

Derivations and Genotypes of Some Mutant Derivatives of *Escherichia coli* K-12

BARBARA J. BACHMANN

133

INTRODUCTION

Escherichia coli K-12 was isolated in the fall of 1922 from the stool of a convalescent diphtheria patient in Palo Alto, California, by Blair (93). In 1925, the culture was deposited in the strain collection of the Department of Bacteriology at Stanford University, where it was given the designation "K-12." Strain K-12 gave positive results in the standard tests being used for the identification of *E. coli* and was used for many years in the teaching laboratories of the department as a typical example of *E. coli*. This culture is still maintained in the department collection in stab cultures of nutrient agar, which are transferred from time to time (B. A. D. Stocker, personal communication).

In the early 1940s, E. L. Tatum, then at Stanford, asked the bacteriology department for some bacteria to test for possible use in his studies of biochemical genetics. By great good luck he was given, along with cultures of other species of bacteria, *E. coli* K-12. Tatum decided to use K-12 in his studies; strain K-12 is prototrophic, easy to cultivate in a defined medium, and has a short generation time. The use of this bacterium permitted easy study of very large populations and thus the accurate analysis of very rare events, presenting a great advantage in this respect over the plants, animals, and fungi previously used in genetic studies. In 1946, Lederberg and Tatum demonstrated sexual recombination in strain K-12 (99, 100). It was found later that the ability to undergo sexual recombination is rare in strains of *E. coli* isolated from nature (95, 123, 124).

In 1944, Tatum and Gray reported the isolation of the first auxotrophic mutants of strain K-12 (56, 149). These same mutants were used later by Lederberg and Tatum in their early studies on sexual recombination (90, 98, 99, 150). Since that time, many thousands of mutant derivatives of strain K-12 have been isolated in laboratories around the world. However, most of the mutant derivatives in use today are derivatives of mutants that were obtained in the first studies in Tatum's laboratory by using irradiation with X rays and, later, UV irradiation.

The chromosome of a mutant strain such as AB1157, widely used as an ancestral stock, has been treated with X rays three times, with UV nine times, and with nitrogen mustard once. Taking into consideration, in addition, the possible effects of selective pressures exerted during growth in laboratory media for over 70 years and the possibility of spontaneous random mutations and rearrangements within the chromosome, it does not seem surprising that "new" mutations are being discovered in "old" mutants as advancing knowledge permits the recognition of more subtle defects.

Already in the 1950s, serological studies showed that (after 30 years of cultivation in the laboratory) strain K-12 did not have the typical antigenic structure of newly isolated wild-type strains, having virtually lost the K and O antigens (123, 125). Still later, in the 1970s, the strain was shown to be ineffective in colonizing the human gut (143).

A recent extensive study revealed the degree of insertion sequence-related variation found in a mutant derivative of K-12 stored in stab cultures for more than 30 years (116). Another extensive study explored sequence divergence among 26 of the early derivatives of strain K-12 included in the pedigrees herein (17). From studies such as these, inferences can be drawn regarding the "original" DNA sequence of strain K-12.

The culture of K-12 wild type which was used in the Tatum laboratory during the isolation of the early mutants and the studies on recombination was lost when the Lederberg laboratory moved to the University of Wisconsin in 1951 and was replaced by another subculture of K-12 from the collection of the bacteriology department at Stanford. When M. L. Morse joined the Lederberg laboratory in that same year, he brought with him a subculture of K-12 which he had obtained from the bacteriology department at Stanford in 1948. Both of these cultures were designated WG1, with no distinction being made between them, and were used as K-12 wild type in studies in the Wisconsin laboratory thereafter (J. Lederberg and M. L. Morse, personal communications).

However, the original culture of K-12 used in the Tatum laboratory had been taken to Europe by L. L. Cavalli-Sforza, who used this culture in his own work and gave a subculture to W. Hayes in England (L. L. Cavalli-Sforza, personal communication). This culture was used by Hayes in his studies on microbial genetics and was given the designation EMG2 by R. C. Clowes and Hayes in their widely used laboratory manual of 1968, *Experiments in Microbial Genetics*. The culture was widely distributed for use with this manual.

All known cultures of K-12 wild type have exhibited a rough phenotype. Liu and Reeves (102) recently identified mutations that account for the rough phenotype of two of the wild-type lines. They demonstrated that a culture of WG1 carries a deletion at the upstream end of the *rfb* gene cluster, $\Delta rfb-51$, and that a culture of EMG2 carries an IS5 insertion at the downstream end of *rfb*, which they designated *rfb-50*. When a plasmid carrying part of the *rfb* region of strain WG1 was introduced into EMG2, the complemented strain produced O antigen of type O16.

The charts in this chapter consist of diagrams showing the derivation and genotypes of the most widely used ancestral stocks and of some later strains that are in particularly wide use in experimental work today. Most of these charts are corrected and updated versions of charts published in 1987 (7).

SOURCES OF THE DATA

Strain pedigrees are presented in Charts 1 through 14. The documentation for these diagrams is given in Table 1 under the strain designations, which are listed in alphanumerical order.

The ultimate sources of the data were, in most cases, the records of the laboratories in which the strains were isolated. These major unpublished sources are given as documentation in Table 1, where they are referred to by capital letters as follows: A, strain notebooks and cards in the laboratory of J. Lederberg; B, strain cards in the laboratory of F. Jacob; C, strain cards in the laboratory of E. A. Adelberg; D, strain notebooks of A. L. Taylor; E, strain records of M. L. Morse; F, strain list of P. Howard-Flanders; G, strain cards of S. Brenner.

In other cases, considerable help in determining strain derivations was given in extensive personal communications, referred to by lowercase letters, from the following individuals: a, R. Appleyard; b, W. Arber; c, A. Campbell; d, A. J. Clark; e, R. C. Clowes; f, B. D. Davis; g, A. Garen; h, D. Hanahan; i, W. Hayes; j, K. B. Low; l, W. Maas; m, M. Meselson; n, P. Reeves; o, P. Treffers; p, N. Willetts; q, E. Wollman; r, C. Yanofsky.

TABLE 1 *E. coli* strains

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
<i>E. coli</i> K-12		56,93,149,150		WG1; see text
(λ)		85,86,88,89		
F ⁺		26,96		
AB250	C			See PA100
AB253	C			See W208 S ^f
AB254	C			See W208 S ^f F ⁺
AB257	C			See Hfr 4000; <i>not</i> Cavalli Hfr!
AB258	C			See W583
AB159	C			Hayes Hfr Thi ⁻ λ^- ; see Hfr 3000
AB262	C			See PA100 S ^f Pro ⁻ ; JW1
AB264	C			<i>E. coli</i> K-12 "wild type"; Ara ⁻ due to the presence of phage Mu 1 in <i>ara</i>
AB265	C			See W2915
AB266	C			See W2961
AB280	C			"58-161," actually W6
AB281	C			Hayes Hfr Thi ⁻ λ^- ; see Hfr 3000
AB284	C	151	2	W208 S ^f F ⁺
AB311	C,D	151	2	AT11
AB312	C,D	151	2,9	AT12
AB313	C,D	151	2	AT13
AB314	C,D		9	AT14
AB352	C,D		9	AT52
AB673	C			See P10 Hfr (J4)
AB674	C			See Reeves 1 Hfr
AB712	C		2	
AB781	C			See W677
AB808	C			See 3300
AB815	C			See JC12
AB856	C			See JC158
AB862	C			See JC182, "double male"
AB869	C			See JC355
AB1103	C		2	
AB1115	C		2	
AB1133	C		2	
AB1157	C	4,72	2	
AB1621	C	2	2	
AB1859	C		2	
AB1884	F	71	4	
AB1885	F	71	4	
AB1886	F	70-72	4	
AB1896	F		4	
AB1899	F	70,72	4	
AB2301	F			See 3300
AB2457	F		4	
AB2462	F	73	4	
AB2463	F	70,71,73	4	
AB2470	F	44,70	4	
AB2474	F	70	4	
AB2487	F	164	4	
AB2495	F	44	4	
AB2497	F	44,71	4	
AB2500	F	71	4	
AB3004	C			See C600 (CR34)
AB3022	F	44	4	
AB3058	F		4	
AB3591	C			See CR63
AB3642	C			See CR34 Thy ⁻
AT11	D			See AB311
AT12	D			See AB312
AT13	D			See AB313
AT14	D			See AB314
AT52	D			See AB352
AT705	D		6	
AT715	D		6	
AT716	D		6	

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
B1–B12				See Broda 1–12 Hfr's
Broda 1–Broda 12 Hfr's		18,64,65,111	5	
C4	g		12	
C6		5	2	
C60		5	2	
C90	g	48	12	
C600	a	5,16,58,128,139	2,14	CR34 standard λ indicator
CA1	G		10	
CA79	G		10	
CA85	G		10	
CA154	G	16	10	
CA161	G	16	10	
CA165	G	16	10	
CA167	G	16	10	
CA244	G	16	10	
CA265	G	16	10	
CA266	G		10	
CA267	G	17	10	
CA273	G		10	
CA274	G		10	
CA275	G		10	
CA374	G		10	
Cavalli Hfr		28	5,11,12	HfrC, W1895, Hfr ₁ (Lederberg), not AB257 See C600
CR34	a	5,43,139	2	
CR34 Thy ⁻		121,122	2	
CR34 Thy ⁻ S ^r		159	2	
CR63		6,17,43,171	8	λ host range indicator
<i>supD60</i>		17,139		
CR67		6	8	λ^{vir} indicator
CS2		141	5	
CS11		140	2	
CS19		140	1	
CS100		141	2	
CS101		141	5,12	
DH1	h	60	11	
DH2	h	60	11	
E15	g	61,83	12	
G240	g	29	12	
G240R4	g	29	12	
H12	g	46,49,160	12	
H12R7A	g	46,49	12	
H12R8A	g	49,161	12	
Hayes Hfr	i,A,B	63	5,6	HfrH, W2323, Hfr ₂ Lederberg, Hfr ₂ or Hfr ₂ (Paris)
Hayes Hfr Thi ⁻	i,q,A,B	76	6	Hfr ₄ or Hfr ₄ (Paris)
Hayes Hfr Thi ⁻ λ^- (Paris)	i,q,A,B,C	76	6,7,10	HfrH, AB259 HfrC, 3000, and HfrH (Paris)
Hayes Hfr Thi ⁻ λ^- (Wisconsin)	A		6	See W3634
Hfr ₁ (Wisconsin)	A			See Cavalli Hfr
Hfr ₂ (Wisconsin)	A			See Hayes Hfr
Hfr ₃ (Wisconsin)	A			See W2924
Hfr ₄ (Wisconsin)	A			See W2945
Hfr ₄ (Paris)	B	76		See Hayes Hfr Thi ⁻
Hfr ₆ (Wisconsin)	A			See W3807
Hfr ₈ (Wisconsin)	A			See W3208
Hfr ₁₃ (Wisconsin)	A			See W3213
Hfr ₁₅ (Wisconsin)	A			See W3201
Hfr7–4	1	40	9	MA1048
Hfr 3000	e,k,r,A,C		6,7,10	HfrH Thi ⁻ λ^- ; "HfrC" (Paris), AB259
Hfr 3000 U118	B	118	6	
Hfr 3000 U169	B	25	6	
Hfr 3000 U281	B	118	6	
Hfr 3000 X74	B	13	6	
Hfr 3000 X90	B	13,118	6	
Hfr 3000 X111	B	13	6	
Hfr 3000 YA14	B	17,139	6,10	

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
Hfr 3000 YA287	B	12	6	
Hfr 3000 YA289	B	12	6	
J1 F ⁺	f	34	8	
J4 F ⁺	f	34	8	
J4-5 F ⁺	f	34	8	J45
J5 F ⁺	f	34	8	
J5-3 F ⁺	f	34	8	J53
J5-10 F ⁺	f	34	8	J510
J6-2 F ⁺	f	34	8	J62
J1 Hfr	B			See P1
J2 Hfr	B			See P4X
J3 Hfr	B			See P3, 4000
J4 Hfr	B			See P10
J5 Hfr	B			See P72
J6 Hfr	B			See P13
J7 Hfr	B			See P808
J45 F ⁺				See J4-5
J53 F ⁺		51		See J5-3
J62 F ⁺				See J6-2
J510 F ⁺				See J5-10
JC12	d	30	9	AB815
JC158	d	30	9	AB856
JC182	d	30,32	9	AB862, "double male"
JC355	d		9	AB869
JC411	d	32,33	9	
JC500	d		9	
JC590	d	32	9	
JC1552	d		9	
JC1553	d	31,33	9	
JC1554	d	31,33	9	
JC1557	d	20	9	
JC1569	d	20	9	
JC2915	d	163	4	
JC2926	d		4	
JC4474	d		6	
JC4490	d		6	
JC5029	d	31,164	6	
JC5088	d	31,164	6	
JC5401	p		6	
JC5408	p	163	4	
JC5412	p	31,163,165	6	
JC5421	p	163	4,6	
JC5422	p	163	4	
JC5426	p	163,165	6	
JC5474	p	163	4	
JC5489	p	10	4	
JC5491	p	163	6	
JC5495	p	163	6	
JC5519	p	163	4	
JC5544	p	163	6	
JC5547	p	163	6	
JC5743	d	10	4	
JC7623	d	69,103	4	
JC7901	d	69	4	
JC8012	d	69	4	
JC8403	d	69	4	
JC8411	d	69	4	
JC8471	d	69	4	
JC9239	d	69	4	
JC9366	d		4	
JC9387	d		4	
JW1	B			See PA100 S ⁺ Pro ⁻
K10		83	5,12	
K-12, wild type				See first entry, above
K802			14	
K803			14	
KL14	j	107	6	
KL16	j	105,107	6	
KL16-99	j	105	6,11	
KL20	j		6	

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
KL25	j	105,107	8	
KL96	j	105	6	
KL98	j	105	8	
KL99	j	107	6	
KL110	j		9	
KL161	j		6	
KL162	j		6	
KL166	j		6	
KL168	j		6	
KL169	j		6	
KL173	j		6	
KL174	j		6	
KL252	j		4	
M series	B		6	Mutants of Hfr 3300 (Paris)
MG1655		59,81	8	
MM28	m	55,127	14	
MM152	m	55	14	152
MM159	m	127	14	159
MM293	m	112	11	
MM294	m	112	11	
MM294A	m	9	11	
MMT	h	60	11	
P1 Hfr	B		5	Hfr P1, Hfr type 1, Hfr J1
P2	B	78,80	5	Hfr P2, Hfr P21
P3	B	75,78,80	5	Hfr P3, Hfr P31, Hfr type 3, 4000, Hfr J3, AB257
P4X	B	75,78,80	5,9	Hfr P4X, Hfr type 2, Hfr J2
P5	B		5	Hfr P5
P6	B		5	Hfr P6
P8	B		5	Hfr P8
P10	B	75,78,80	2	Hfr P10, Hfr type 4, Hfr J4, AB673
P21	B			See P2
P22	B	43	13	
P25	B		13	
P31	B			See P3
P72	B	75,78,80	5	Hfr P72, Hfr type 5
P676	B	76,80	2	
		169		
P678	B	39,80,168,169	2,3,13	
P678 λ^-	B	80,169	3	
P678 F ⁺	B		3	
P678 S ^f	B		3,7	2001w
P697	B			See PA100
P697 S ^f	B			See PA100 S ^f
P802	B		13	
P804	B		13	
P804G	B		13	
P808	B	75,80	13	Hfr P808, Hfr type 7
p3478		38,57	8	
PA100	B		3,9	P697
PA100 S ^f	B		3	P697 S ^f
PA100 S ^f Pro ⁻	B		3	JW1
PA100–PA125	B		3	Series of auxotrophic mutants
PA200	B		3	
PA200–PA266	B		3	Series of auxotrophic mutants
PA200 S ^f	B		3	
PA265	B		9	
PA300–PA394	B		3	Series of auxotrophic mutants
PA351	B		3	
PA601–PA640	B		3	Series of auxotrophic mutants
PA610	B		3	
PA641–PA644	B		3	Series of auxotrophic mutants
R1–R5	n			See Reeves 1–5
R594		24	14	
Ra-1	j	82,104	8	
Ra-2	j	82,104,105	8	
RC709	e	114	8	
RC711	e	114	8	
RC712	e		8	

