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Pedigrees of Some Mutant Strains of *Escherichia coli* K-12

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INTRODUCTION

The strain K-12 of *Escherichia coli* has been in cultivation in the laboratory for 50 years now. It was isolated from the stool of a convalescent diphtheria patient in the fall of 1922 (64). For many years therafter, it served as a standard culture in the bacteriology department of Stanford University and was used extensively in the teaching laboratories there (99). That this virile strain of *E. coli*, one of the relatively few found to possess significant fertility in the laboratory (66, 81*b*, 81*c*), should have been the one which C. E. Clifton chose to give to E. L. Tatum as the latter set out to produce mutant strains of bacteria was apparently just a particularly happy accident. K-12 was thought to be an entirely typical coli culture (but see the work of the Orskovs [81*a*, 81*b*, 81*c*] regarding its antigenic structure.)

Gray and Tatum reported the isolation of X-ray-induced auxotrophic mutants of bacteria, including *E. coli* K-12, in 1944 (35). These same strains, and others, were used by Lederberg and Tatum in their early studies on genetic recombination in bacteria (69, 70). Since that time, literally thousands of mutant strains of K-12 have been produced. Their contribution to the development of molecular biology is by now well documented and widely appreciated. In the belief that there is still much to be learned from the study of this microbe, we have been involved, over the past few years, in setting up a center for the preservation and dissemination of genetic stock cultures of this organism.

When the *E. coli* Genetic Stock Center was first set up and we began acquiring strains from other laboratories, it became apparent that we were receiving the same mutational event (mutant allele) in a variety of strains under a variety of mutant allele designations from different laboratories. We soon realized that, in order to assign meaningful and unambiguous mutant allele designations to the mutations carried in the strains in the collection, it would

be necessary to trace the derivation of all incoming strains. The donors of the cultures were unable to give us complete derivations for their strains in most cases, and it proved to be impossible to trace them completely through the published literature. We therefore set out to trace the strains through the unpublished records of the laboratories in which they were made. This task, which gradually became the major effort in setting up the Stock Center, has led finally to a reconstruction of much of the history of *E. coli* K-12. Some of the results of this project, which is still going on, are presented here.

The pedigrees of K-12 derivatives that are presented here have been chosen with the following considerations in mind. We have tried to include the derivation of most of the very early strains of the Stanford, Yale, Wisconsin, and Paris laboratories, which served as ancestral stocks for almost all other collections and would thus be of the widest possible interest. Among later strains, we have included those that seem to have been most widely used as ancestors. And among contemporaneous strains, we have again chosen those that appear to be widely used in constructing strains for genetic analysis, based in large part on the frequency of requests received by the Stock Center. Obvious limitations influencing our selections have been our lack of experience with strains from some major collections that we have not yet explored and, in some cases, our inability to establish the history of important strains due to gaps in laboratory records.

SOURCES OF THE DATA

The strain pedigrees are presented in Charts 1 through 11. The documentation for these diagrams is given in Table 1, under the strain designations, listed in alphanumerical order.

The ultimate sources of the data were, in most cases, the laboratory records (strain notebooks and strain cards) of the laboratories in which the strains were made. I visited these laboratories and worked with their records in constructing the strain pedigrees. I wish to assume full responsibility for any misinterpretations applied to these data, while acknowledging considerable generous assistance from the "owners" of the records. In cases of conflict with reports published in the literature, these laboratory records were accepted as being correct. These major unpublished sources of data 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 notebook of A. L. Taylor.

E, Strain records of M. L. Morse.

F, Strain list of P. Howard-Flanders.

In other cases, as well, I have called upon the assistance of those who constructed the strains. These investigators, in response to questions, either generously searched their laboratory records and extracted the essential information, or verified pedigrees which I had constructed on the basis of published information and experience with the strains in question. These extensive personal communications are acknowledged as sources of data in Table 1, lower case letters designating data received from these investigators, as follows: (a) R. Appleyard, (b) A. J. Clark, (c) B. D. Davis, (d) A. Garen, (e) W. Hayes, (f) K. B. Low, (g) W. Maas, (h) P. Reeves, (i) P. Treffers (j) N. Willetts, (k) E. Wollman, (l) C. Yanofsky.

In addition to the above unpublished sources of data, we have included in Table 1 references to published descriptions of strains and their derivations. These sources are listed under Literature Cited. In a few cases, where published reports were sufficiently detailed or where we were unable to reach more direct sources, citations to the literature are used as sole documentation for the pedigrees and strain descriptions.

PEDIGREE CHARTS

Conventions

The pedigree charts consist of strain descriptions with lines of descent indicated by arrows. The genetic step involved in the production of most of the strains was mutation, either spontaneous or induced. The mutagenic or selective agents used are indicated beside the arrows. 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. In the cases 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, in all cases, the original strain designations assigned by those who constructed the strains. Widely used syno-

