the genome is divided into at least two regions of early and quasilate transcription, including a total of about 88 identified genes, and two regions of late transcription, including a total of about 46 identified genes. The promoter classes of individual genes, where known, are indicated in Table 1. T4 transcriptional controls and their significance have been reviewed recently by Rabussay and Geiduschek (155a).

Broad classes of gene functions are indicated in Fig. 1 outside the map circle. More detailed descriptions of functions and defective phenotypes are listed in Tables 1 to 3. A breakdown

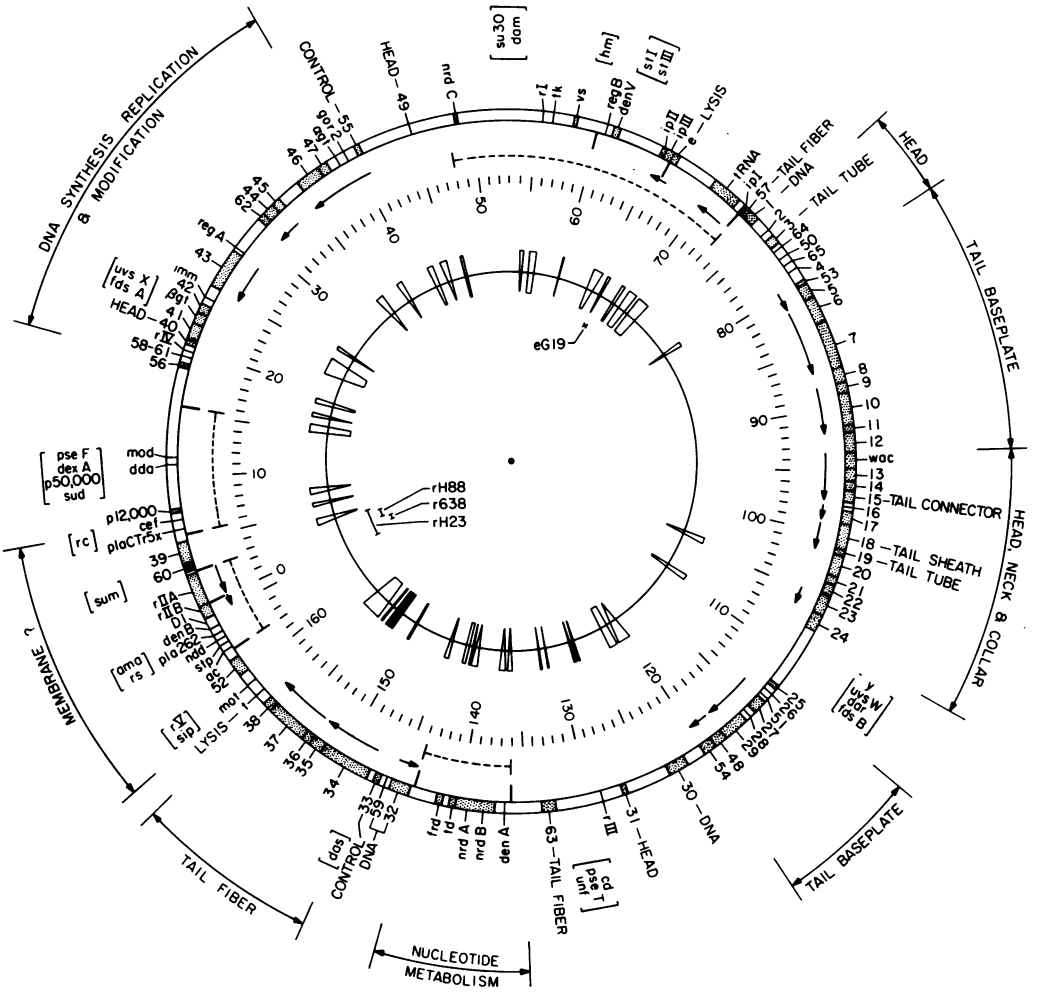

FIG. 1. *Map of the bacteriophage T4 genome. Construction of the map is described in the text. The circular numerical scale indicates physical distances in kilobase pairs (kb) from an arbitrary zero point. The innermost circle is a heteroduplex map showing regions of nonhomology betwee T4 and the related phage T2 (106). Labeled arcs inside this circle show the positions of the deletions used as reference points. The map circle outside of the numerical scale indicates the locations of T4 genes, positioned as described in the text. Heavy radial lines inside the map circle indicate positions determined by heteroduplex mapping of deletion mutations in the electron microscope. Arrows indicate transcription direction; those that extend over more than one gene indicate cotranscription. Dashed circular segments indicate maximum lengths of nonessential sequences as defined by overlapping nonlethal deletion mutations. (The left end of the region between 48 and 73 kb has been positioned by genetic mapping only [33]; all other end points have been located physically by heteroduplex mapping.) Stippled bars on the map circle represent the minimum lengths of genes whose polypeptide products have been identified and sized by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. The size of gene 60 (cross-hatched bars) has been estimated from extensive intracistronic mapping (141). Radial lines on the map circle indicate positions of genes whose polypeptide products have not been identified. Gene names are given