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
Reeves 1	n	64,65	5	Hfr R1
Reeves 2	n	Reeves, thesis	2	Hfr R2
Reeves 3	n	Reeves, thesis	2	Hfr R3
Reeves 4	n	64,65; Reeves, thesis	5	Hfr R4
Reeves 5	n	Reeves, thesis	13	Hfr R5
S10	g	46,117,148	12	
S26	g	49,50,148	12	
S26R1d	g	49,50	12	
S26R1e	g	49	12	
S26Su6 ⁺	g		12	
T94A	o			See 58–278M
U series	B		6	UV mutants of Hfr 3000
U11	g	46	12	
U11R1d	g	46	2	
W1	A	50,161		
W6	A	54,96	1,5	58–161 <i>bio</i> ⁺ ; AB280
<i>bio</i> ⁺		54,96,135,158		
<i>relA1</i>		3,14,15,147		
W13	A	54	1,5	Y40 <i>bio</i> ⁺
W14	A	54	1,5	Y87 <i>bio</i> ⁺
W45	A		5	
W67	A	93,94,97	5	
W102	A	162	2	
W112	A	87	2	
W133	A	87	2	
W208	A		2,11	
W208 S ^r	C		2	AB253
W416	A		5	
W435		89	5	
W480	A		2	
W516		54,87	5	
W518	A	54,87,89,115	5	
W566	A		2	
W582	A		2	
W583	A	115	2	AB258
W595	A		2	
W620	A		5	
W660	A		2	
W677	A	27,39,92,95,155	2,6,13	AB781
<i>gal-3</i>		115		
W677 F ⁺	i		2,6	
W750	A	87,115	5	
W894	A		2	
W902	A	87,115	2	
W904	A		2	
W922	A		2	
W945	A	158	2	
W1163	A		5	
W1177	A	92,95	2	W677 S ^r
W1210	A	115	5	
W1394	A		2	
W1485	A	65,152	8	
W1485E			8	
W1603	A	89	2	
W1655	A	89	5	
W1655 F ⁻		136	5	
W1655 F ⁺ λ ^r		18	5	
W1872	A	89	8	
W1895	A			Hfr ₂ , Cavalli Hfr
W2207	A		5	
W2252	A		5	
W2323	A			See HfrH; Hfr ₁
W2324	A			See HfrH Thi ⁻ λ ⁺
W2637	A		8	
W2660	A	132	2	
W2817	A	132	2	
W2914	A		2	
W2915	A		2	
W2924	A	130–132	2	Hfr ₃

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
W2945		Richter, thesis	2	Hfr ₃
W2961	A		2	AB266
W3091	E		8	
W3092	E		8	
W3094	E		8	
W3096	E		8	
W3097	E		8	
W3098	A		8	
W3099	A		8	
W3100	A,E		8	
W3101	E		8,14	
W3102	E		8,14	
W3104	E		8	
W3106	E		8	
W3107	E		8	
W3108	A		8	
W3109	A		8	
W3110	A,E	66,81	8	
W3110 Thy ⁻			8	
W3135	A	132	5	
W3201	A		5	See text
W3208	A	67,111,143	5	See text
W3213	A	20,111	5	Hfr ₁₃
W3236	A		5	
W3350		23	14	
W3350A	c		14	
W3634	A		6	HfrH Thi ⁻ λ ⁻ , not Paris strain 3000
W3787	A	36	5	
W3807	A		5	Hfr ₆
W4032	A		5,14	
W4354	A		5	
WA144	b	170	14	
WA605	b	170	14	
WA612	b	170	14	
WA704	b	170	14	
WA802	b	170	14	K802
WA803	b	170	14	K803
"X" series	B	13	6	X-ray mutants of HfrH 3000 (Paris)
XA2	G	16	7	
XA3	G	16	7	
XA20	G	11	7	
XA21	G	11	7	
XA22	G	11	7	
Y10		87,90,100,150	1,2,13	
<i>supE44</i>		43		
Y24		90,99,150	1	
Y40		87,91	1,5	
<i>bio</i> ⁺		54		
Y46		90	1	
Y53		87,90	1,2	
Y64		90	1	
Y70		87,128,129	1,2	
Y80		90	1	
Y86		90	1	
Y87	A	87,90	1,5	
<i>bio</i> ⁺		54		
Y91		90	1	
Y94		90	1	
Y100		90	1	
Y-Mel		133,139,152	1	
<i>supF58</i>	1	139		
"YA" series	B	12	6	N-mustard mutants of Hfr 3000 (Paris)
58		56,91,149,150	1,5,13	W3301
58-161		91,149,150	1,5,13	See also W6
Reversion to <i>bio</i> ⁺		54,96,135,156,15		
Mutation to <i>relA1</i>		3,14,15,145		

Strain designation	Sources of data ^a	Published references	Chart	Synonyms and comments
58-161 F ⁻		62,63,141	5	58-161 F ⁻ Spicer; W6 derivative
58-161 F ⁻ S ^r		62,63	5	58-161 F ⁻ Hayes; W6 derivative
58-161 F ⁻ S ^r Az ^r		62,63	5	W6 derivative
58-161 F ⁺ S ^r Az ^r		62,63	5	W6 derivative
58-278		91	1	
58-278M		154	1	T94A; Treffers mutator strain
58-309		149	1	
58-336		149	1	
58-580		149	1	
58-593		149	1	
58-610		149	1	
58-741		149	1	
58-2651		149	1	
679		56,90,149,150	1,2	
679-183		149,150	1	
679-440		149	1	
679-680		37,90,119,149,150	1,2	
F ⁻		96		
1000		41	11	
1100		41	11	
1200		41	11	
1500		41	11	
2000	B	45	7	
2001w				See P678 S ^r
20SO	B	21,22,45,109	7	
20SOK		22	7	
2320	B	16,109	7	
3000	B	45,109,126	6,7	See Hfr 3000
30SO	B	12,109	6,7	
30SOU1-U7	B	12,21	6	Series of Pyr ⁻ mutants
3300	B	45,109,126	6,7,9,10	
3310	B	45,126	6	
3320	B	126	6,7,10	
3340	B	126	6	
4000	B	126,167	5	P3, P31, Hfr type 3, AB257

^aSee text.

^bP. Reeves, Ph.D thesis, London University, London, U.K., 1959.

In addition to these unpublished sources of information, published descriptions of strains and their derivations are cited in Table 1. These sources are listed in Literature Cited. In a few cases, where published reports were sufficiently detailed or where more direct sources were not available, citations of the literature are used as the sole documentation of pedigrees and strain descriptions. Personal communications regarding recently discovered markers are cited in the text under comments on the charts.

PEDIGREE CHARTS

Conventions

The pedigree charts consist of strain descriptions with lines of descent indicated by arrows. The genetic step involved in the isolation of most strains was mutation, either spontaneous or induced. The mutagenic or selective agent used is indicated beside the arrow. Relatively few recombinant strains are included in the charts; in these cases, the selective conditions used in the isolation of recombinants (where known) are given beside the arrows indicating these steps. Where markers were introduced by transduction, the bacteriophages used and the donor strains are indicated beside the arrows, e.g., P1 from AB1234.

Strain Designations

An effort has been made to use in the charts the original strain designations assigned by the persons who constructed the strains. Widely used synonyms are given in Table 1 and are cross-indexed to the original designations.

In addition to identifying the strains, the letter prefixes used in the original strain designations may indicate the laboratories in which the strains were isolated, and they sometimes convey other information as well. Some of the prefixes used in the charts and the laboratories in which they were assigned are as follows: AB was used by E. A. Adelberg and his collaborators at the University of California at Berkeley and later at Yale University for K-12 derivatives; non-K-12 strains and “hybrid” strains were designated by AC numbers. P. Howard-Flanders, A. J. Pittard, A. L. Taylor, and others had AB “number blocks” and designated their strains accordingly. Howard-Flanders, Pittard, and Taylor later used their own designation prefixes. The prefix AT was used by A. L. Taylor (University of Colorado Medical Center). The prefix C was used at the California Institute of Technology. CR was used by R. Appleyard after he moved from the California Institute of Technology to Chalk River, Canada. CS was used by P. D. Skaar and others for strains isolated at Cold Spring Harbor Laboratory in the 1950s.

The prefix J was used by B. D. Davis at Cornell University Medical College for some K-12 derivatives. (Davis worked extensively with the “W,” or Waksman, wild-type strain of *E. coli* [ATCC 9637], not to be confused with the W [Wisconsin] derivatives of K-12 isolated in the J. Lederberg laboratory.) J is also applied to some of the Hfr strains isolated by F. Jacob and E. Wollman at the Pasteur Institute in Paris. JC is used by A. J. Clark (University of California at Berkeley). KL is used by K. B. Low of Yale University.

P was used by F. Jacob, E. L. Wollman, and others at the Pasteur Institute for F and Hfr strains, while PA was used to designate their F⁻ strains (with the exception of a few early F⁻ strains that had P designations). The various synonymous strain designations applied to some of the Paris Hfr strains have led to considerable confusion, which is dealt with in Table 2.

The prefix W was used by J. Lederberg and collaborators at the University of Wisconsin to designate mutant derivatives of *E. coli* K-12 and other strains of *E. coli*. They used the prefix WG (Wisconsin Genetics) to designate wild-type strains: *E. coli* K-12 was designated WG1. This system continued to be used by the Lederberg laboratory at Stanford.

The prefix Y was used in the laboratory of E. L. Tatum at Yale University in the 1940s. The very earliest derivatives of *E. coli* K-12, those produced by Gray and Tatum at Stanford University, were given only number designations.

TABLE 2 The Paris Hfr's

Type ^a	Synonyms	Point of origin	Derivation	Genotype	Comments
HfrH	Hfr2, original Hayes Hfr Hfr4, Hfr ₄	PO1 <i>valSant^H</i>	Spont. from 58-161 F ⁺ S ^r Azi ^r Recombination: HfrH × W677 F ⁺	<i>azi-7 relA1 rpsL100</i> <i>spoT1 metB1</i> <i>relA1 spoT1 thi-1</i>	
Type 1	Hfr 3000, “HfrC” P1	PO103 <i>leu azi</i>	Hfr4, cured of λ by UV Spont. from 58-161 (W6)	λ ⁻ <i>relA1 spoT1 thi-1</i> <i>relA1 spoT1 metB1</i>	Lost very early
Type 2	P4X, P4X6, J2	PO3 <i>argF lac</i>	Spont. from 58-161 (W6)	<i>relA1 spoT1 metB1</i>	
Type 3	P3, P31, Hfr 4000	PO2B <i>pure fep</i>	Spont. from 58-161 (W6)	<i>relA1 spoT1 metB1</i>	Source of F ₁ - <i>gal</i> (F100) and F ₂ - <i>gal</i> (F152)
Type 4	P10, J4	PO18 <i>thi malK thr</i>	Spont. from C600 F ⁺	<i>thr-1 leuB6 tonA2</i> <i>lacY1 λ⁻ thi-1 malK16</i>	F is integrated in <i>malB</i> locus
Type 5	P72, J5	PO102 <i>metE metB</i>	Spont. from 58-161 (W6)	<i>relA1 spoT1 metB1</i>	
Type 6	P13	PO104 <i>mtl ilv</i>	Spont. from 112 S ^r	<i>gal-5 λ⁻ his-49 cys-23</i> <i>rpsL58</i>	
Type 7	P808	PO105 <i>tonA pro</i>	Spont. from cross: P25 F ⁺ × P678	<i>tonA2 lacY1 xyl-7</i> <i>mtlA2</i> <i>thi-1</i>	
	P2, P21 P804	PO106 <i>lac purE</i> PO65 <i>argF lac</i>	Spont. from 58-161 (W6) Spont. from cross: P25 F ⁻ × P678	<i>relA1 spoT1 metB1</i> <i>thi-1</i>	Source of F- <i>lac</i> of Jacob and Adelberg (F42)

^aAs listed in references 75, 77–79, and 80, p. 162.

Gene Symbols

The gene symbols used are those used by Bachmann (8) with a few exceptions and additions. The older symbols *malA* and *malB* are used for mutations originally mapped in terms of these complex loci. The symbol PO is used to designate the points of origin of Hfr strains, each individual mutation to the Hfr state being assigned a unique number; the Hfr descendants of and the episomes derived from an Hfr strain are assumed to have inherited the point of origin of their Hfr ancestor. (For a description of the locations of points of origin of Hfr strains, see chapter 127 in this volume.) The symbol *sfa* is used to designate sex factor affinity sites as defined by Adelberg and Burns (1).

The mutant allele numbers used throughout are those assigned for use at the *E. coli* Genetic Stock Center and do not necessarily correspond to those used in other laboratories. Three sets of mutant allele designations are included alongside the Stock Center designations in the strain descriptions. These are *gal* designations of the Wisconsin laboratory (along with the *gal_b* designation assigned in the Paris laboratory), some of the *lac* designations of the Paris laboratory, and the designations used by S. Brenner, A. Garen, and their coworkers for suppressor alleles. The Wisconsin *gal* designation is given for a *gal* allele the first time it appears on a pedigree chart and in the descriptions of the HFT *gal* lysates in Chart 8. The *gal_b* symbol is indicated on Charts 2 and 3 in the description of strain P678. A few of the Paris *lac* allele designations are given in Charts 6 and 7.

The symbol F1 is used to refer to the wild-type F factor of *E. coli* K-12. The symbol λ^- is used to indicate the absence of bacteriophage λ . The presence of lambda is not noted in strain descriptions, as this is the wild-type state. Resistance to lambda is symbolized by λ^r .

The symbol S^r occurs in some of the early strain designations, where it indicates resistance to streptomycin.

Other Symbols and Abbreviations

The following symbols and abbreviations are used to designate mutagenic agents: AO, acridine orange; EMS, ethyl methanesulfonate; NA, nitrous acid; NG, *N*-methyl-*N*-nitro-*N*-nitrosoguanidine; N-mustard, nitrogen mustard; Spont., mutation occurring in the absence of deliberate mutagenic treatment; UV, UV irradiation; X-ray, X irradiation.

The following symbols and abbreviations are used to designate selective (Sel'n) agents: APT, aminopterin; Azi, azide; Blood agar, selection for lysis on blood agar plates; EMB-Lac, screening for utilization of the indicated sugar (lactose here) on eosin methylene blue agar plates; λ^x , selection with indicated bacteriophage λ derivative; Motility agar, selection on basis of motility in semisolid agar; Nal, nalidixic acid; Spc, spectinomycin; Str, streptomycin; T1, T2, and T6, bacteriophages T1, T2, and T6, respectively; Trim, trimethoprim. The abbreviations used for sugars are as follows: Ara, arabinose, Gal, galactose; Lac, lactose; Mal, maltose, Mtl, mannitol; and Xyl, xylose.

Comments on the Charts

Chart 1: Stanford and Yale Strains. As can be seen from Chart 1, many of the early strains were isolated after rather drastic treatment with X rays. An appreciation of this fact has led to these strains being abandoned by mSectany in later years as ancestral stocks. Nevertheless, by far the majority of the strains now in use can be traced back to these early lines.

Strains W6, W13, W17, and CS19 are included in this chart to emphasize the instability of the *bio-1* mutation, which reverted very early in several important ancestral stocks (54). This allele is discussed at more length in the comments on Chart 5.

The suppressor mutation *supE44* (*glnV44 = suII⁺*) was detected first in C600 (16) and later traced to strain Y10 (43). The presence of this suppressor in a great many of the derivatives of Y10 has been confirmed since then. It is presumably present in all direct descendants of Y10, including the major F⁻

lines leading to strains P678 and AB1157 and their derivatives.

The allele *leu-6*, from strain 679–680, has been identified as a mutation in *leuB* (34).

Nikaido et al. (119) identified the *rfdD* mutation in strain Y10 and showed that it is present in W1177 (Chart 2). This mutation is presumably present in the major F⁻ lines just mentioned.

Strain 58 carries a mutation in *spoT* (M. Cashel, personal communication), which was first detected in derivatives of strain 58–161 (84). This allele presumably occurs in all of the widely used Hfr derivatives of strain W6.

Strain Y-Mel carries only one suppressor, *supF58* (*tyrT58*), and not two, as was erroneously reported in reference 6.