CHART 1. *Some early Stanford and Yale strains*

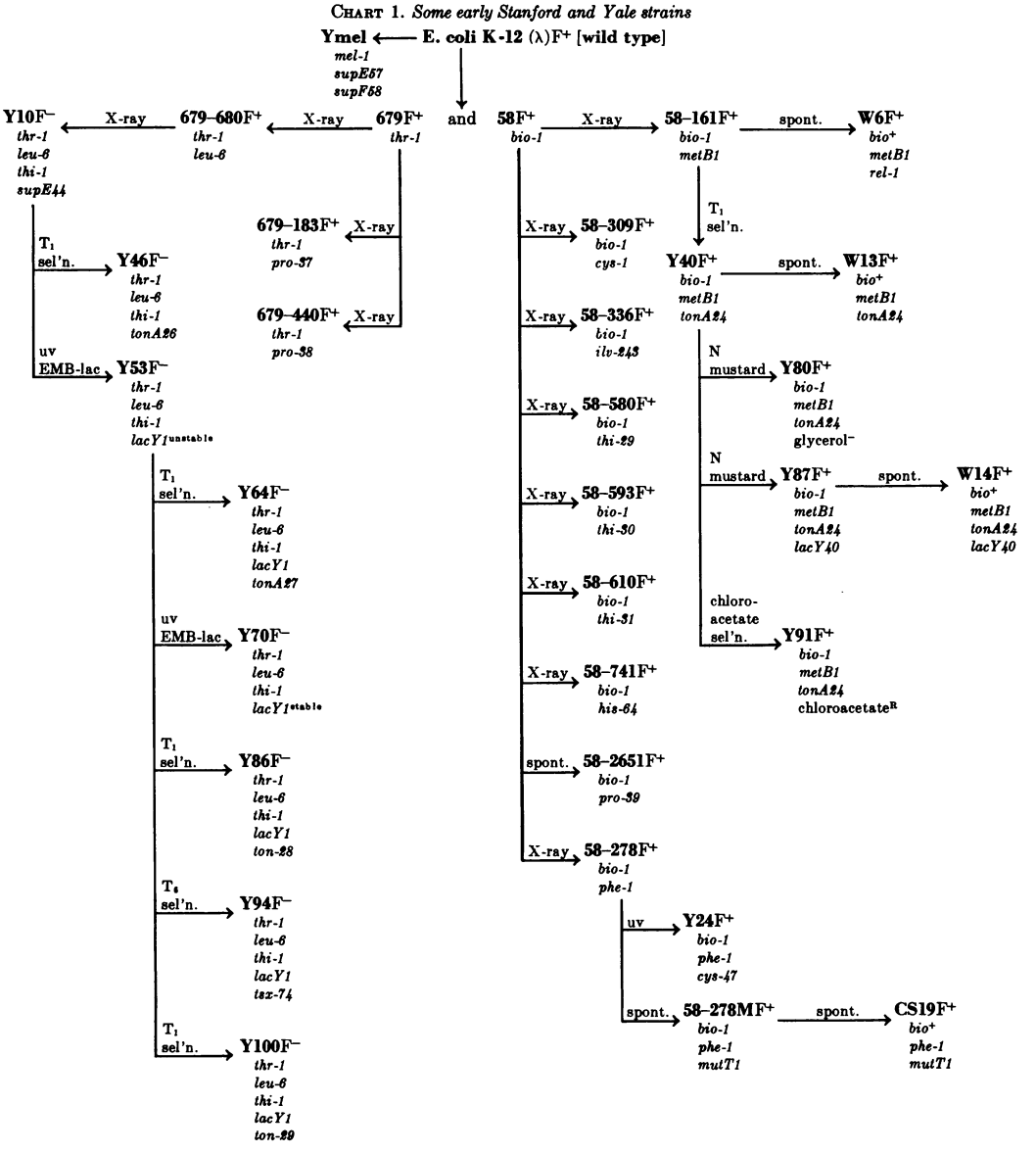

CHART 2. *Some derivatives of strain Y10*

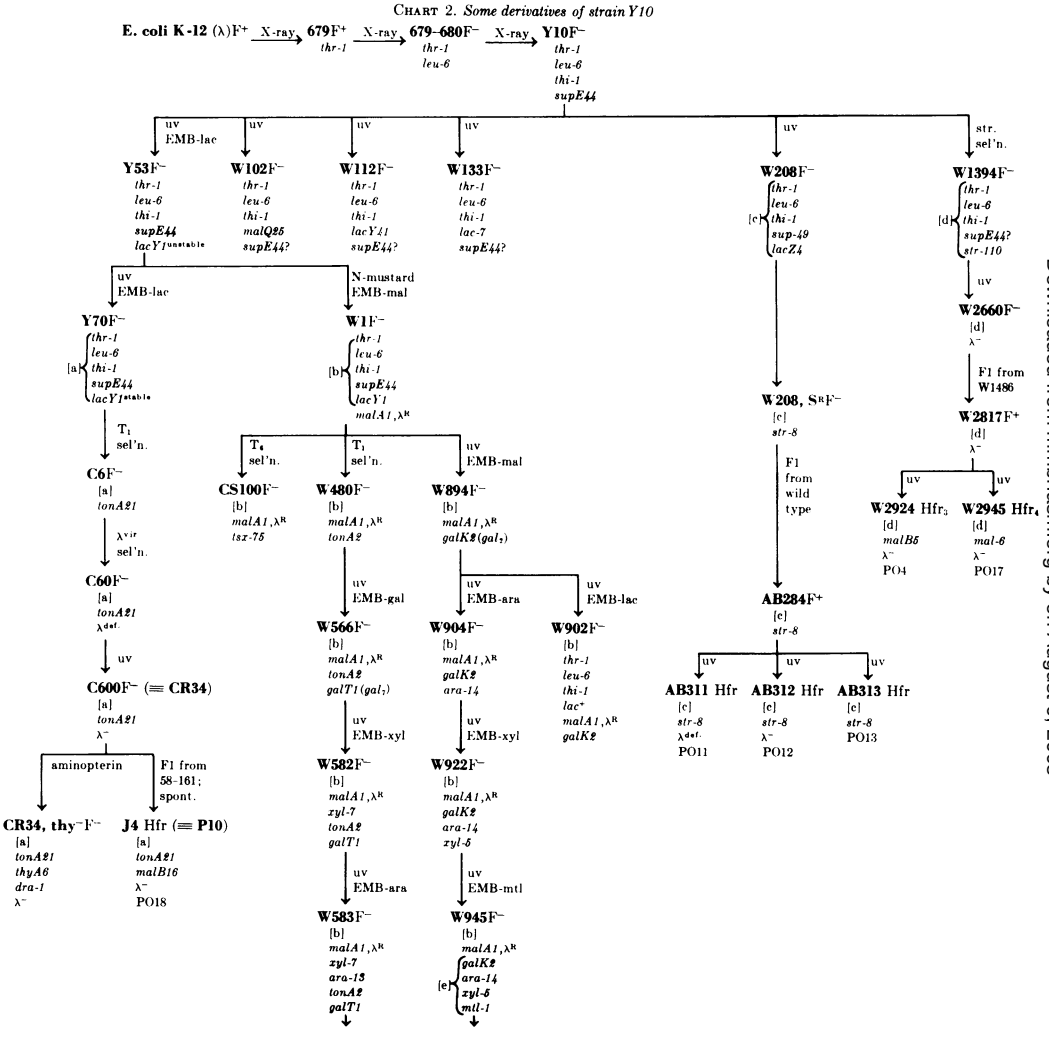

CHART 2. (*cont'd*)