on the outside of the map circle. Gene names in brackets represent loci whose positions and/or map order are known only approximately. The outermost circle indicates the clustering of functionally related genes into broad classes. Smaller radial labels adjacent to gene names indicate functions that differ from those of the surrounding genes in a cluster. Additional features of the map are described in the text.*

of gene functions into specific classes is shown in Fig. 3. These functions can be divided conveniently into two major categories (58), designated *cell metabolism* and *phage particle assembly*. Metabolic functions, almost all of which are controlled by genes with early or quasilate promoters, include DNA metabolism, programming (transcription and translation),

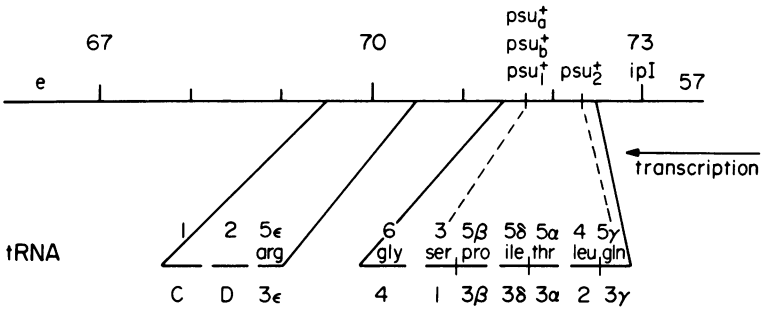

FIG. 2. *Expansion of the T4 tRNA gene region, 69.5 to 72.5 kb. Modified from Abelson et al. (1 and personal communication) and from Rabussey and Geiduschek (155a). Genes are identified by tRNA function, where known, as well as by the numbering systems employed by Abelson et al. (1; upper designations) and by Guthrie et al. (82; lower designations) for the corresponding tRNA bands on polyacrylamide gel electropherograms. The order of the tRNA genes in the right hand cluster has been derived from an analysis of deletion mutants. Recent restriction enzyme sequence studies suggest that part of this order may be permuted and that the true linear arrangement of the tRNA genes is (in the order of transcription) gln leu gly pro ser, thr ile. This new sequence is compatible with the deletion mutant analysis (J. Abelson, personal communication). The stable low-molecular-weight RNA transcripts corresponding to bands 1(C) and 2(D) are of unknown function. psu_1^+ and psu_a^+ are mutations that convert $tRNA^{ser}$ to a suppressor of amber mutations. psu_b^+ converts $tRNA^{ser}$ to a suppressor of ochre mutations. psu_2^+ converts $tRNA^{gln}$ to a suppressor of ochre mutations. For references see appropriate entries in Table 1.*

and cell maintenance. Most of the assembly functions are controlled by genes with late promoters.