CHART 1 Stanford and Yale strains

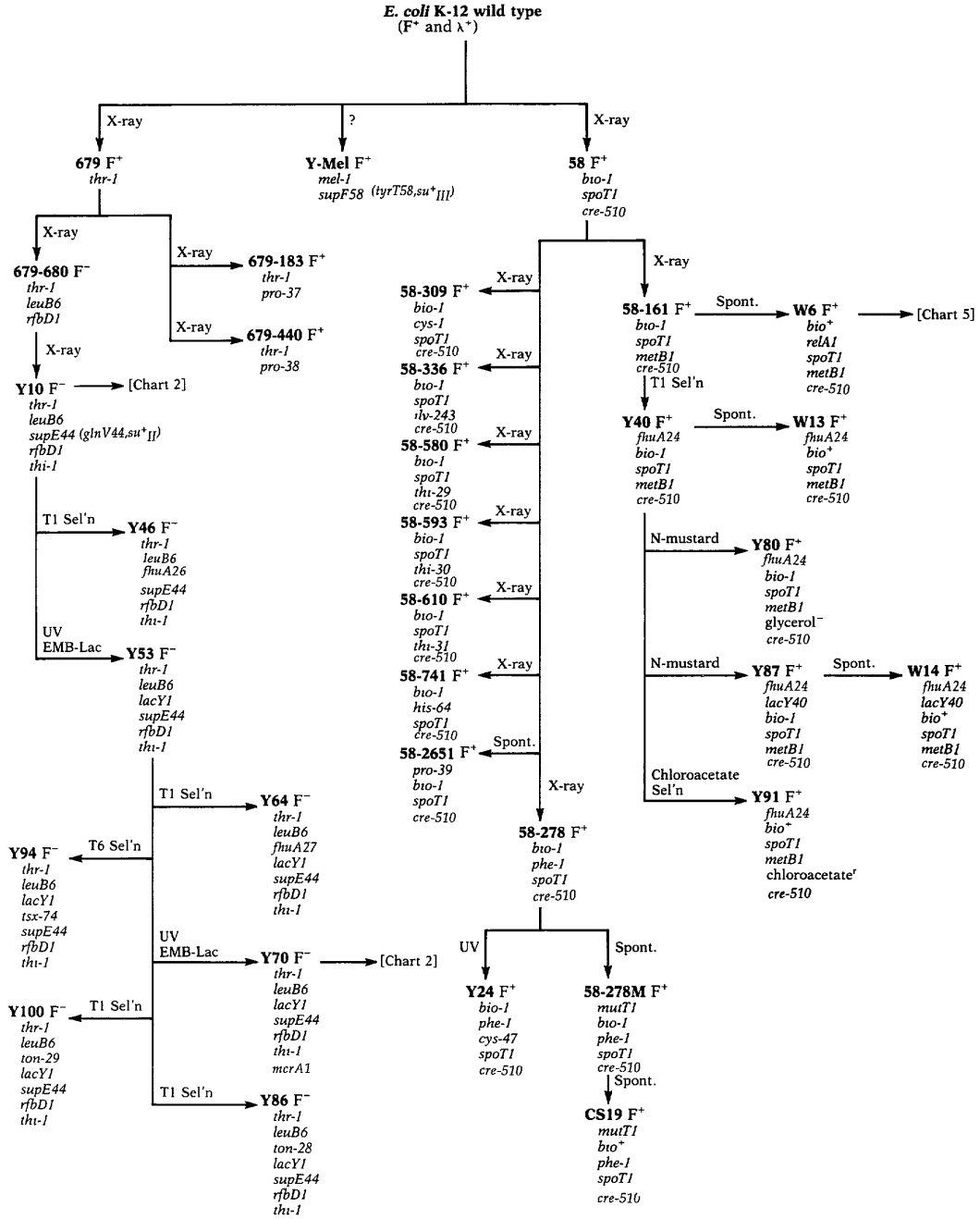
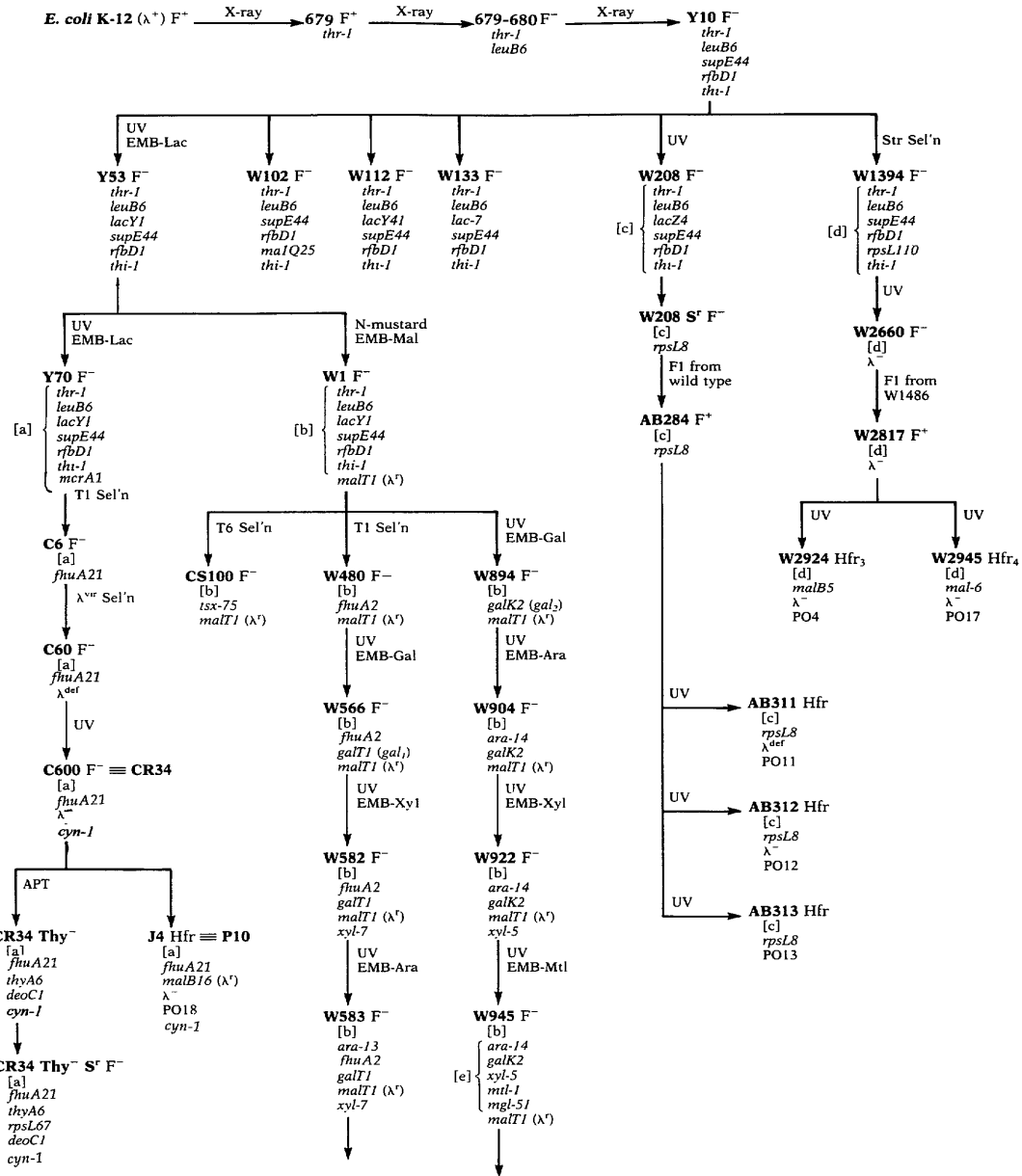


CHART 2 Some derivatives of strain Y10



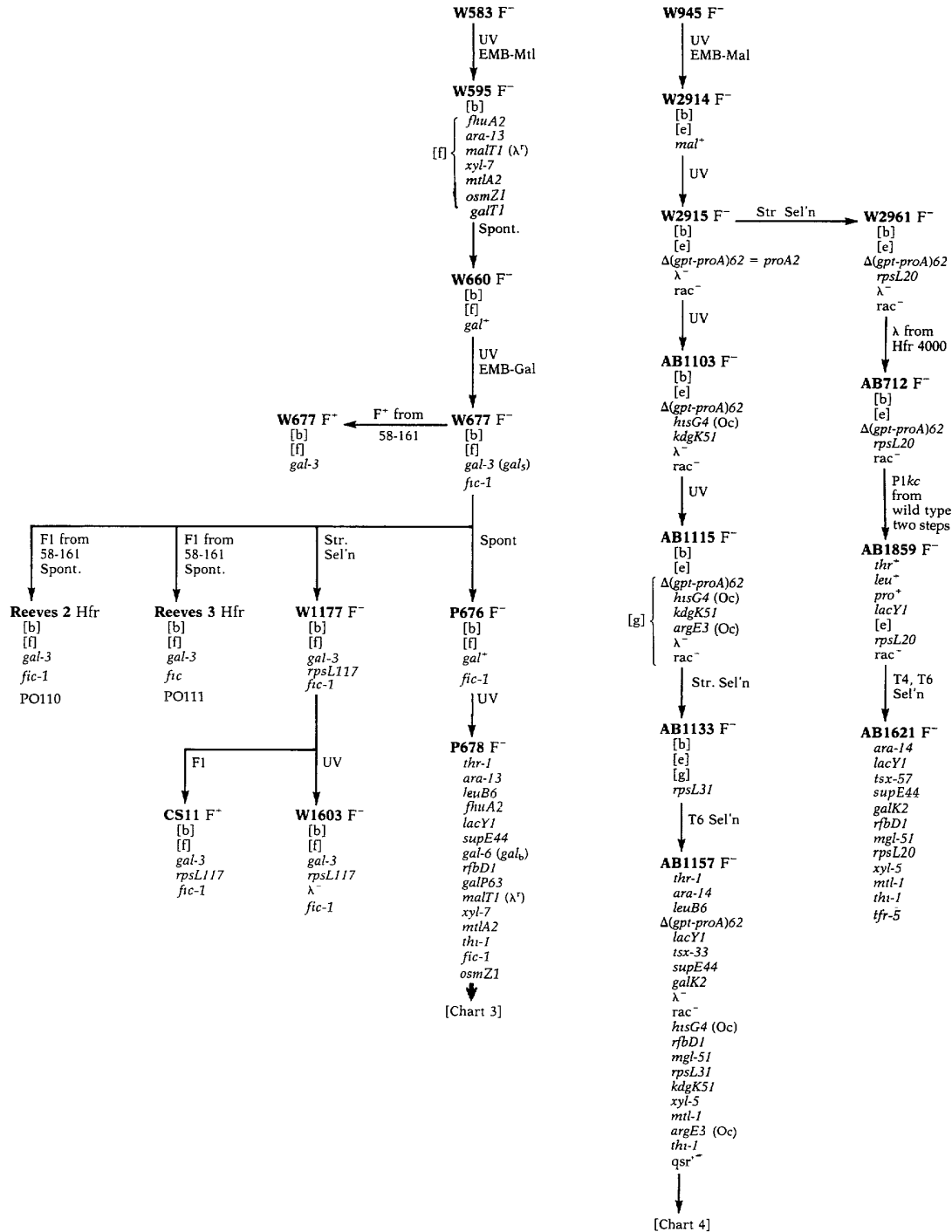


Chart 2: Some Derivatives of Strain Y10. As in Chart 1, *supE44* and *rfbD1* are present in Y10 and its direct descendants.

The strain designations CR34 and C600 are synonymous. Strain C600 was “reisolated” from a single colony by R. Appleyard after he moved from the California Institute of Technology to Chalk River and was redesignated CR34 at that time (R. Appleyard, personal communication). Considerable confusion has arisen from this renaming and from the unfortunate fact that when Okada et al. (121, 122) isolated a Thy⁻ derivative of this strain, they did not give this derivative a unique strain designation. The latter is called simply CR34 Thy⁻.

Another source of confusion has been the *gal* markers in the line from W677 to the Paris strain P678 and its descendants. As shown in Chart 2, the sequence of events involved three *gal* mutations and two reversions. Morse et al. in 1956 (115) recognized that their *gal*₅ (*gal-3*) marker in W677 is complex. The *gal*_b marker (*gal-6*) in P678 and derivatives of this marker give a variety of Gal phenotypes upon recombination (E. A. Adelberg, personal communication) and may even involve a chromosomal rearrangement (R. Curtiss III, personal communication). G. Buttin has shown that P678 carries a mutation in *galP* (22).

The *mtl-2* marker, which arose in strain W595, is in the *mtlA* locus (101).

In Hfr strain J4 (P10), the marker *malB16* resulted from the integration of F into the *malB* locus (138). Wisconsin Hfr₃ (W2924) also arose by the integration of F into the *malB* locus (131, 132; A. Richter, M.S. thesis, University of Wisconsin, Madison, 1957).

The mutation designated *proA2* by E. A. Adelberg, which arose in Lederberg strain W2915, was shown by W. P. M. Hoekstra and H. G. Vis to be a deletion of *gpt-proA* (68). The deletion of the defective prophage *rac*, first detected in strain AB1157 (106), has been traced back to W2915; the parent strain W2914 is *rac*⁺ (K. B. Low, personal communication).

The mutation *his-4* that arose in AB1103 is in the *hisG* locus (P. E. Hartman and F. W. Pons, personal communications) and is an *ochre* mutation (42; A. J. Clark, personal communication). The mutation *kdgK51*, first detected in AB1157 (110), has been traced back to strain AB1103, strain W2915 being *kdgK*⁺ (S. K. Mahajan, personal communication).

An *mgl* mutation has been detected in strains W945 and AB1157 (B. Rotman, personal communication).

The membrane defects of strain AB1621 have been discussed by Schnaitman (137).

CHART 3 Some derivatives of strain P678

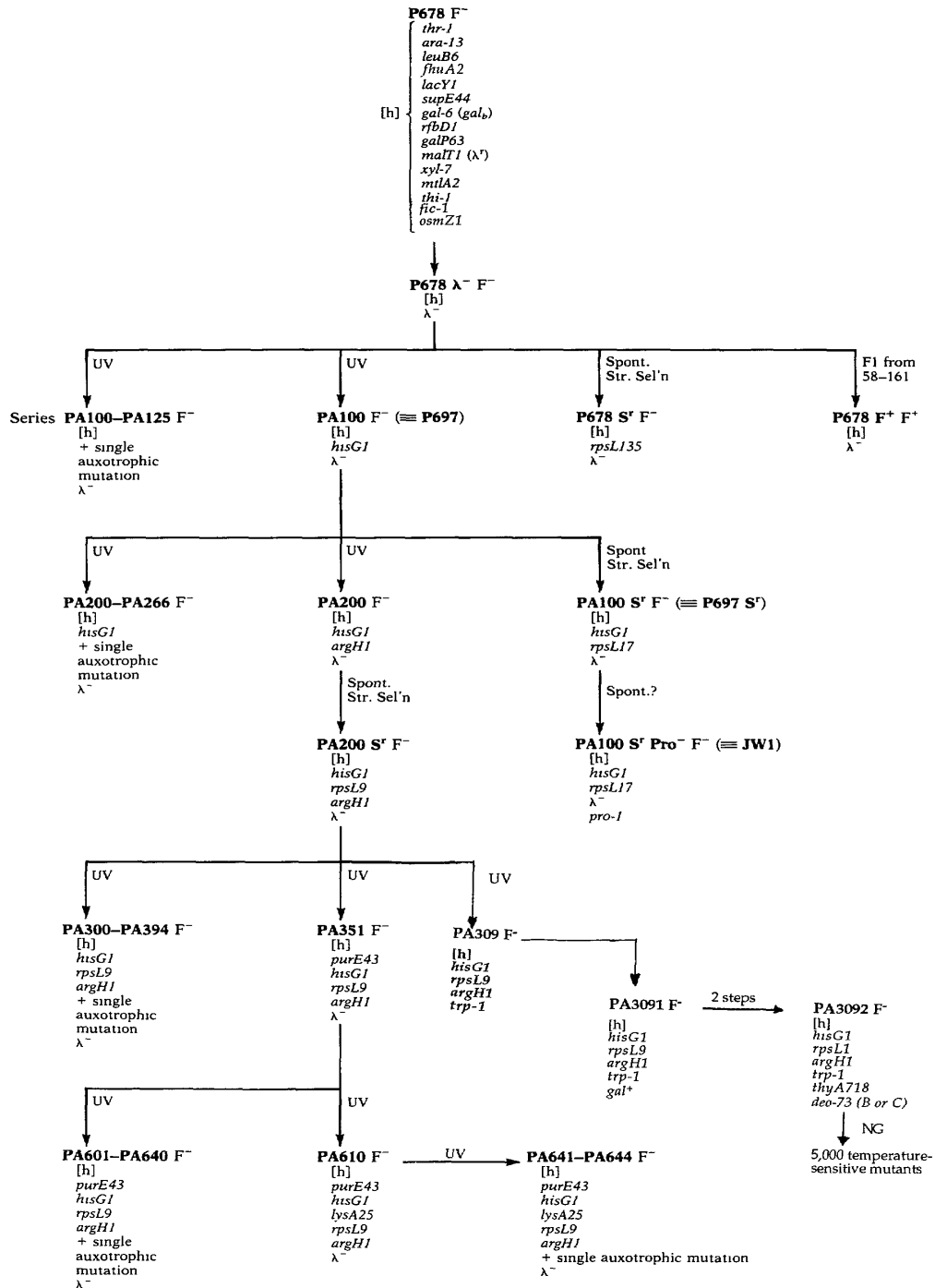


Chart 3: Some Derivatives of Strain P678. The widely used early derivatives of Paris strain P678 consist for the most part of five series of auxotrophic derivatives, each series being isolated from a single strain in the previous series. All are descended from a derivative of P678 that was cured of phage lambda.

The *his* allele that arose in strain PA100 is in the *hisG* locus. This identification was reported by Goldschmidt et al. in 1970 (53), but the allele was inaccurately called *hisG2743* in that publication (T. S. Matney, personal communication).