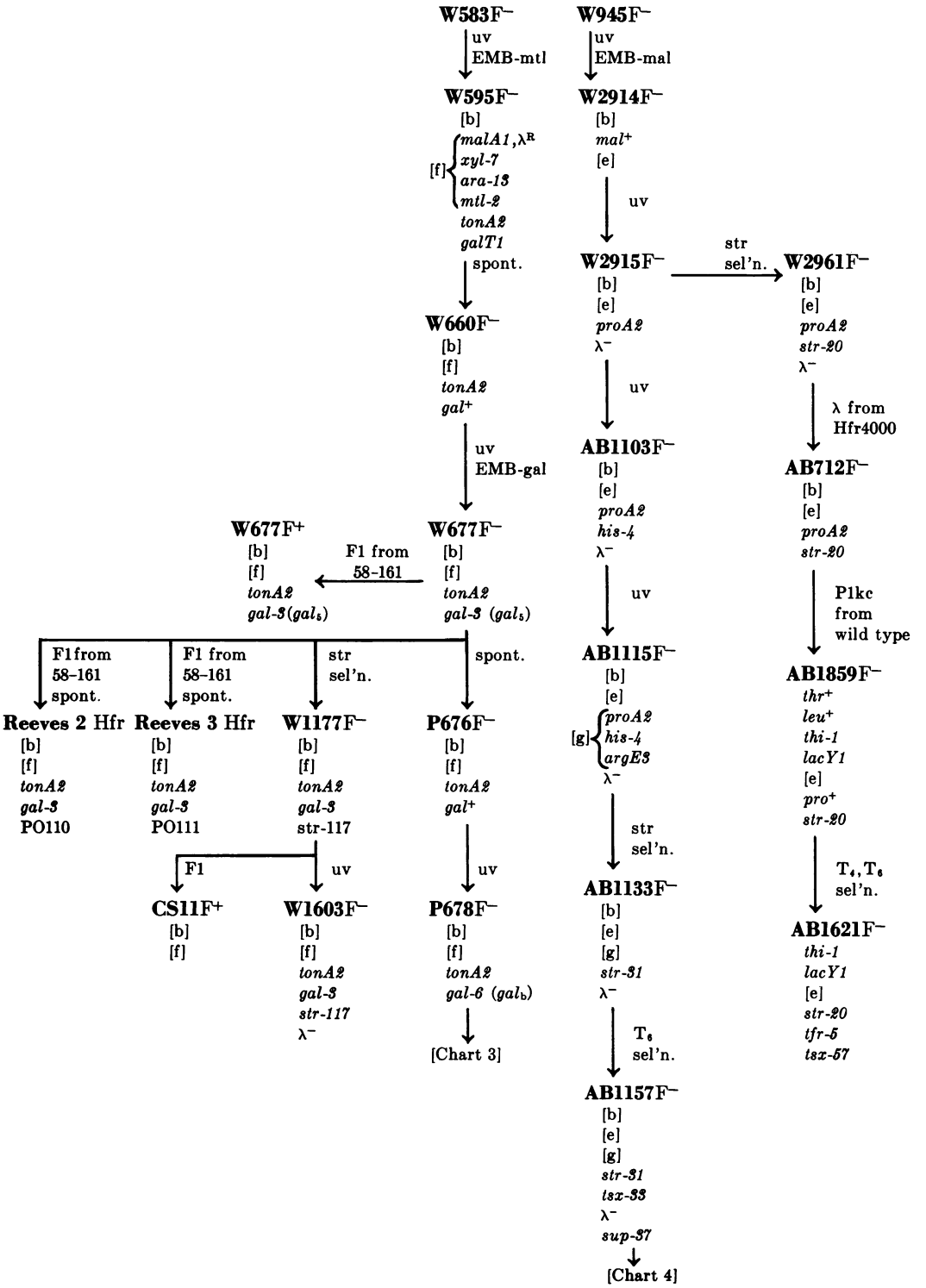

CHART 3. *Some derivatives of strain P678*

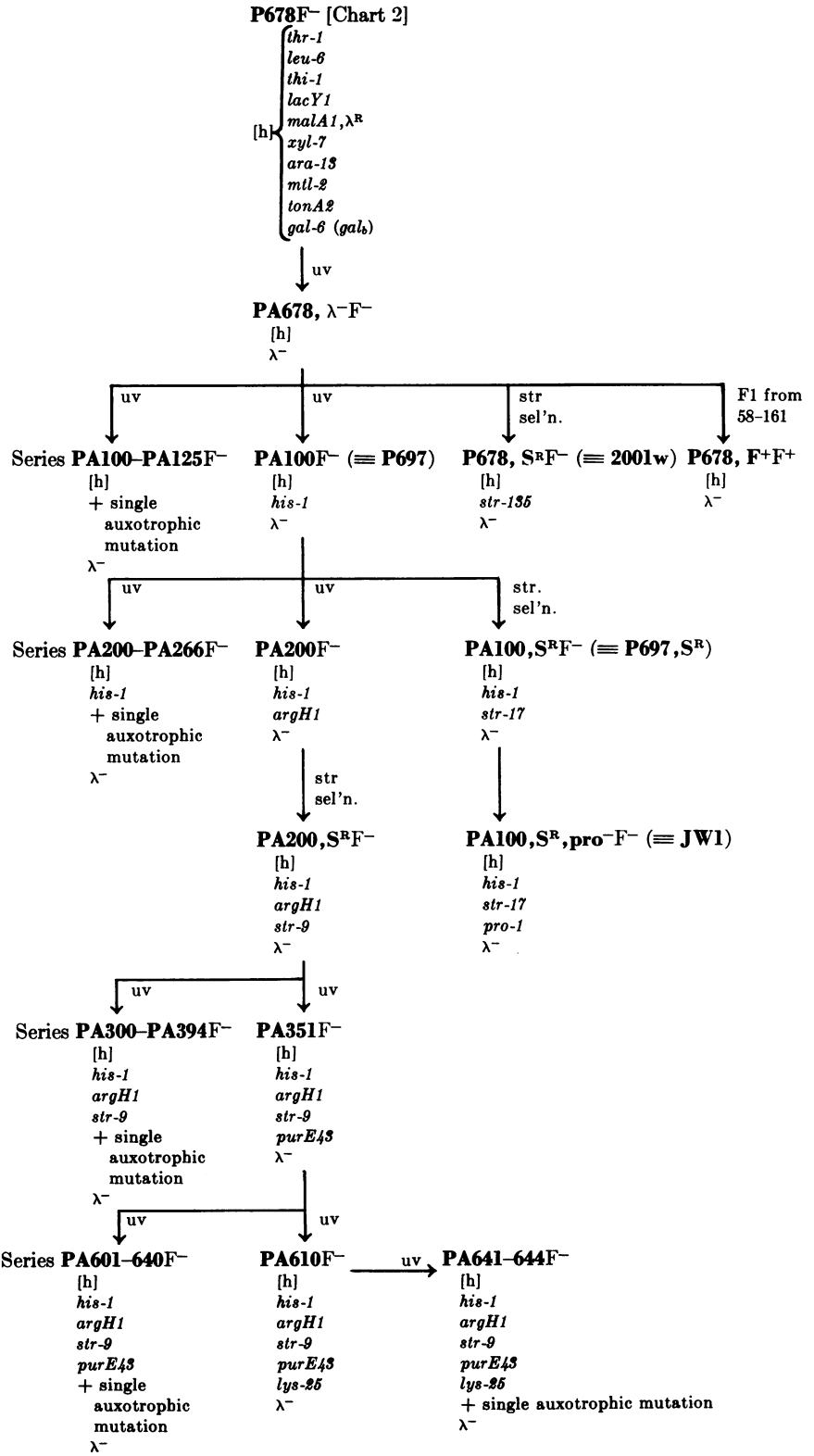