Of the metabolic gene functions, 22 are essential, as defined earlier. Six of these genes code for known proteins required for phage DNA synthesis (67), either as enzymes of nucleotide metabolism or components of the actual DNA replication and recombination machinery (4, 128, 129). A list of the known T4-induced enzymes is given in Table 3. The products of two genes, 33 and 55, are required for the control of transcription, and the products of two more, *e* and *t*, bring about cell lysis at the end of the infectious cycle (for references, see Table 1). However, there remain 12 essential metabolic genes whose functions are unknown. The protein products of 7 of these are assumed to play roles in DNA replication, since mutational defects in them lead to absence of early arrest of DNA synthesis (shown in parentheses in Fig. 3). Defects in genes 39, 52, 58 to 61, and 60 delay the onset of DNA synthesis at 37°C, but prevent DNA synthesis entirely at 25°C (141). The functions of these genes remain intriguingly obscure.

Most the known nonessential genes fall into the metabolic category. The general function of many nonessential genes apparently is to augment the phage burst size, for example, by providing more abundant substrates for DNA replication (see references 128 and 129, and references in Table 1). Others of these genes equip T4 to deal with differing host intracellular environments, for example, by providing supplementary tRNA species, and also perhaps by modifying the host cell membrane. Again, however, the functions of 38 out of the total of about 63 metabolic nonessential genes are not known (shown in parentheses or listed at the extreme right in Fig. 3). The large number of apparently nonessential genes involved in DNA synthesis suggests the possibility of gene redundancy for recombination and repair in T4 as has been found in *E. coli* (37). Conceivably the products of some of these T4 genes perform *essential* functions that can be carried out by alternative pathways under separate gene control (57).

There are 55 identified assembly gene functions, all but 10 of which are essential (29, 58, 63, 67, 171, 212, 219, 221, and references in Table 1). Of the corresponding gene products, 36 are known to be structural proteins of the phage, whereas 7, indicated by asterisks in Fig. 3, appear to be nonstructural accessory proteins that somehow promote or direct assembly (212, 213). The remaining 12 assembly gene products are known to be required at a certain stage in the assembly process (219), but it is not known whether their roles are structural or accessory. The genes that code for these proteins are shown in parentheses in Fig. 3. It can be seen that the gene functions listed under "capsid completion" and "DNA packaging" represent intriguing classes whose roles are almost completely obscure. Recent evidence on gene 2 function suggests the possibility that some of the genes in the "capsid completion" group are not true assembly genes, in that they code for internal proteins that are injected with the phage DNA and serve their principal functions very early in infection (174, 175).