CHART 4 Some derivatives of strain AB1157

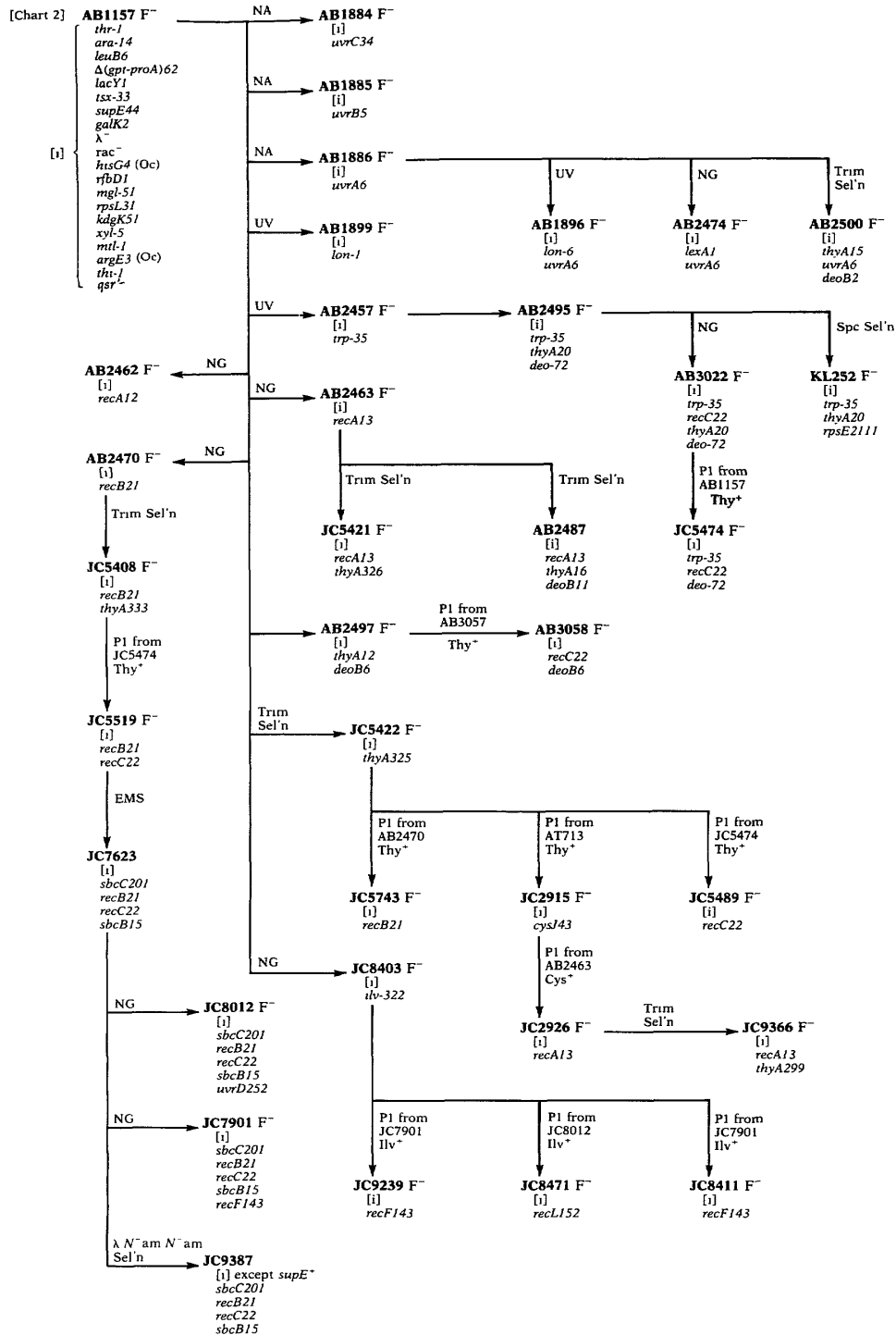


Chart 4: Some Derivatives of Strain AB1157. The markers newly listed for AB1157 have already been discussed in the comments on Chart 2.

Strain JC7623 and other strains carrying mutations in *recB*, *recC*, and *sbcB* have been shown by Lloyd and Buckman to carry compensatory mutations in a newly recognized locus, *sbcC* (103).

CHART 5 Some derivatives of strains 58-161 and W6

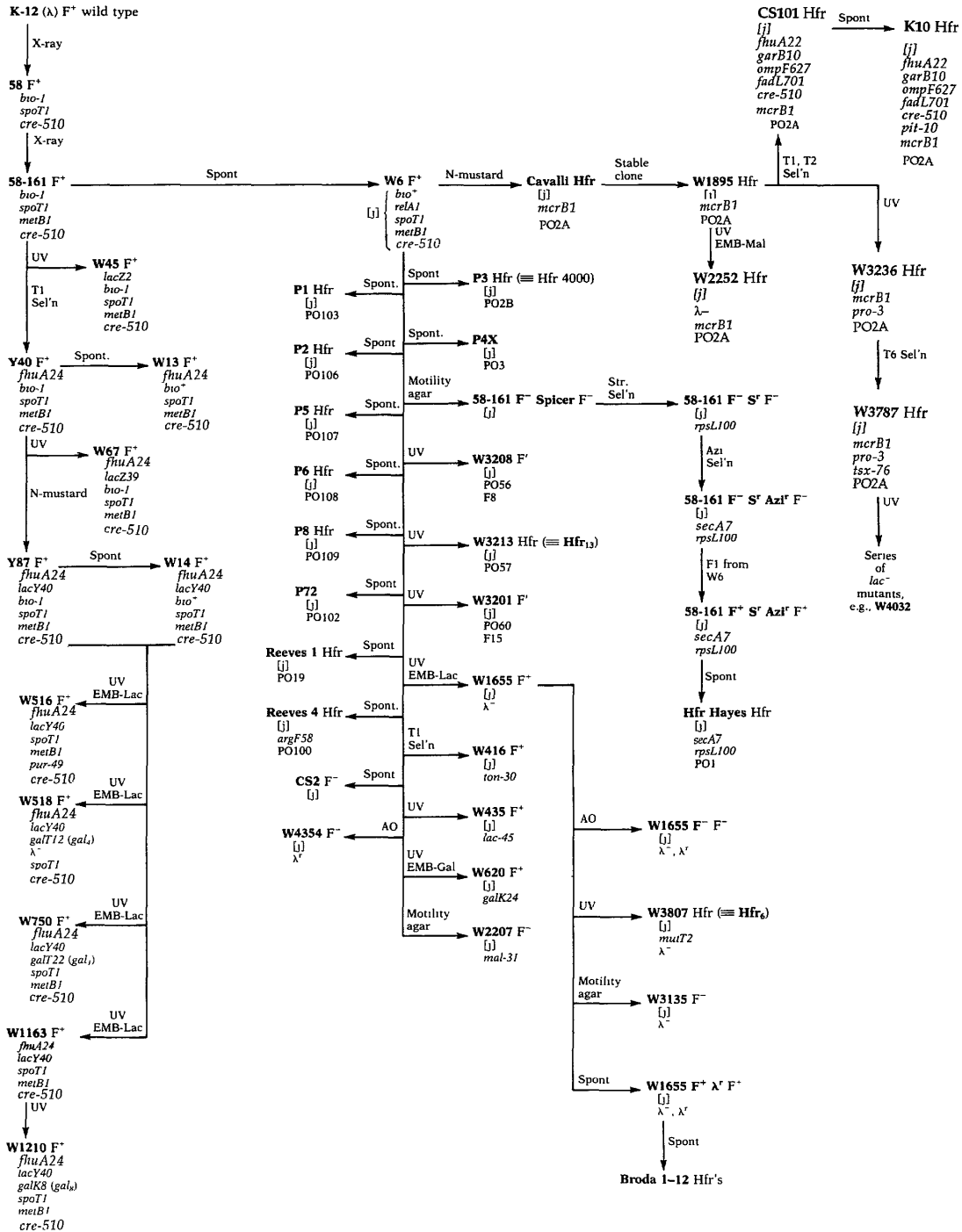


Chart 5: Some Derivatives of Strains 58-161 and W6. Many of the most widely used Hfr strains are to be found in Chart 5. At the time they were isolated, they were thought to have been derived from strain 58-161 (*bio-1 metB1*), but it was later noted (54, 96, 135, 158) that the *bio-1* marker had reverted, apparently soon after 58-161 was isolated. The *bio*⁺ revertant was designated W6 (54). Later still, it was discovered that the mutation *relA1* had appeared in strain W6 very early in its history (3, 14, 15, 135). Finally, the *spoT1* mutation was identified in the “58-61 line” (84) and is now known to have occurred in strain 58 (M. Cashel, personal communication). Thus, most of the widely used Hfr strains are Bio⁺ and carry *relA1* and *spoT1*, being descendants of strain W6. Most of the cultures labeled 58-61 in

various laboratory collections have proved to be W6. The *bio-1* mutation reverted in strain Y40 and Y87 also. Strains that are described in the literature as having come from Y87 most likely came from the *bio*⁺ revertant, which is designated W14 (54).

The Hayes Hfr strain is a spontaneous Hfr derivative of an Str^r Azi^r derivative of W6. The more widely used Thi⁻ λ⁻ derivative, Hfr 3000, is a recombinant derivative of Hfr Hayes, as shown on Chart 6.

The Hfr strains isolated at the Pasteur Institute, Paris, by F. Jacob, E. Wollman, and their collaborators were in some cases given more than one designation. This has led to a certain amount of confusion. The designations, derivations, and genotypes of these strains are given in Table 2. Note particularly that the Thi⁻ λ⁻ derivative of the Hayes Hfr, best referred to as Hfr 3000, was also called HfrC, an informal designation used elsewhere for the Cavalli Hfr. The strain called Hfr type 3, P3, P31, or 4000 is a spontaneous Hfr derivative of W6 (75, 77, 78, 80 [p. 162]) and appears to resemble in all respects the Cavalli Hfr. This strain was called AB257 in the collection of E. A. Adelberg and was distributed as Cavalli Hfr.

Strain CS101 carries a mutation in *ompF* (153) that is involved in the T2^r phenotype of this strain. CS101 has also been reported to carry a mutation at the *garB* locus (134). K10 carries mutations in *pit* (166) and *fadL* (120).

The parent of Cook and Lederberg's (36) series of several hundred *lac* mutants, W3787, is shown in Chart 5.

The Wisconsin strains W3208 and W3201, sometimes referred to as Hfr₈ and Hfr₁₅, respectively, were assumed to be Hfr strains because of their ability to transfer chromosomal markers at high frequency. When these strains were examined in greater detail, both were found to be F' strains, harboring F8 and F15, respectively (67, 74, 144). Thus, nothing is known of the Hfr strains that may have been the immediate ancestors of these two F' strains (J. Lederberg, personal communication).

Hfr Reeves 4 carries a mutation in the *argF* locus (52).

CHART 6 HfrH Thi⁻ λ⁻ (=Hfr 3000) and some of its derivatives

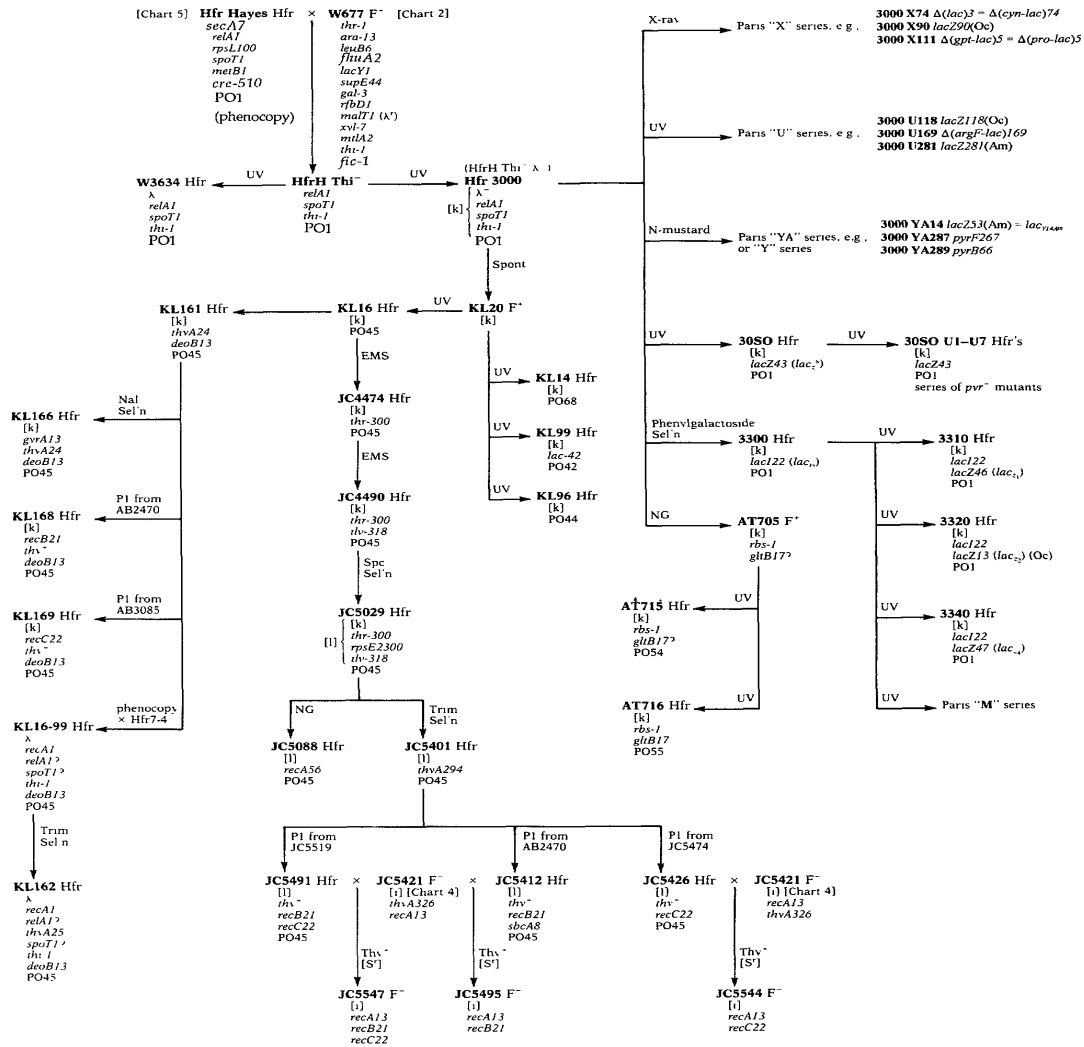


Chart 6: HfrH Thi⁻ λ⁻ (=Hfr 3000) and Some of Its Derivatives. The most widely used derivative of Hayes Hfr, Hfr 3000, is sometimes mistakenly thought to be the original Hfr strain isolated by Hayes. It is, however, a derivative of a recombinant strain that had been isolated from a cross between a phenocopy of Hfr Hayes and the heavily marked Wisconsin strain W677 F⁺. Hfr 3000 carries *spoT1* as well as *relA1* (W. D. Nunn, personal communication).

Several series of mutant strains were produced from Hfr 3000 in the Paris laboratory. The series produced by X irradiation included some of the widely used *lac* deletion strains, such as strains (Hfr) 3000 X74 and (Hfr) 3000 X111. Another series was isolated after UV irradiation (3000 U1 to 3000 U488). This series includes, e.g., Hfr 3000 U118 (*lacZ118*) and Hfr 3000 U169 [Δ (*argF-lac*)169]. A third series, isolated after treatment with nitrogen mustard, consisted of some 279 auxotrophic mutants called (Hfr) 3000 YA1 through (Hfr) 3000 YA297. (Strains bearing higher YA numbers were isolated in a different manner.)

The clusters of markers (*gltS7*, *gadS1*, *gadR2*) listed for strain AT705 in reference 6 were identified in a derivative of AT705 (108); it now appears that the derivative, KL141, does not carry these markers but may carry a *gltB* mutation (113). This marker could be in AT705.