CHART 4. *Some derivatives of strain AB1157*

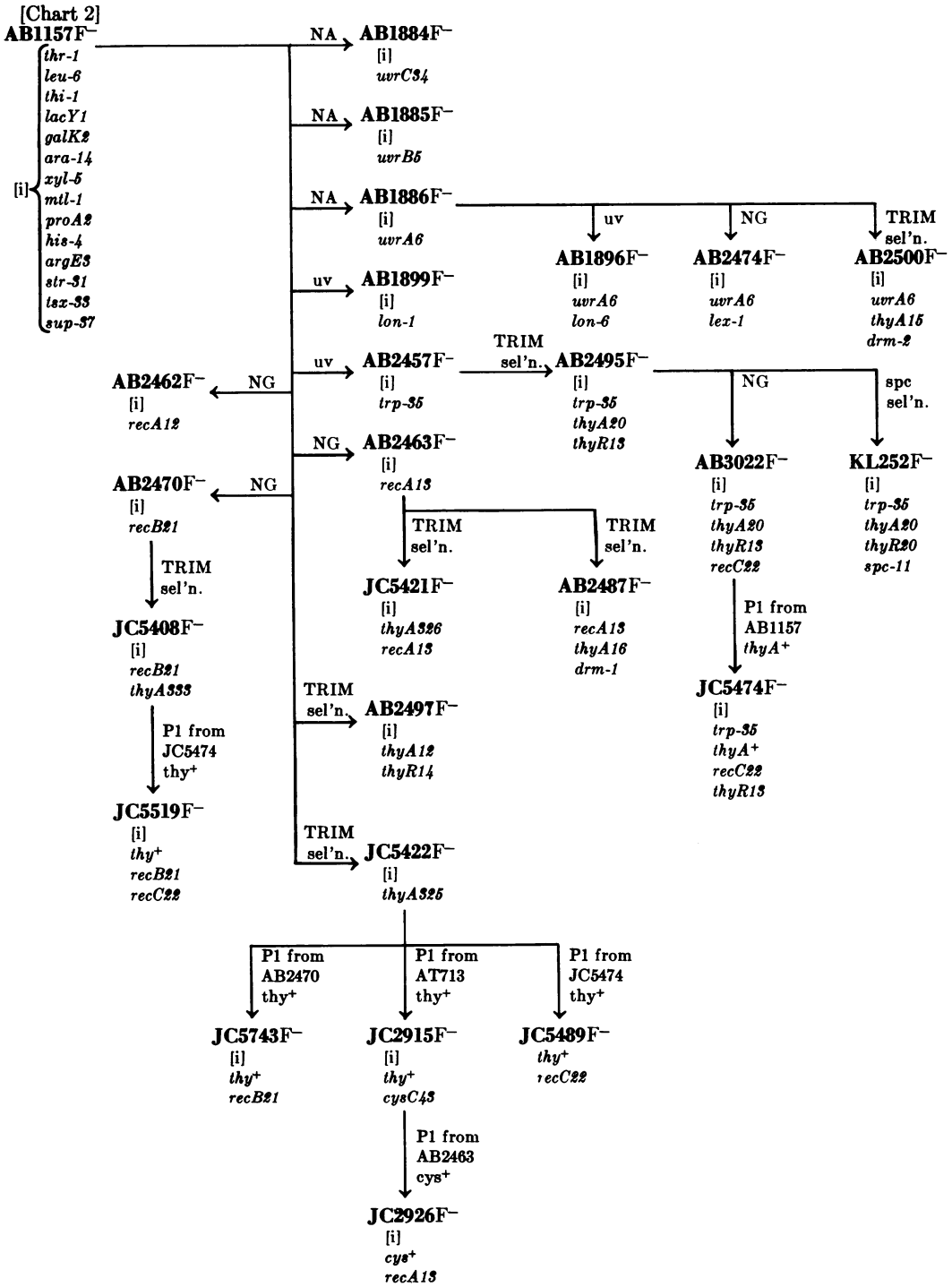

CHART 5. *Some derivatives of strains 58–161 and W6*