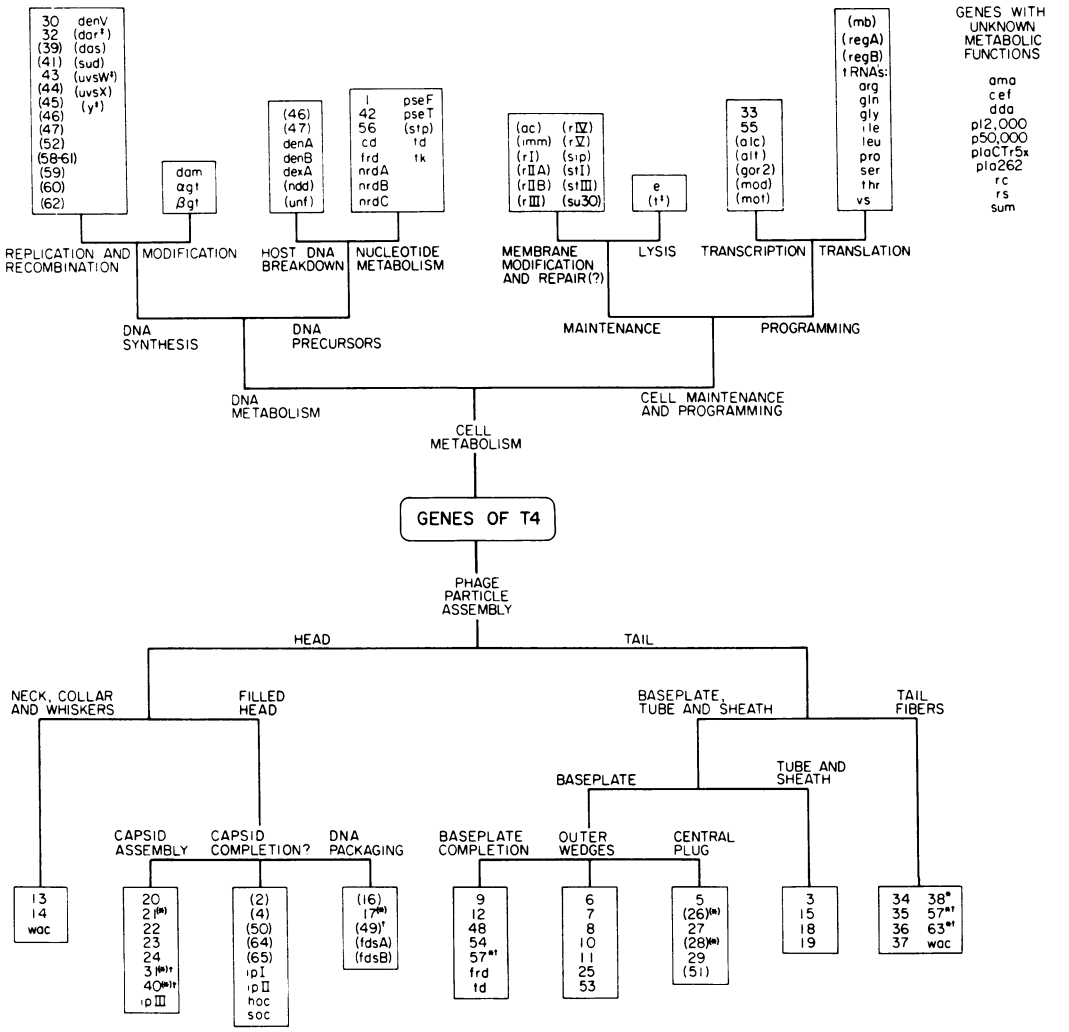

FIG. 3. *Functional classification of T4 genes. Genes whose functions are at least generally known are listed in boxes representing different functional categories. In the upper half of the chart, genes listed without parentheses are those whose protein products have been identified with specific enzymatic functions. Genes listed in parentheses are those whose functions are known generally but not specifically. Genes defined by suppressors only (e.g., dar, das, sud, etc.) generally are assumed to belong to the same functional category as the genes on which these suppressors act. Exceptions are some ligase (gene 30) defect suppressors, which appear to act by altering cell membrane properties (98). The genes listed at the far right are not known to fall into any of the functional categories shown. In the lower half of the chart, genes listed without parentheses are those whose protein products have been identified and shown to be either structural components of the phage or nonstructural accessory proteins in assembly (indicated by *). Genes listed in parentheses are those whose protein products have not been identified, so that the structural or nonstructural nature of their roles is unclear. A total of 138 genes is shown in the figure. Some genes with dual functions are listed under two functional categories. Not included are bracketed gene designations from Table 1, as well as five genes, not yet mutationally identified, that code for known T4-induced enzymes of uncertain function (see Table 3). Symbols: §, expressed early, but gene product functions later in assembly; ‡, probably expressed late, based on map position, but gene product apparently functions in cell metabolism; * gene product performs a nonstructural accessory function in assembly; (*), gene product is probably nonstructural.*