CHART 7 Some early Paris Lac⁻ strains and some of their derivatives

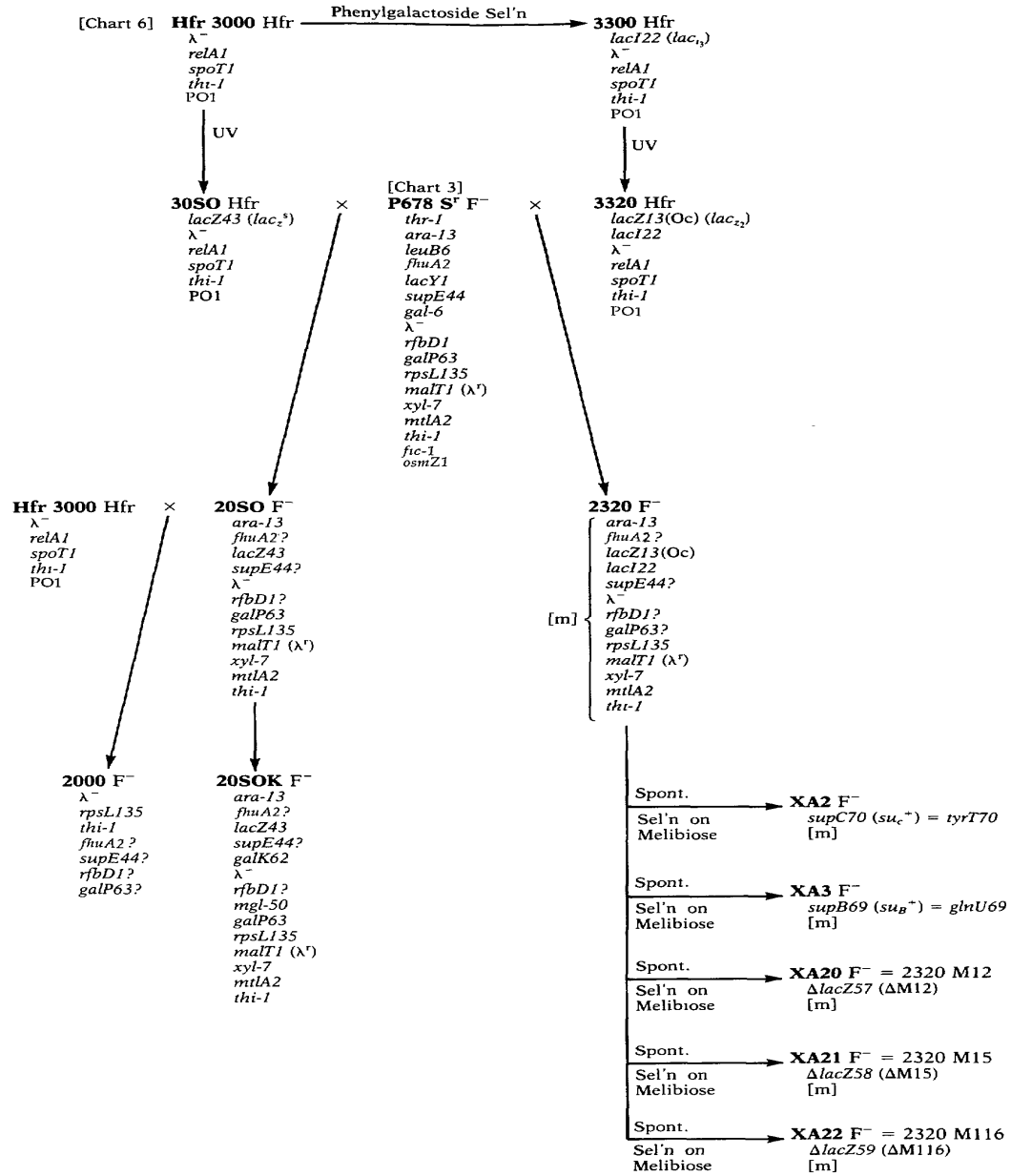


Chart 7: Some Early Paris Lac⁻ Strains and Some of Their Derivatives. Chart 7 shows the rather complex interrelationships among some of the derivatives of Paris strains 30SO and 3300 and also the derivation of some widely used strains isolated by S. Brenner and J. Beckwith.

Strain 20SOK carries an *mgl* mutation that is not in *mglB* or *mglD* (47; Rotman, personal communication).

CHART 8 Other lines derived from the wild type

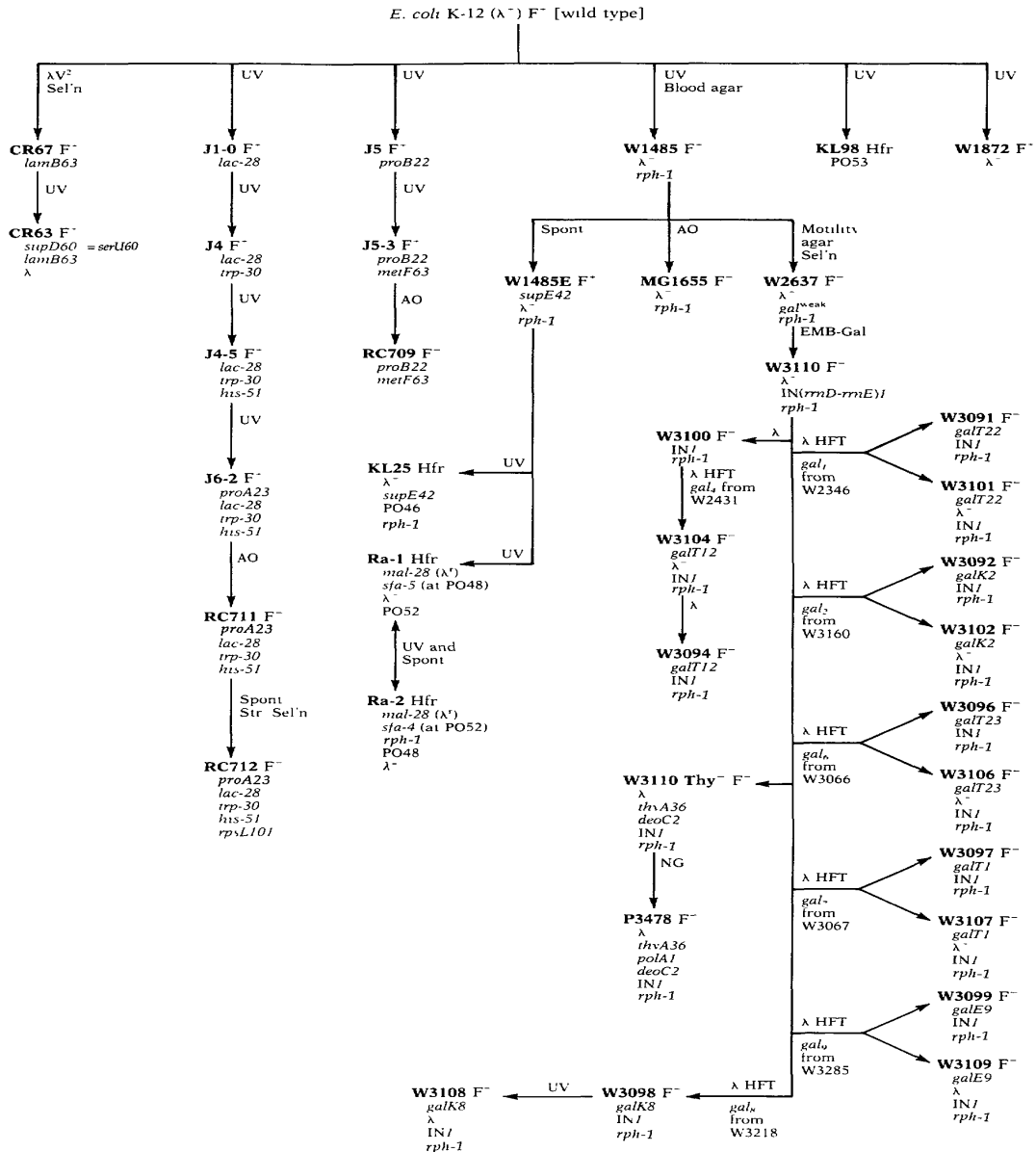


Chart 8: Other Lines Derived from the Wild Type. The *pro* marker in strain J6-2 has been identified as a mutation in *proA*, and marker J5 is a *proB* mutation (114). The *met* allele strain J5-3 is a *metF* allele (51).

Strain W1485 does not carry a suppressor. However, the strain distributed by C. Yanofsky as W1485 does carry a suppressor mutation (*supE42 = glnV42*) (J. Lederberg and C. Yanofsky, personal communications). This strain, which was inadvertently used in a number of laboratories as an ancestral stock under the impression that it was W1485, is now designated W1485E. W1485E carries one copy of $\gamma\delta$ (*Tn1000*) inserted in the chromosome (59). The culture of "W1485" from the laboratory of N. Davidson has three copies of $\gamma\delta$ inserted in the chromosome (59); this strain is now designated W1485D.

Strain W3110 carries an inversion of the chromosome between *rrnD* and *rrnE* (61) that is designated *IN(rrnD-rrnE)1*. This defect is in derivatives of W3110 that were isolated soon after W3110 was isolated (66) and may be in all of its direct descendants.

CHART 9 JC12 and JC411 and some of their derivatives

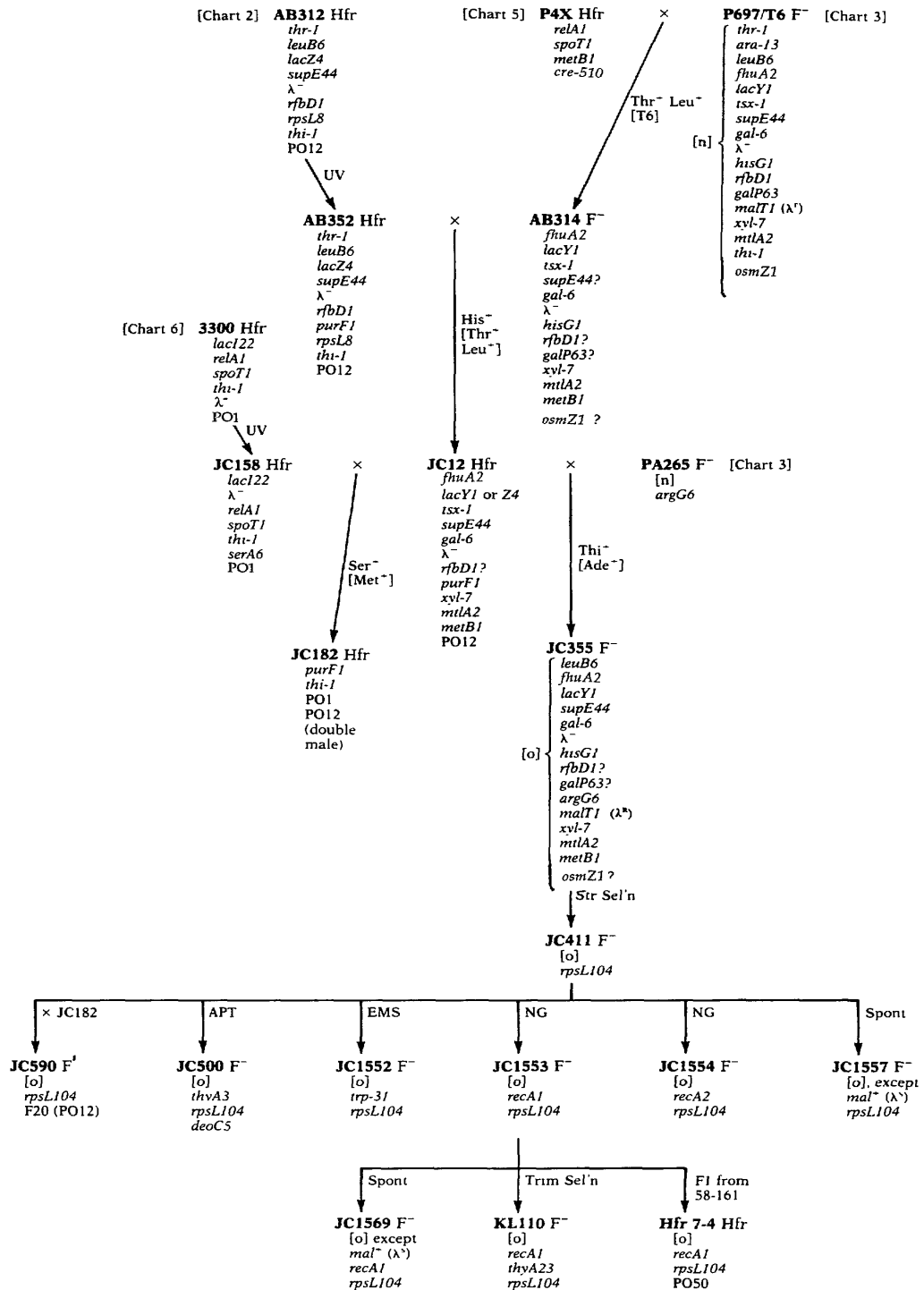


Chart 9: JC12 and JC411 and Some of Their Derivatives. The recombinant strains JC12 and JC411 are shown in Chart 9 because they have been used widely as ancestral stocks. Note that the *gal-6* (*galb*) “marker” is in most of these strains.

CHART 10 S. Brenner set of suppressor strains

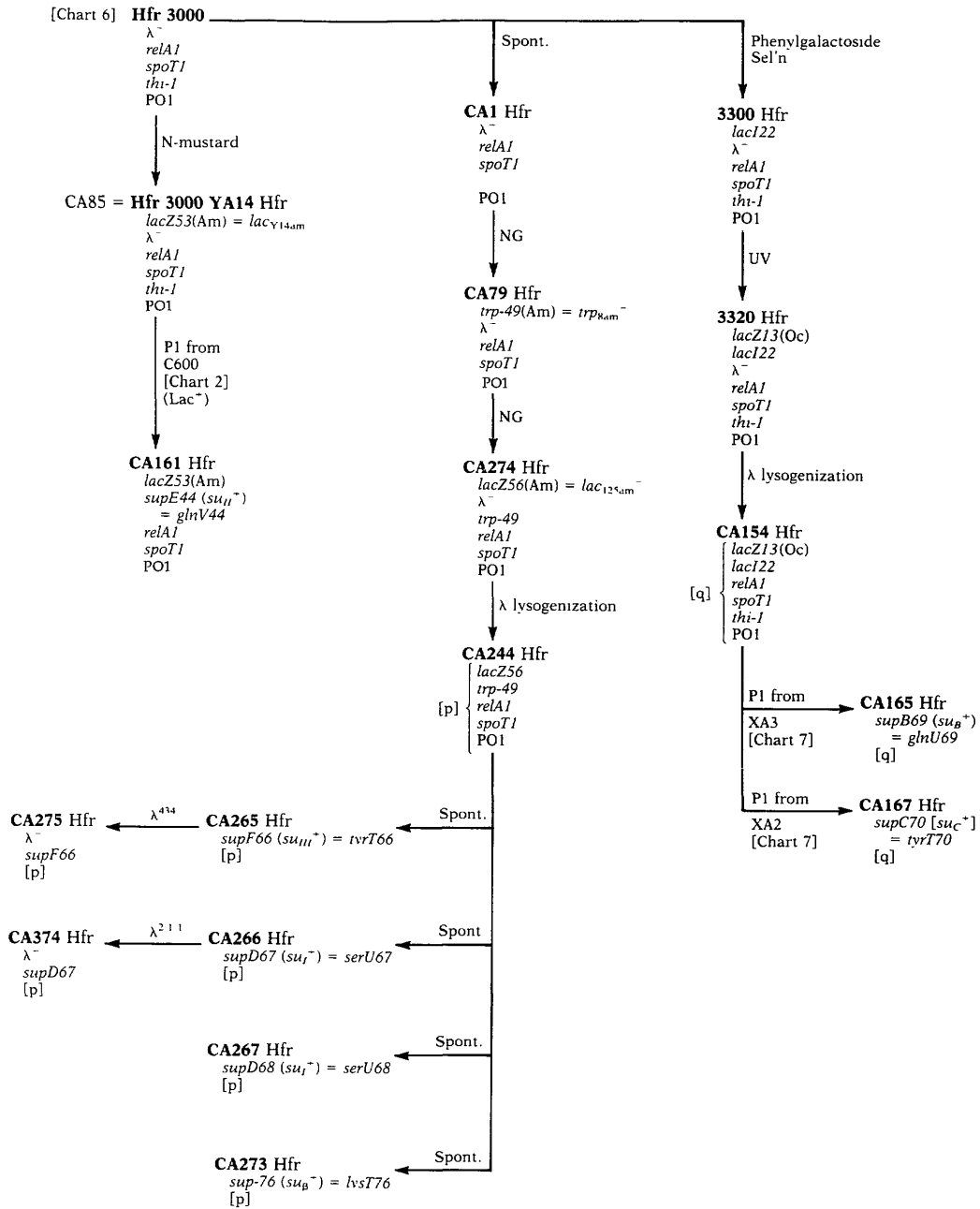


Chart 10: S. Brenner Set of Suppressor Strains. The strains whose derivation is shown in Chart 10 constitute a nearly isogenic set of strains carrying suppressor mutations