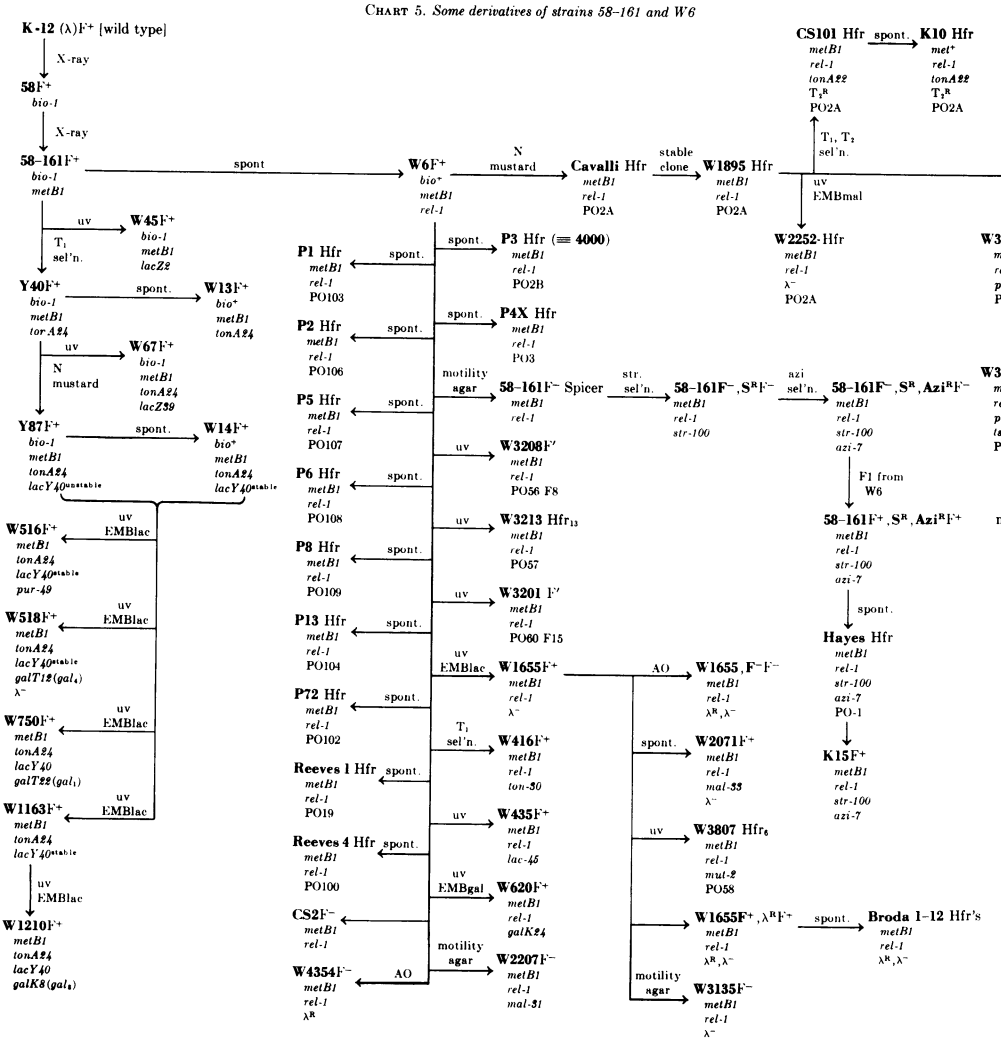

CHART 6. *Hfr H thi⁻,λ⁻ and some of its derivatives*