Comparison of Fig. 3 with Fig. 1 indicates that, in general, T4 genes exhibit considerable clustering according to function. However, it is noteworthy that at least one of the genes in every functional class is located outside of the major cluster, often in association with another cluster of different but related functions. Stahl and Murray (186) have discussed the possibility that clustering is selected for because it minimizes recombination between genes for pro-

teins that must interact structurally, and thereby decreases the frequency of nonviable hybrids in interstrain matings. King and Laemmli (109) have postulated that the observed departures from clustering may be important in regulating production of assembly components, by allowing key proteins of two different components to be translated from the same messenger RNA, thereby ensuring synthesis in fixed relative amounts. Alternatively, the observed gene organization may be primarily a reflection of evolutionary history. For example, baseplate genes are segregated exclusively into two clusters, whereas genes for the tail sheath, tube, and connector proteins are interspersed among head genes. Conceivably, an ancestor of T4 may have had only a baseplate attached directly to the head as in present-day simpler phages such as T3 and T7, and genes for the sheath, tube, and connector may have arisen from head genes by duplication and evolution. This notion could be tested by determining and comparing the amino acid sequences of appropriate proteins for residual homology.

The homology map suggests that, in general, regions of essential genes are less evolutionarily divergent than regions of nonessential genes and that, in particular, the genes coding for structural proteins of the phage head and tail are highly conserved.

The mean polypeptide molecular weight of the 31 identified metabolic gene products, which are primarily cytoplasmic enzymes, is 40,000, corresponding to a gene size of about 1.1 kb (Table 4). The mean molecular weight of the 41 identified assembly gene products, which are primarily phage structural proteins, is 45,000, corresponding to a gene size of 1.2 kb. If we assume that these known gene sizes are representative for their respective classes (Table 4) we can estimate that about 15% of the phage genome is used to code for essential metabolic functions, 39% for nonessential metabolic functions, and 36% for assembly of the phage particle. How much of the total coding capacity of the phage DNA can be accounted for by mutationally identified genes? An accurate answer cannot be given without additional information on gene sizes. However, the assumptions just stated lead to an estimate that known genes can account for a combined sequence length of about 150 kb, or about 90% of the coding capacity of the genome. Genetic saturation of the T4 map is nearly accomplished.

TABLE 4. *Characteristics of T4 genes in various functional classes*

Gene functional class[a]	Mean of known polypeptide gene product molecular weights[b]	Approximate % of genome[c]
Metabolic genes, essential (22)	41,000 (17)	15
Metabolic genes, nonessential (60)[d]	39,000 (14)[d]	39[e]
Particle assembly, genes for structural proteins (34)[f]	48,000 (34)	27
Particle assembly, genes for nonstructural proteins and unknown functions (19)	30,000 (7)[g]	9.4
Total		90

[a] Number of genes in each class is listed in parentheses (total: 135 genes). For the purposes of these calculations, only unbracketed genes from Table 1 were counted, in addition to five genes, not yet mutationally identified, that code for known T4-induced enzymes (see Table 3).

[b] Number of molecular weight values averaged to obtain each mean is indicated in parentheses (total: 72 known polypeptide molecular weights).

[c] Figures were obtained by multiplying the mean molecular-weight value for each class by the total number of genes in the class, converting the resulting aggregate polypeptide molecular weight to minimum DNA sequence length in kilobase pairs (assuming an average amino acid residue weight of 110), dividing by the genome size in kilobase pairs, and multiplying by 100. Total accounted for: 90% of genome. See text for further discussion.

[d] Does not include the eight tRNA genes; includes *frd*, *td*, and mutationally unidentified genes for five T4-induced enzymes (see Table 3) assumed to be nonessential.

[e] tRNA genes added to total calculated from polypeptide molecular weights.

[f] Does not include *frd*, *td*.

[g] Using a molecular weight of 18,000 for the product of gene 57.

ACKNOWLEDGMENTS

We thank Peter Geiduschek, Gisela Mosig, and D. Rabussay for providing us with manuscripts of their forthcoming reviews (140, 155a) before publication. We also are grateful to the many colleagues who communicated suggestions and unpublished information to us.

Work from our laboratory was supported by Public Health Service grant AI-09238 from the National Institute of Allergy and Infectious Diseases.

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