CHART 11 Derivation of MM294 and DH1

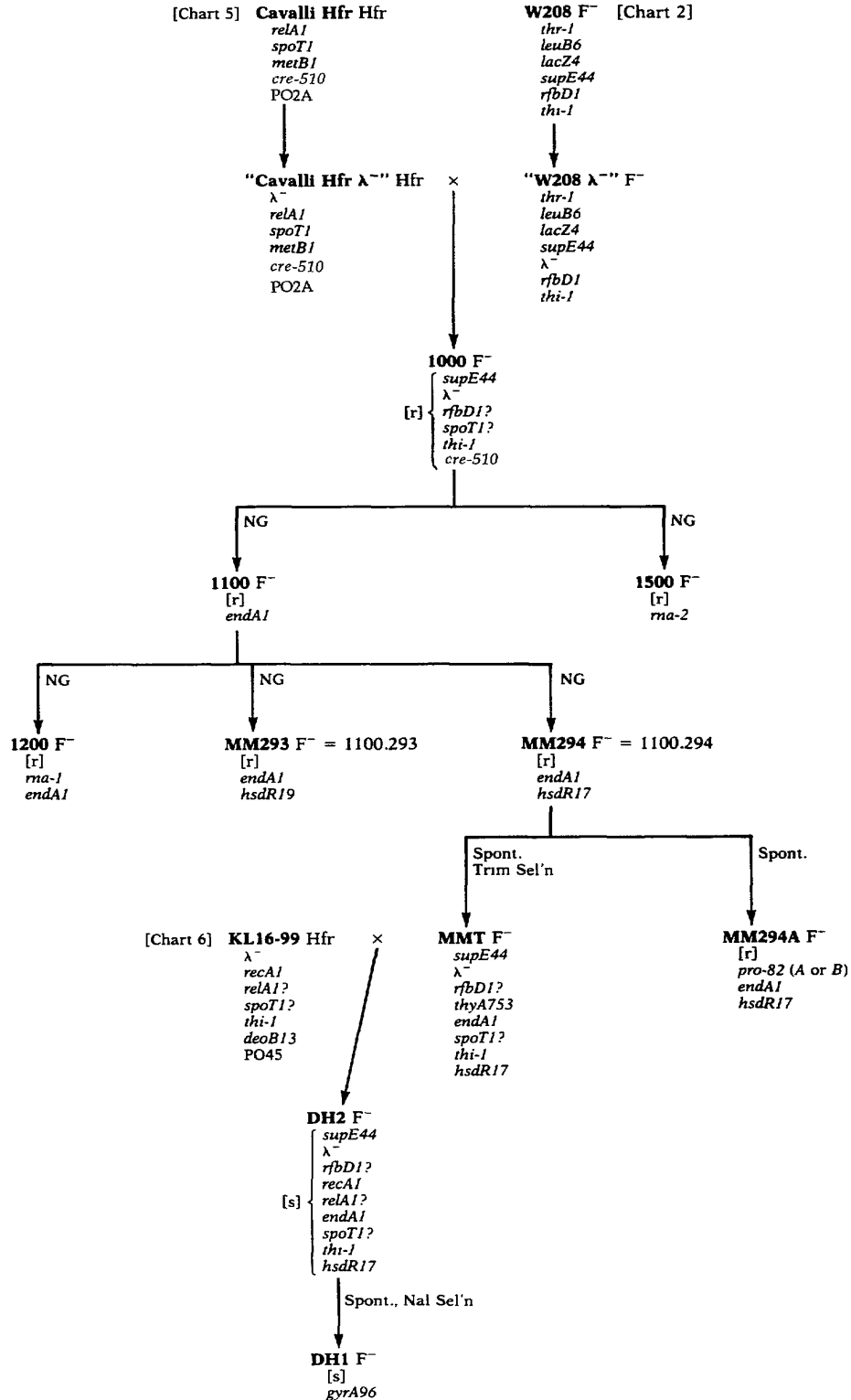


Chart 11: Derivation of MM294 and DH1. MM294 cultured in the laboratory of M. Ptashne acquired a mutation in either *proA* or *proB* (9). This spontaneous derivative of MM294 is designated MM294A. Also shown in Chart 11 are the EndA⁻ and Rna⁻ mutants of Dürwald and Hoffmann-Berling (41).

CHART 12 Derivation of some Garen Pho^- and “ Su^+ ” strains

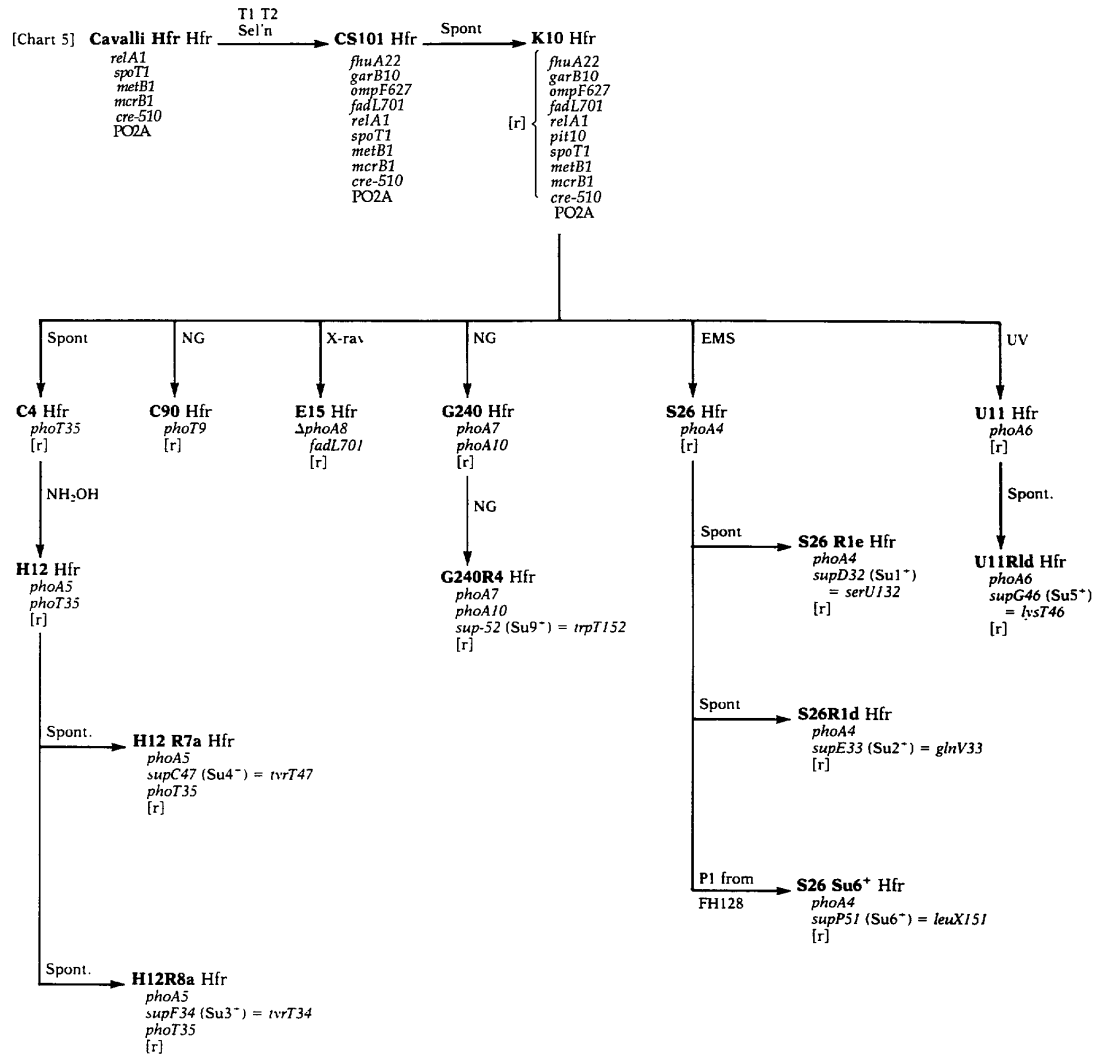
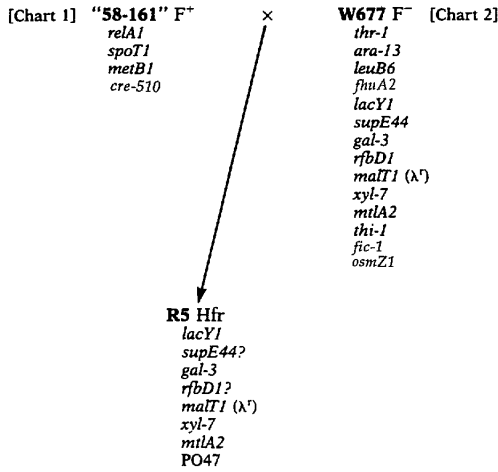


Chart 12: Derivation of Some Garen Pho^- and “ Su^+ ” Strains. The set of Pho^- and “ Su^+ ” strains is often used as a source of suppressor alleles. All of these strains are expected to carry the markers discovered in strain K10 in recent years. Strain C90 produces alkaline phosphatase constitutively. The mutation *phoA8* in strain E15 is a deletion, thought to be intragenic. The mutation *fadL701* was detected by Nunn and Simons (120).

The derivation of strain H12 was given incorrectly in reference 6 and has been corrected here.

CHART 13 Derivations of some Hfr strains

A. Hfr R5 (Reeves 5)



B. Hfr's P802, P804, and P808

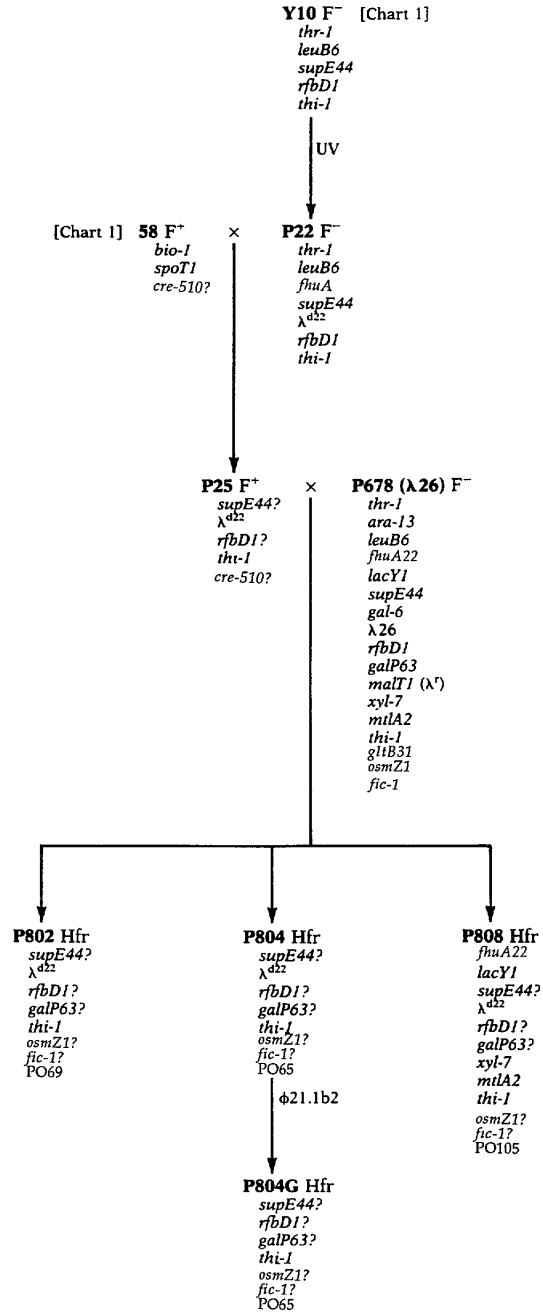


Chart 13: Derivations of Some Hfr Strains. The Hfr Reeves 5, or R5, strain arose spontaneously during a cross between strains 58-161 F⁺ (presumably W6) and W677 F⁻ (P. Reeves, personal communication). It is a recombinant derivative of these two strains.

Hfr P804 is the source of the Paris F *lac*⁺ episome (F42).

CHART 14 Derivation of some widely used strains isolated by Campbell, Meselson, and Wood

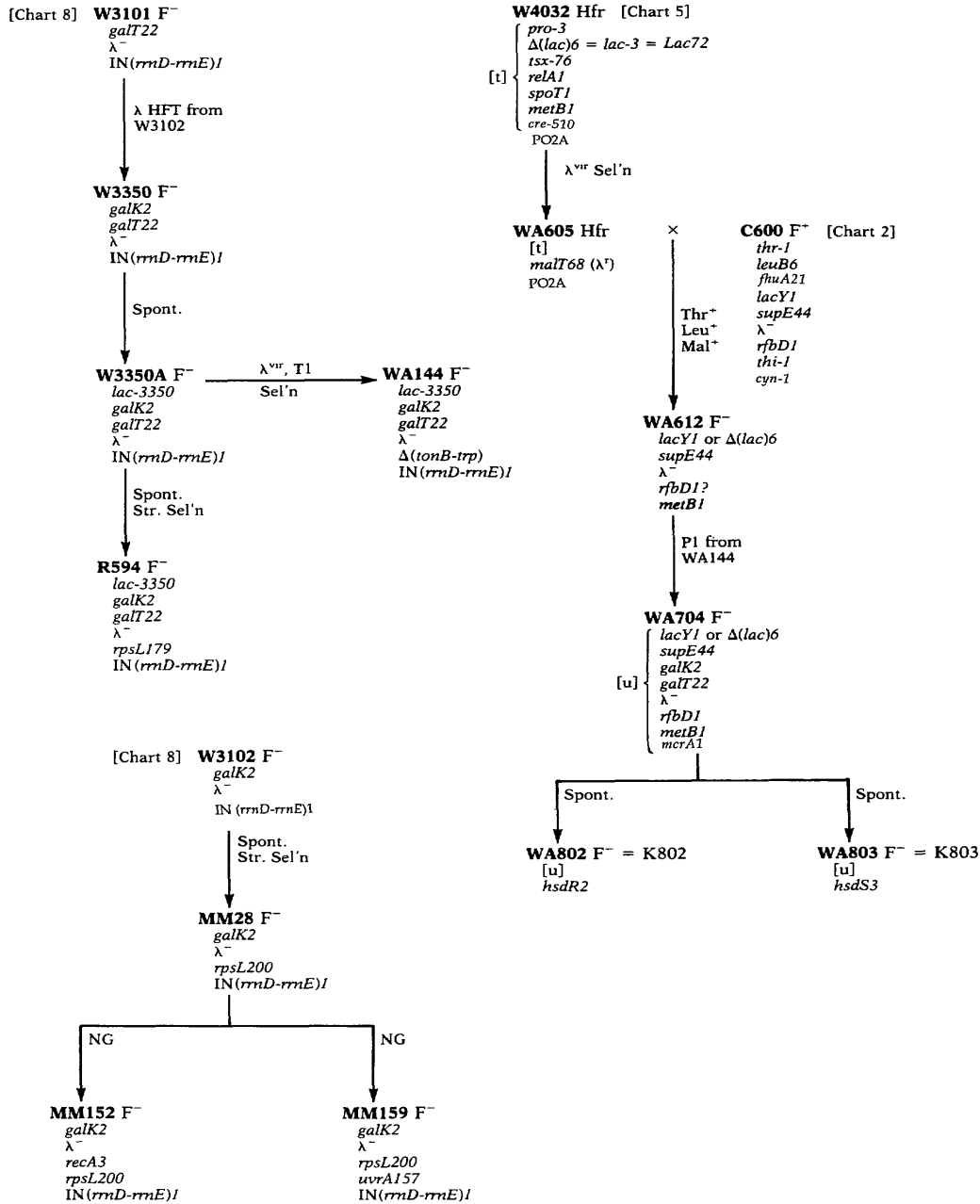


Chart 14: Derivation of Some Widely Used Strains Isolated by Campbell, Meselson, and Wood. Strain W3350 was constructed by J. Weigle and given soon after its isolation to A. Campbell, who isolated the derivative R594 (23). It was later noted that R594 carries a *lac* mutation. Examination of derivatives of W3550 isolated in Campbell's laboratory has led to the conclusion that the strain was Lac⁺ when received by Campbell but acquired the *lac* mutation sometime during 1957 (A. Campbell, personal communication). This Lac⁻ derivative is designated W3550A.

FURTHER COMMENTS

In addition to the pedigrees presented here, the derivations of a few thousand other descendants of *E. coli* K-12 have been traced and are individually available from the *E. coli* Genetic Stock Center upon request.

It is becoming apparent, however, that the derivation of many strains being isolated today can never be traced because of the failure of some laboratories to maintain adequate records of strain constructions. This unfortunate trend is leading to a regrettable loss of continuity in strain derivations. Knowledge of the derivations and genotypes of the strains being used (including “irrelevant” markers) can contribute significantly to the interpretation of experimental results in some cases and can save considerable time and effort that otherwise might be spent in repeating history.

ACKNOWLEDGMENTS

This work has been supported by the following grants awarded by the National Science Foundation for the support of the *E. coli* Genetic Stock Center: BSR8807021, BSR8501620, and BIR9315421. This support is gratefully acknowledged. I express my gratitude to all of those, now too numerous to mention, who patiently went through their old records and contributed information essential to the tracing of pedigrees and, in particular, to those who made their laboratory strain records available to me.