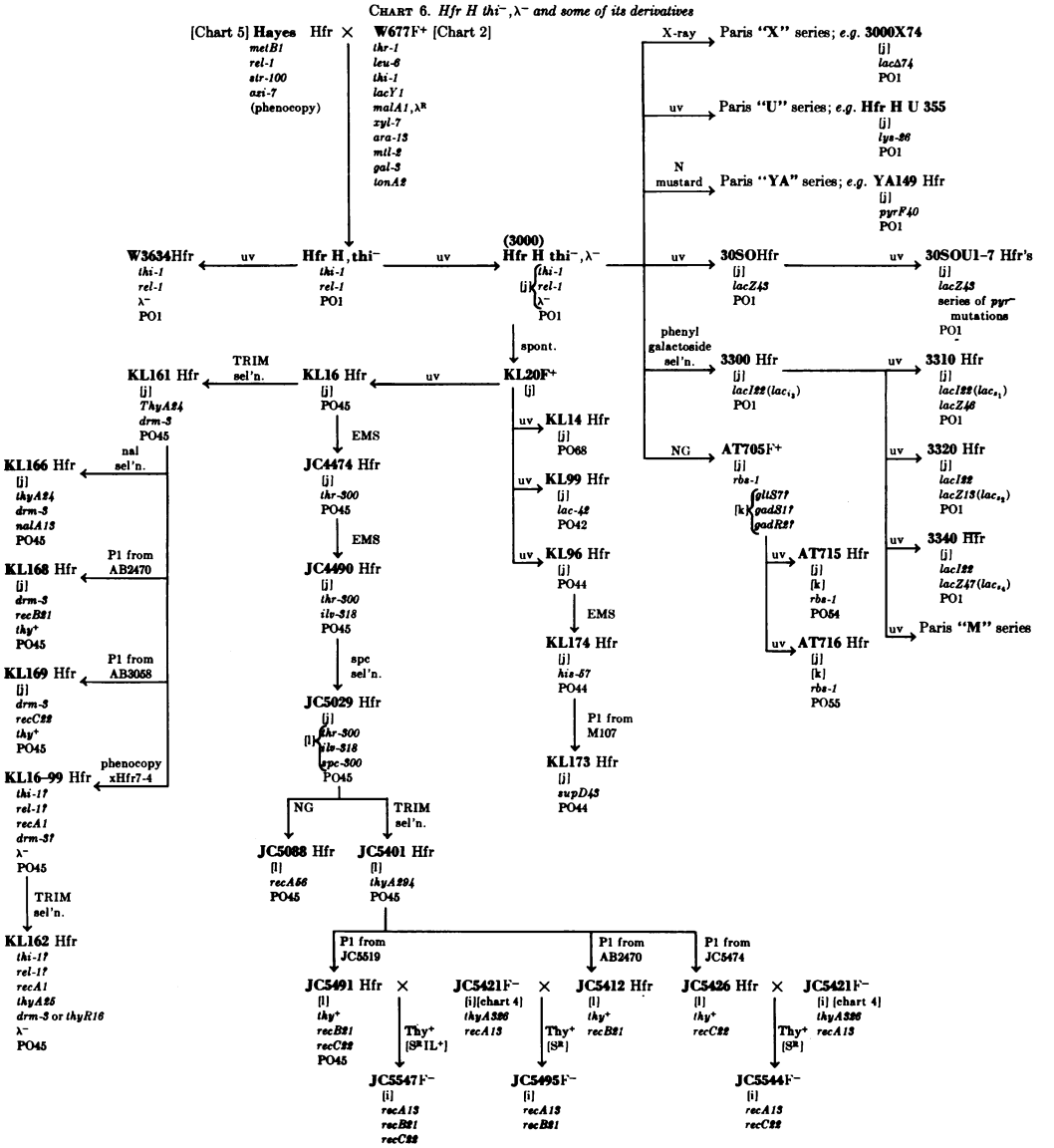

CHART 7. *Some of the early Paris lac⁻ strains*

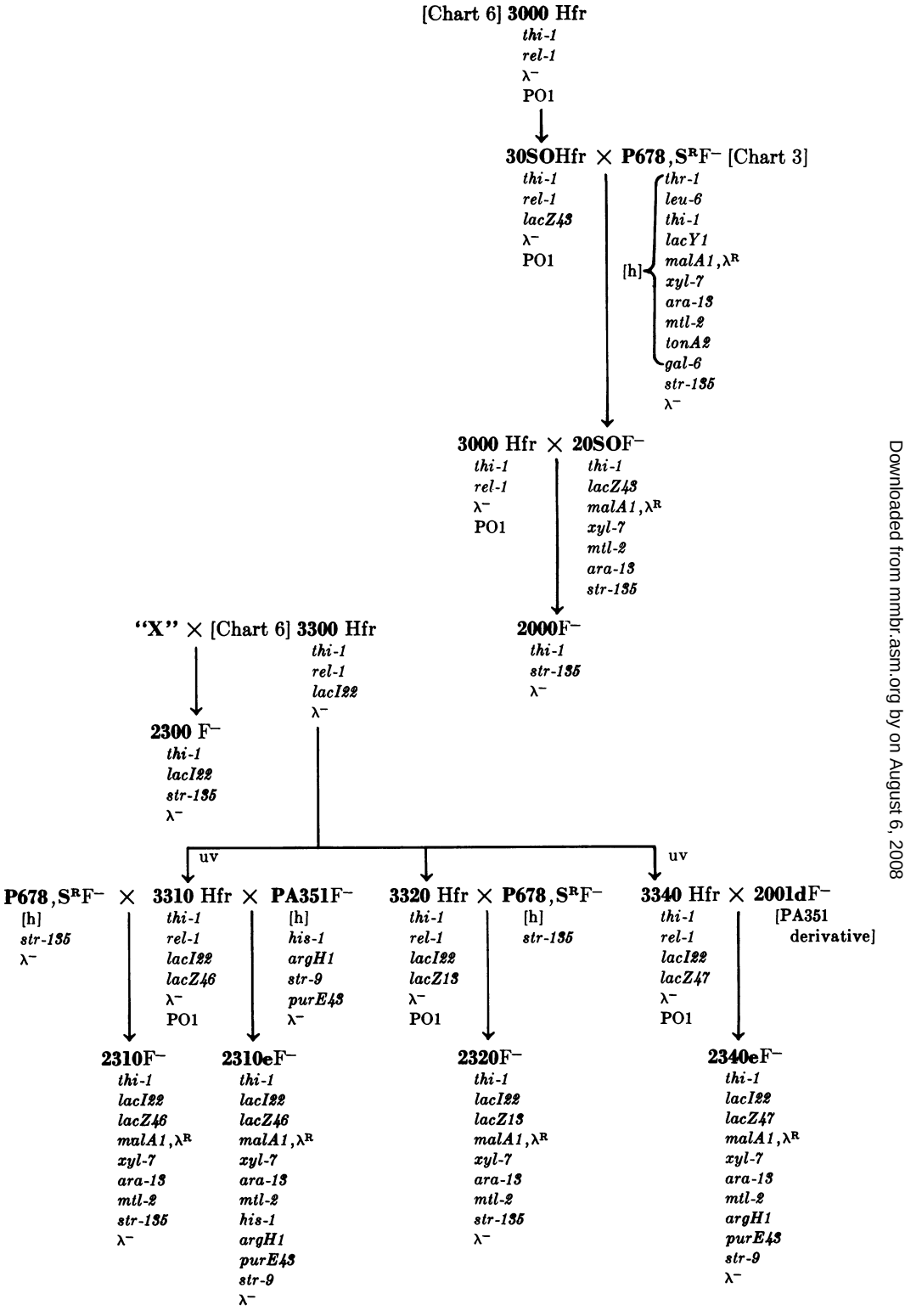