LITERATURE CITED

1. **Adelberg, E. A., and S. N. Burns.** 1960. Genetic variation in the sex factor of *Escherichia coli*. *J. Bacteriol.* **79**:321–330.
2. **Adelberg, E. A., M. Mandel, and G. C. C. Chen.** 1965. Optimal conditions for mutagenesis by N-methyl-N'-nitro-N-nitrosoguanidine in *Escherichia coli* K-12. *Biochem. Biophys. Res. Commun.* **18**:788–795.
3. **Alfoldi, L., G. S. Stent, and R. C. Clowes.** 1962. The chromosomal site of the RNA control (RC) locus in *Escherichia coli*. *J. Mol. Biol.* **5**:348–355.
4. **Anilionis, A., P. Ostapchuk, and M. Riley.** 1980. Identification of a second cryptic lambdoid prophage locus in the *E. coli* K-12 chromosome. *Mol. Gen. Genet.* **180**:479–481.
5. **Appleyard, R. K.** 1954. Segregation of new lysogenic types during growth of a doubly lysogenic strain derived from *Escherichia coli* K12. *Genetics* **39**:440–452.
6. **Appleyard, R. K., J. F. McGregor, and K. M. Baird.** 1956. Mutation to extended host range and the occurrence of phenotypic mixing in the temperate coliphage lambda. *Virology* **2**:565–574.
7. **Bachmann, B. J.** 1987. Derivations and genotypes of some mutant derivatives of *Escherichia coli* K-12, p. 1192–1219. In F. C. Neidhardt, J. I. Ingraham, K. B. Low, B. Magasanik, M. Schaechter, and H. E. Umbarger (ed.), *Escherichia coli and Salmonella typhimurium: Cellular and Molecular Biology*, vol. 2. American Society for Microbiology, Washington, D.C.
8. **Bachmann, B. J.** 1990. Linkage map of *Escherichia coli* K-12, edition 8. *Microbiol. Rev.* **54**:130–197.
9. **Backman, K., and H. W. Boyer.** 1983. Tetracycline resistance determined by pBR322 is mediated by one polypeptide. *Gene* **26**:197–203.
10. **Barbour, S. D., H. Nagaishi, A. Templin, and A. J. Clark.** 1970. Biochemical and genetic studies of recombination proficiency in *Escherichia coli*. II. *rec*⁺ revertants caused by indirect suppression of *rec*⁻ mutations. *Proc. Natl. Acad. Sci. USA* **67**:128–135.
11. **Beckwith, J. R.** 1964. A deletion analysis of the *Lac* operator region in *Escherichia coli*. *J. Mol. Biol.* **8**:427–430.
12. **Beckwith, J. R., A. B. Pardee, R. Austrian, and F. Jacob.** 1962. Coordination of the synthesis of the enzymes in the pyrimidine pathway of *E. coli*. *J. Mol. Biol.* **5**:618–634.
13. **Beckwith, J. R., and E. R. Signer.** 1966. Transportation of the *Lac* region of *Escherichia coli*. I. Inversion of the *Lac* operon and transduction of *Lac* by ϕ 80. *J. Mol. Biol.* **19**:254–265.

14. **Borek, E., J. Rockenbach, and A. Ryan.** 1956. Studies on a mutant of *Escherichia coli* with unbalanced ribonucleic acid synthesis. *J. Bacteriol.* **71**:318–323.
15. **Borek, E., A. Ryan, and J. Rockenbach.** 1955. Nucleic acid metabolism in relation to the lysogenic phenomenon. *J. Bacteriol.* **69**:460–467.
16. **Brenner, S., and J. R. Beckwith.** 1965. *Ochre* mutants, a new class of suppressible nonsense mutants. *J. Mol. Biol.* **13**:629–637.
17. **Brikun, I., K. Suziedelis, and D. E. Berg.** 1994. DNA sequence divergence among derivatives of *Escherichia coli* K-12 detected by arbitrary primer PCR (random amplified polymorphic DNA) fingerprinting. *J. Bacteriol.* **176**:1673–1682.
18. **Broda, P.** 1967. The formation of Hfr strains in *Escherichia coli* K12. *Genet. Res.* **9**:35–47.
19. **Broda, P., J. R. Beckwith, and J. Scaife.** 1964. The characterization of a new type of F-prime factor in *Escherichia coli* K12. *Genet. Res.* **5**:489–494.
20. **Brooks, K., and A. J. Clark.** 1967. Behavior of λ bacteriophage in a recombination-deficient strain of *Escherichia coli*. *J. Virol.* **1**:283–293.
21. **Buttin, G.** 1963. Mécanismes régulateurs dans la biosynthèse des enzymes du métabolisme du galactose chez *Escherichia coli* K12. I. La biosynthèse induite de la galactokinase et l'induction simultanée de la sequence enzymatique. *J. Mol. Biol.* **7**:164–182.
22. **Buttin, G.** 1963. Mécanismes régulateurs dans la biosynthèse des enzymes du métabolisme du galactose chez *Escherichia coli* K12. II. Le déterminisme génétique de la régulation. *J. Mol. Biol.* **7**:183–205.
23. **Campbell, A.** 1961. Sensitive mutants of bacteriophage λ . *Virology* **14**:22–32.
24. **Campbell, A.** 1965. The steric effect in lysogenization by bacteriophage lambda. I. Lysogenization of a partially diploid strain of *Escherichia coli* K12. *Virology* **27**:329–339.
25. **Casadaban, M. J.** 1976. Transposition and fusion of the *lac* genes to selected promoters in *Escherichia coli* using bacteriophage lambda and Mu. *J. Mol. Biol.* **104**:541–555.
26. **Cavalli, L. L., J. Lederberg, and E. M. Lederberg.** 1953. An infective factor controlling sex compatibility in *Bacterium coli*. *J. Gen. Microbiol.* **8**:89–103.
27. **Cavalli, L. L., and G. A. Maccacaro.** 1950. Chloromycetin resistance in *E. coli*, a case of quantitative inheritance in bacteria. *Nature (London)* **166**:991–992.
28. **Cavalli-Sforza, L. L.** 1950. La sessualita nei batteri. *Boll. Ist. Sieroter. Milano* **29**:281–289.
29. **Chan, T.-S., and A. Garen.** 1970. Amino acid substitutions resulting from suppression of nonsense mutations. V. Tryptophan insertion by the $Su9^+$ gene, a suppressor of the UGA nonsense triplet. *J. Mol. Biol.* **49**:231–234.
30. **Clark, A. J.** 1963. Genetic analysis of a “double male” strain of *Escherichia coli* K-12. *Genetics* **48**:105–120.
31. **Clark, A. J.** 1967. The beginning of a genetic analysis of recombination proficiency. *J. Cell. Physiol.* **70**(Suppl. 1):165–180.
32. **Clark, A. J., W. K. Maas, and B. Low.** 1969. Production of a merodiploid strain from a double male strain of *E. coli* K12. *Mol. Gen. Genet.* **105**:1–15.
33. **Clark, A. J., and A. D. Margulies.** 1965. Isolation and characterization of recombination-deficient mutants of *Escherichia coli* K12. *Proc. Natl. Acad. Sci. USA* **53**:451–459.
34. **Clowes, R., and D. Rowley.** 1954. Some observations on linkage effects in genetic recombination in *Escherichia coli* K12. *J. Gen. Microbiol.* **11**:250–260.
35. **Clowes, R. C., and W. Hayes.** 1968. *Experiments in Microbial Genetics*. John Wiley & Sons, Inc., New York.
36. **Cook, A., and J. Lederberg.** 1962. Recombination studies of lactose nonfermenting mutants of *Escherichia coli* K-12. *Genetics* **47**:1335–1353.
37. **Davis, M. G., and J. M. Calvo.** 1977. Relationship between messenger ribonucleic acid and enzyme levels specified by the leucine operon of *Escherichia coli* K-12. *J. Bacteriol.* **131**:997–1007.
38. **DeLucia, P., and J. Cairns.** 1969. Isolation of an *E. coli* strain with a mutation affecting DNA polymerase. *Nature (London)* **224**:1164–1166.
39. **Diderichsen, B.** 1980. *cur-1*, a mutation affecting the phenotype of sup^+ strains of *Escherichia coli*.

- Mol. Gen. Genet.* **180**:425–428.
40. **Dubnau, E., and W. K. Maas.** 1968. Inhibition of replication of an F' *lac* episome in Hfr cells of *Escherichia coli*. *J. Bacteriol.* **95**:531–539.
 41. **Dürwald, H., and H. Hoffmann-Berling.** 1968. Endonuclease I-deficient and ribonuclease I-deficient *Escherichia coli* mutants. *J. Mol. Biol.* **34**:331–346.
 42. **Eggertsson, G., and E. A. Adelberg.** 1965. Map positions and specificities of suppressor mutations in *Escherichia coli* K-12. *Genetics* **52**:319–340.
 43. **Eisen, H. A., C. R. Fuerst, L. Siminovitch, R. Thomas, L. Lambert, L. Pereira da Silva, and F. Jacob.** 1966. Genetics and physiology of defective lysogeny in K12(λ): studies of early mutants. *Virology* **30**:224–241.
 44. **Emmerson, P. T.** 1968. Recombination deficient mutants of *Escherichia coli* K12 that map between *thyA* and *argA*. *Genetics* **60**:19–30.
 45. **Franklin, N. C., and S. E. Luria.** 1961. Transduction by bacteriophage P1 and the properties of the *lac* genetic region in *E. coli* and *S. dysenteriae*. *Virology* **15**:299–311.
 46. **Gallucci, E., and A. Garen.** 1966. Suppressor genes for nonsense mutations. II. The *Su-4* and *Su-5* suppressor genes of *E. coli*. *J. Mol. Biol.* **15**:193–200.
 47. **Ganesan, A. K., and B. Rotman.** 1966. Transport systems for galactose and galactosides in *Escherichia coli*. I. Genetic determination and regulation of the methyl-galactoside permease. *J. Mol. Biol.* **16**:42–50.
 48. **Garen, A., and S. Garen.** 1963. Genetic evidence on the nature of the repressor for alkaline phosphatase in *E. coli*. *J. Mol. Biol.* **6**:433–438.
 49. **Garen, A., S. Garen, and R. Wilhelm.** 1965. Suppressor genes for nonsense mutations. I. The *Su-1*, *Su-2* and *Su-3* genes of *Escherichia coli*. *J. Mol. Biol.* **14**:167–178.
 50. **Garen, A., and O. Siddiqi.** 1962. Suppression of mutations in the alkaline phosphatase structural cistron of *E. coli*. *Proc. Natl. Acad. Sci. USA* **48**:1121–1127.
 51. **Glansdorff, N.** 1965. Topography of cotransducible arginine mutations in *Escherichia coli* K-12. *Genetics* **51**:167–179.
 52. **Glansdorff, N., G. Sand, and C. Verhoef.** 1967. The dual genetic control of ornithine transcarbamylase synthesis in *Escherichia coli* K-12. *Mutat. Res.* **4**:743–751.
 53. **Goldschmidt, E. P., M. S. Cater, T. S. Matney, M. A. Butler, and A. Greene.** 1970. Genetic analysis of the histidine operon in *Escherichia coli* K-12. *Genetics* **66**:219–229.
 54. **Gosting, D. C., and J. Lederberg.** 1956. Biotin requirement of cultures derived from *E. coli* K-12 strain 58. *Microb. Genet. Bull.* **13**:13–14.
 55. **Gottesman, M. E., and M. B. Yarmolinsky.** 1968. Integration-negative mutants of bacteriophage lambda. *J. Mol. Biol.* **31**:487–505.
 56. **Gray, C. H., and E. L. Tatum.** 1944. X-ray induced growth factor requirements in bacteria. *Proc. Natl. Acad. Sci. USA* **30**:404–410.
 57. **Gross, J., and M. Gross.** 1969. Genetic analysis of an *E. coli* strain with a mutation affecting DNA polymerase. *Nature (London)* **224**:1166–1168.
 58. **Guilloton, M., and F. Karst.** 1987. Isolation and characterization of *Escherichia coli* mutants lacking inducible cyanase. *J. Gen. Microbiol.* **133**:645–653, 655–665.
 59. **Guyer, M. S., R. R. Reed, J. A. Steitz, and K. B. Low.** 1980. Identification of a sex-factor-affinity site in *E. coli* as $\gamma\delta$. *Cold Spring Harbor Symp. Quant. Biol.* **45**:135–140.
 60. **Hanahan, D.** 1983. Studies on transformation of *Escherichia coli* with plasmids. *J. Mol. Biol.* **166**:557–580.
 61. **Hayashi, S. I., J. P. Koch, and E. C. C. Lin.** 1964. Active transport of L- α -glycerophosphate in *Escherichia coli*. *J. Biol. Chem.* **239**:3098–3105.
 62. **Hayes, W.** 1953. Observations on a transmissible agent determining sexual differentiation in *Bacterium coli*. *J. Gen. Microbiol.* **8**:72–88.
 63. **Hayes, W.** 1953. The mechanism of genetic recombination in *Escherichia coli*. *Cold Spring Harbor Symp. Quant. Biol.* **18**:75–93.
 64. **Hayes, W.** 1964. *The Genetics of Bacteria and Their Viruses*, p. 568–569. John Wiley & Sons,

Inc., New York.

65. **Hayes, W.** 1968. *The Genetics of Bacteria and Their Viruses*, 2nd ed., p. 666–667. John Wiley & Sons, Inc., New York.
66. **Hill, C. W., and B. W. Harnish.** 1981. Inversions between ribosomal RNA genes of *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* **78**:7069–7072.
67. **Hirota, Y., and P. H. A. Sneath.** 1961. F' and F mediated transduction in *Escherichia coli* K-12. *Jpn. J. Genet.* **36**:307–318.
68. **Hoekstra, W. P. M., and H. G. Vis.** 1977. Characterization of the *Escherichia coli* K-12 strain AB1157 as impaired in guanine/xanthine metabolism. *Antonie van Leeuwenhoek J. Microbiol. Serol.* **43**:199–204.
69. **Horii, Z. I., and A. J. Clark.** 1973. Genetic analysis of the RecF pathway to genetic recombination in *Escherichia coli* K-12: isolation and characterization of mutants. *J. Mol. Biol.* **80**:327–344.
70. **Howard-Flanders, P.** 1968. Genes that control DNA repair and genetic recombination in *Escherichia coli*. *Adv. Biol. Med. Phys.* **12**:299–317.
71. **Howard-Flanders, P., R. P. Boyce, and L. Theriot.** 1966. Three loci in *Escherichia coli* K-12 that control the excision of pyrimidine dimers and certain other mutagen products from DNA. *Genetics* **53**:1119–1136.
72. **Howard-Flanders, P., E. Simson, and L. Theriot.** 1964. A locus that controls filament formation and sensitivity to radiation in *Escherichia coli* K-12. *Genetics* **49**:237–246.
73. **Howard-Flanders, P., and L. Theriot.** 1966. Mutants of *Escherichia coli* K-12 defective in DNA repair and in genetic recombination. *Genetics* **53**:1137–1150.
74. **Ishibashi, M., Y. Sugino, and Y. Hirota.** 1964. Chromosomal location of thymine and arginine genes in *Escherichia coli* and an F' incorporating them. *J. Bacteriol.* **87**:554–561.
75. **Jacob, F., S. Brenner, and F. Cuzin.** 1963. On the regulation of DNA replication in bacteria. *Cold Spring Harbor Symp. Quant. Biol.* **28**:329–348.
76. **Jacob, F., and E. L. Wollman.** 1956. Sur les processus de conjugaison et de recombinaison chez *Escherichia coli*. 1. L'induction par conjugaison ou induction zygotique. *Ann. Inst. Pasteur (Paris)* **91**:486–510.
77. **Jacob, F., and E. L. Wollman.** 1956. Recombinaison génétique et mutants de fertilité chez *Escherichia coli*. *C.R. Acad. Sci.* **242**:303–306.
78. **Jacob, F., and E. L. Wollman.** 1957. Analyse des groupes de liaison génétique de différentes souches donatrices d'*Escherichia coli*. *C.R. Acad. Sci.* **245**:1840–1843.
79. **Jacob, F., and E. L. Wollman.** 1958. Genetic and physical determinations of chromosomal segments in *Escherichia coli*. *Symp. Soc. Exp. Biol.* **12**:75–92.
80. **Jacob, F., and E. L. Wollman.** 1961. *Sexuality and the Genetics of Bacteria*. Academic Press, Inc., New York.
81. **Jensen, K. F.** 1993. The *Escherichia coli* K-12 “wild types” W3110 and MG1655 have an *rph* frameshift mutation that leads to pyrimidine starvation due to low *pyrE* expression levels. *J. Bacteriol.* **175**:3401–3407.
82. **Joset, F., B. Low, and R. Krisch.** 1964. Induction by radiation of a new direction of chromosome transfer during conjugation in an Hfr strain of *Escherichia coli*. *Biochem. Biophys. Res. Commun.* **17**:742–747.
83. **Koch, J. P., S. I. Hayashi, and E. C. C. Lin.** 1964. The control of dissimilation of glycerol and L- α -glycerophosphate in *Escherichia coli*. *J. Biol. Chem.* **239**:3106–3108.
84. **Laffler, T., and J. Gallant.** 1974. *spoT*, a new genetic locus involved in the stringent response in *E. coli*. *Cell* **1**:27–30.
85. **Lederberg, E. M.** 1950. Lysogenicity of *Escherichia coli* strain K-12. *Microb. Genet. Bull.* **1**:5–7.
86. **Lederberg, E. M.** 1951. Lysogenicity of *E. coli* K-12. *Genetics* **36**:560.
87. **Lederberg, E. M.** 1952. Allelic relationships and reverse mutation in *Escherichia coli*. *Genetics* **37**:469–483.
88. **Lederberg, E. M.** 1954. The inheritance of lysogenicity in interstrain crosses of *Escherichia coli*. *Genetics* **39**:978.

89. **Lederberg, E. M., and J. Lederberg.** 1953. Genetic studies of lysogenicity in *Escherichia coli*. *Genetics* **38**:51–64.
90. **Lederberg, J.** 1947. Gene recombination and linked segregations in *Escherichia coli*. *Genetics* **32**:505–525.
91. **Lederberg, J.** 1949. Aberrant heterozygotes in *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* **35**:178–184.
92. **Lederberg, J.** 1950. The selection of genetic recombinations with bacterial growth inhibitors. *J. Bacteriol.* **59**:211–215.
93. **Lederberg, J.** 1951. Genetic studies with bacteria, p. 263–289. In L. C. Dunn (ed.), *Genetics in the 20th Century*. Macmillan, New York.
94. **Lederberg, J.** 1951. Streptomycin resistance: a genetically recessive mutation. *J. Bacteriol.* **61**:549–550.
95. **Lederberg, J.** 1951. Prevalence of *Escherichia coli* strains exhibiting genetic recombination. *Science* **114**:68–69.
96. **Lederberg, J., L. L. Cavalli, and E. M. Lederberg.** 1952. Sex compatibility in *Escherichia coli*. *Genetics* **37**:720–730.
97. **Lederberg, J., E. M. Lederberg, N. D. Zinder, and E. R. Lively.** 1951. Recombination analysis of bacterial heredity. *Cold Spring Harbor Symp. Quant. Biol.* **16**:413–443.
98. **Lederberg, J., and E. L. Tatum.** 1946. Novel genotypes in mixed cultures of biochemical mutants of bacteria. *Cold Spring Harbor Symp. Quant. Biol.* **116**:113–114.
99. **Lederberg, J., and E. L. Tatum.** 1946. Gene recombination in *Escherichia coli*. *Nature (London)* **158**:558.
100. **Lederberg, J., and E. L. Tatum.** 1953. Sex in bacteria: genetic studies, 1945–1952. *Science* **118**:169–175.
101. **Lengeler, J., and H. Steinberger.** 1978. Analysis of regulatory mechanisms controlling the activity of the hexitol transport systems in *Escherichia coli* K12. *Mol. Gen. Genet.* **167**:75–82.
102. **Liu, D., and P. R. Reeves.** 1994. *Escherichia coli* regains its O antigen. *Microbiology* **140**:49–57.
103. **Lloyd, R. G., and C. Buckman.** 1985. Identification and genetic analysis of *sbC* mutations in commonly used *recBC sbcB* strains of *Escherichia coli*. *J. Bacteriol.* **164**:836–844.
104. **Low, B.** 1967. Inversion of transfer modes and sex factor-chromosome interactions in conjugation in *Escherichia coli*. *J. Bacteriol.* **93**:98–106.
105. **Low, B.** 1968. Formation of merodiploids in matings with a class of Rec⁻ recipient strains of *Escherichia coli* K12. *Proc. Natl. Acad. Sci. USA* **60**:160–167.
106. **Low, B.** 1973. Restoration by the *rac* locus of recombinant forming ability in *recB⁻* and *recC⁻* merozygotes of *Escherichia coli* K12. *Mol. Gen. Genet.* **122**:119–130.
107. **Low, B.** 1973. Rapid mapping of conditional and auxotrophic mutants of *Escherichia coli* K12. *J. Bacteriol.* **113**:798–812.
108. **Lupo, M., and Y. S. Halpern.** 1970. Gene controlling L-glutamic acid decarboxylase synthesis in *Escherichia coli* K-12. *J. Bacteriol.* **103**:382–386.
109. **Luria, S. E., J. N. Adams, and R. C. Ting.** 1960. Transduction of lactose-utilizing ability among strains of *E. coli* and *S. dysenteriae* and the properties of the transducing phage particles. *Virology* **12**:348–390.
110. **Mahajan, S. K., N. N. Pandit, and J. F. Sarkarai.** 1984. Host functions in amplification/deamplification of Tn9 in *Escherichia coli* K-12: a new model for amplification. *Cold Spring Harbor Symp. Quant. Biol.* **49**:443–451.
111. **Matney, T. S., E. P. Goldschmidt, N. S. Erwin, and R. A. Scroggs.** 1964. A preliminary map of genomic sites for F-attachment in *Escherichia coli* K-12. *Biochem. Biophys. Res. Commun.* **17**:278–281.
112. **Meselson, M., and R. Yuan.** 1968. DNA restriction enzyme from *E. coli*. *Nature (London)* **217**:1110–1114.
113. **Metzer, E., R. Levitz, and Y. S. Halpern.** 1979. Isolation and properties of *Escherichia coli* K-12 mutants impaired in the utilization of γ -aminobutyrate. *J. Bacteriol.* **137**:1111–1118.

114. **Meynell, E., and N. Datta.** 1966. The relation of resistance transfer factors to the F-factor (sex-factor) of *Escherichia coli* K-12. *Genet. Res.* **7**:134–140.
115. **Morse, M. L., E. M. Lederberg, and J. Lederberg.** 1956. Transductional heterogenotes in *Escherichia coli*. *Genetics* **41**:758–779.
116. **Naas, T., M. Blot, W. M. Fitch, and W. Arber.** 1994. Insertion sequence-related genetic variation in resting *Escherichia coli* K-12. *Genetics* **136**:721–730.
117. **Natori, S., and A. Garen.** 1970. Molecular heterogeneity in the amino-terminal region of alkaline phosphatase. *J. Mol. Biol.* **49**:577–588.
118. **Newton, W. A., J. R. Beckwith, D. Zipser, and S. Brenner.** 1965. Nonsense mutants and polarity in the *lac* operon of *Escherichia coli*. *J. Mol. Biol.* **14**:290–296.
119. **Nikaido, H., K. Nikaido, and A. M. C. Rapin.** 1965. Biosynthesis of thymidine diphosphate L-rhamnose in *Escherichia coli* K-12. *Biochim. Biophys. Acta* **111**:548–551.
120. **Nunn, W. D., and R. W. Simons.** 1978. Transport of long-chain fatty acids by *Escherichia coli*: mapping and characterization of mutants in the *fadL* gene. *Proc. Natl. Acad. Sci. USA* **75**:3377–3381.
121. **Okada, T., K. Yanagisawa, and F. J. Ryan.** 1960. Elective production of thymine-less mutants. *Nature (London)* **188**:340–341.
122. **Okada, T., K. Yanagisawa, and F. J. Ryan.** 1961. A method for securing thymineless mutants from strains of *E. coli*. *Z. Vererbungsl.* **92**:403–412.
123. **Orskov, F., and I. Orskov.** 1961. The fertility of *Escherichia coli* antigen test strains in crosses with K12. *Acta Pathol. Mikrobiol. Scand.* **51**:280–290.
124. **Orskov, F., and I. Orskov.** 1962. Behavior of *E. coli* antigens in sexual recombination. *Acta Pathol. Mikrobiol. Scand.* **95**:99–109.
125. **Orskov, I., and F. Orskov.** 1960. The H antigen of the “K12” strain. A new *E. coli* H antigen: H48. *Acta Pathol. Mikrobiol. Scand.* **48**:47.
126. **Pardee, A. B., F. Jacob, and J. Monod.** 1959. The genetic control and cytoplasmic expression of “inducibility” in the synthesis of β -galactosidase by *E. coli*. *J. Mol. Biol.* **1**:165–178.
127. **Pratt, D., and W. S. Erdahl.** 1968. Genetic control of bacteriophage M13 DNA synthesis. *J. Mol. Biol.* **37**:181–200.
128. **Raleigh, E. A., R. Trimachi, and H. Revel.** 1989. Genetic and physical mapping of the *mcrA* (*rglA*) and *mcrB* (*rglB*) loci of *Escherichia coli* K-12. *Genetics* **122**:279–296.
129. **Raleigh, E. A., N. E. Murray, H. Revel, R. M. Blumenthal, D. Westaway, A. D. Reith, P. W. J. Rigby, J. Elhai, and D. Hanahan.** 1988. McrA and McrB restriction phenotypes of some *E. coli* strains and implications for gene cloning. *Nucleic Acids Res.* **16**:1563–1575.
130. **Richter, A.** 1957. Complementary determinants of an Hfr phenotype in *E. coli* K12. *Genetics* **42**:391.
131. **Richter, A. A.** 1958. Recombination analysis of mating type in *Escherichia coli* K-12, p. 232. *In Proc. 10th Int. Congr. Genet.*, vol. 2.
132. **Richter, A.** 1961. Attachment of wild type F factor to a specific chromosomal region in a variant strain of *Escherichia coli* K12: the phenomenon of episomic alternation. *Genet. Res.* **2**:333–345.
133. **Rickenberg, H. V., and G. Lester.** 1955. The preferential synthesis of beta-galactosidase in *Escherichia coli*. *J. Gen. Microbiol.* **13**:279–284.
134. **Roberton, A. M., P. A. Sullivan, M. C. Jones-Mortimer, and H. L. Kornberg.** 1980. Two genes affecting glucarate utilization in *Escherichia coli*. *J. Gen. Microbiol.* **117**:377–382.
135. **Rothfels, K. H.** 1952. Gene linearity and negative interference in crosses of *Escherichia coli*. *Genetics* **37**:297–311.
136. **Scaife, J., and A. P. Pekhov.** 1964. Deletion of chromosomal markers in association with F-prime factor formation in *Escherichia coli* K-12. *Genet. Res.* **5**:495–498.
137. **Schnaitman, C. A.** 1974. Outer membrane proteins of *Escherichia coli*. IV. Differences in outer membrane proteins due to strain and culture differences. *J. Bacteriol.* **118**:454–464.
138. **Schwartz, M.** 1966. Location of the maltose A and B loci on the genetic map of *Escherichia coli*. *J. Bacteriol.* **92**:1083–1089.
139. **Signer, E. R., J. R. Beckwith, and S. Brenner.** 1965. Mapping of suppressor loci in *Escherichia*

- coli*. *J. Mol. Biol.* **14**:153–166.
140. **Skaar, P. D.** 1956. A binary mutability system in *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* **42**:245–249.
141. **Skaar, P. D., and A. Garen.** 1956. The orientation and extent of gene transfer in *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* **42**:619–624.
142. **Skaar, P. D., A. Richter, and J. Lederberg.** 1957. Correlated selection for motility and sex incompatibility in *Escherichia coli* K12. *Proc. Natl. Acad. Sci. USA* **43**:329–333.
143. **Smith, H. W.** 1975. Survival of orally administered *E. coli* K12 in alimentary tract of man. *Nature* (London) **255**:500–502.
144. **Sneath, P. H. A.** 1962. Sex factors as episomes. *Br. Med. Bull.* **18**:41–45.
145. **Stent, G. S., and S. Brenner.** 1961. A genetic locus for the regulation of ribonucleic acid synthesis. *Proc. Natl. Acad. Sci. USA* **47**:2005–2014.
146. **Stevenson, G., B. Neal, D. Liu, M. Hobbs, N. H. Packer, M. Batley, J. W. Redmond, L. Lindquist, and P. Reeves.** 1994. Structure of the O antigen of *Escherichia coli* K-12 and the sequence of its *rfb* gene cluster. *J. Bacteriol.* **176**:4144–4156.
147. **Sung, Y.-C., D. Parsell, P. M. Anderson, and J. A. Fuchs.** 1987. Identification, mapping, and cloning of the gene encoding cyanase in *Escherichia coli* K-12. *J. Bacteriol.* **169**:2639–2642.
148. **Suzuki, T., and A. Garen.** 1969. Fragments of alkaline phosphatase from nonsense mutants. I. Isolation and characterization of fragments from amber and ochre mutants. *J. Mol. Biol.* **45**:549–566.
149. **Tatum, E. L.** 1945. X-ray induced mutant strains of *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* **31**:215–219.
150. **Tatum, E. L., and J. Lederberg.** 1947. Gene recombination in the bacterium *Escherichia coli*. *J. Bacteriol.* **53**:673–684.
151. **Taylor, A. L., and E. A. Adelberg.** 1960. Linkage analysis with very high frequency males of *Escherichia coli*. *Genetics* **45**:1233–1243.
152. **Taylor, M., and C. Yanofsky.** 1964. Transformation of bacterial markers and transfer of phage markers with DNA isolated from λ - ϕ -80 hybrid phage carrying the tryptophan genes of *E. coli*. *Biochem. Biophys. Res. Commun.* **17**:798–804.
153. **Tommassen, J., and B. Lugtenberg.** 1981. Localization of *phoE*, the structural gene for outer membrane protein e in *Escherichia coli* K-12. *J. Bacteriol.* **147**:118–123.
154. **Treffers, H. P., V. Spinelli, and N. O. Belser.** 1954. A factor (or mutator gene) influencing mutation rates in *Escherichia coli*. *Proc. Natl. Acad. Sci. USA* **40**:1064–1071.
155. **Utsumi, R., Y. Nakamoto, M. Kawamukai, M. Himeno, and T. Komano.** 1982. Involvement of cyclic AMP and its receptor protein in filamentation of an *Escherichia coli* *fic* mutant. *J. Bacteriol.* **151**:807–812.
156. **Wanner, B. L.** 1987. Control of *phoR*-dependent bacterial alkaline phosphatase clonal variation by the *phoM* region. *J. Bacteriol.* **169**:900–903.
157. **Wanner, B. L., and M. R. Wilmes-Riesenberg.** 1992. Involvement of phosphotransacetylase, acetate kinase, and acetyl phosphate synthesis in control of the phosphate regulon in *Escherichia coli*. *J. Bacteriol.* **174**:2124–2130.
158. **Watson, J. D., and W. Hayes.** 1953. Genetic exchange in *Escherichia coli* K-12: evidence for three linkage groups. *Proc. Natl. Acad. Sci. USA* **39**:416–426.
159. **Wechsler, J. A., and J. D. Gross.** 1971. *Escherichia coli* mutants temperature-sensitive for DNA synthesis. *Mol. Gen. Genet.* **113**:273–284.
160. **Weigert, M. G., and A. Garen.** 1965. Amino acid substitutions resulting from suppression of nonsense mutations. I. Serine insertion by the *Su-1* suppressor gene. *J. Mol. Biol.* **12**:448–455.
161. **Weigert, M. G., E. Lanka, and A. Garen.** 1965. Amino acid substitutions resulting from suppression of nonsense mutations. II. Glutamine insertion by the *Su-2* gene; tyrosine insertion by the *Su-3* gene. *J. Mol. Biol.* **14**:522–527.
162. **Wiesmeyer, H., and M. Cohn.** 1960. The characterization of the pathway of maltose utilization by *Escherichia coli*. III. A description of the concentrating mechanism. *Biochim. Biophys. Acta* **39**:440–447.

163. **Willets, N. S., and A. J. Clark.** 1969. Characteristics of some multiply recombination-deficient strains of *Escherichia coli*. *J. Bacteriol.* **100**:231–239.
164. **Willets, N. S., A. J. Clark, and B. Low.** 1969. Genetic locations of certain mutations conferring recombination deficiency in *Escherichia coli*. *J. Bacteriol.* **97**:244–249.
165. **Willets, N. S., and D. W. Mount.** 1969. Genetic analysis of recombination-deficient mutants of *Escherichia coli* K-12 carrying *rec* mutations cotransducible with *thyA*. *J. Bacteriol.* **100**:923–934.
166. **Willsky, G. R., and M. H. Malamy.** 1976. Control of the synthesis of alkaline phosphatase and the phosphate-binding protein in *Escherichia coli*. *J. Bacteriol.* **127**:595–609.
167. **Wollman, E. L.** 1953. Sur le déterminisme génétique de la lysogénie. *Ann. Inst. Pasteur (Paris)* **84**:281–293.
168. **Wollman, E. L., and F. Jacob.** 1957. Sur les processus de conjugaison et de recombinaison chez *E. coli*. II. La localisation chromosomique du prophage λ et les conséquences génétique de l'induction zygotique. *Ann. Inst. Pasteur (Paris)* **93**:323–339.
169. **Wollman, E. L., F. Jacob, and W. Hayes.** 1956. Conjugation and genetic recombination in *Escherichia coli* K-12. *Cold Spring Harbor Symp. Quant. Biol.* **21**:141–162.
170. **Wood, W. B.** 1966. Host specificity of DNA produced by *Escherichia coli*: bacterial mutations affecting the restriction and modification of DNA. *J. Mol. Biol.* **16**:118–133.
171. **Yanofsky, C., and J. Ito.** 1966. Nonsense codons and polarity in the tryptophan operon. *J. Mol. Biol.* **21**:313–334.