CHART 8. *Other lines derived from wild type*

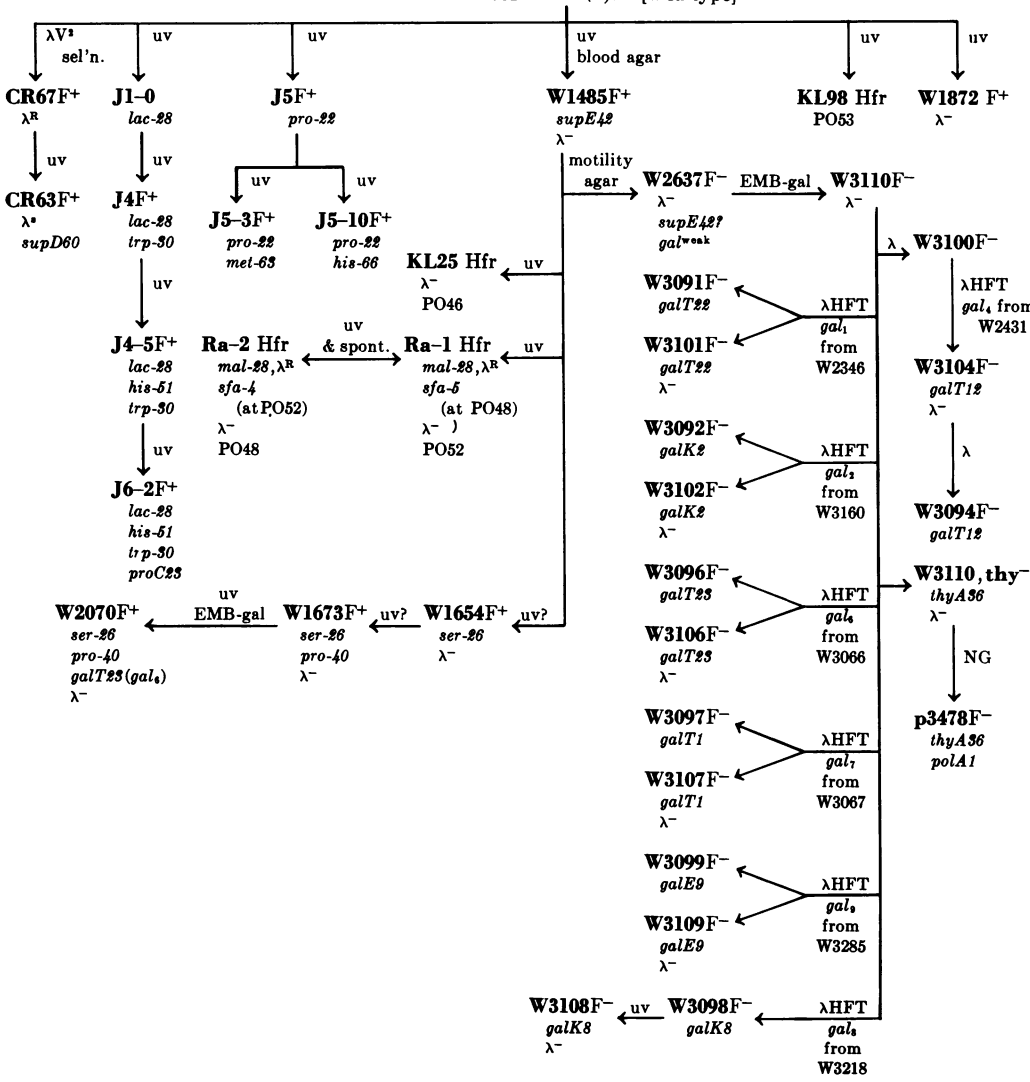

CHART 9. *The derivation of JC12 and JC411 and some of their derivatives*

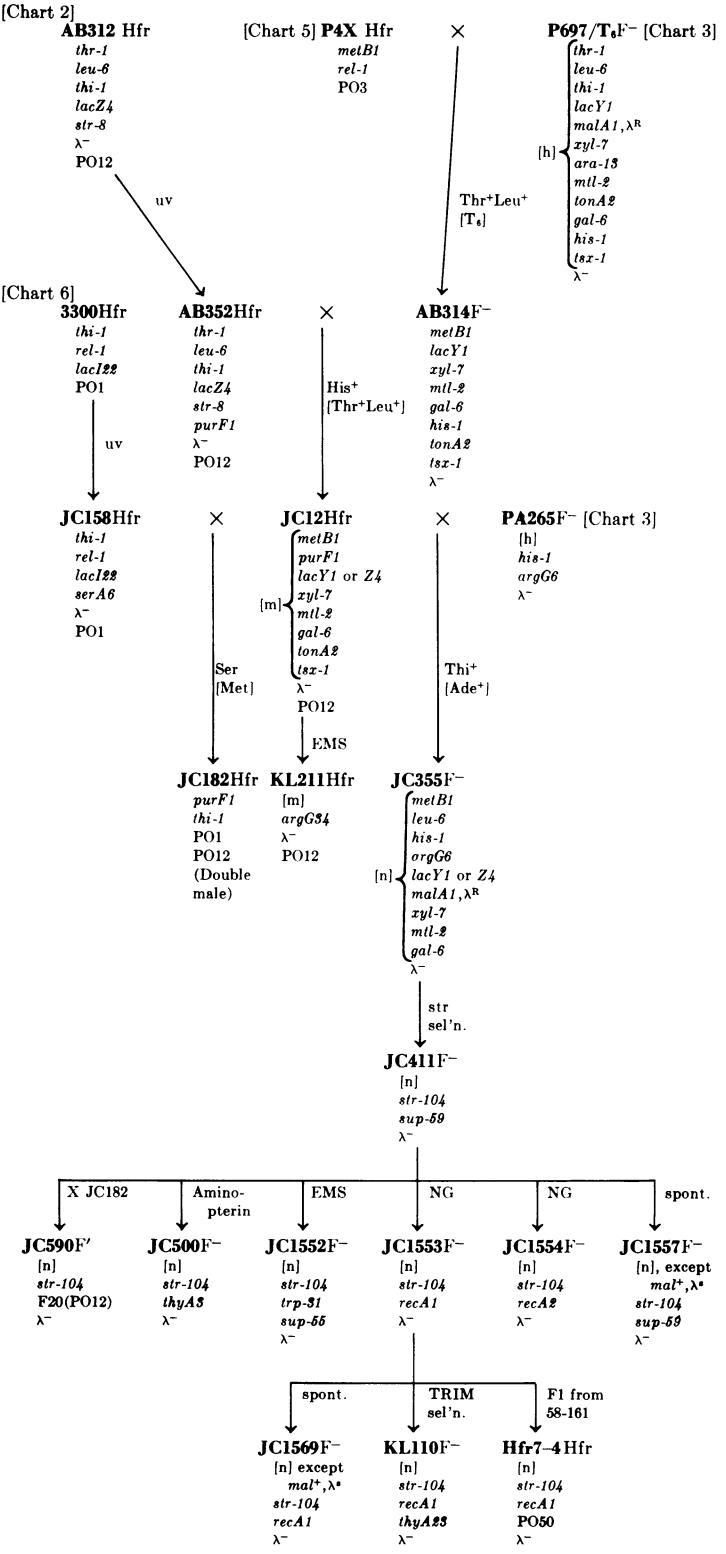

CHART 10. *Paris strain 200PS and the Paris F-lac+*

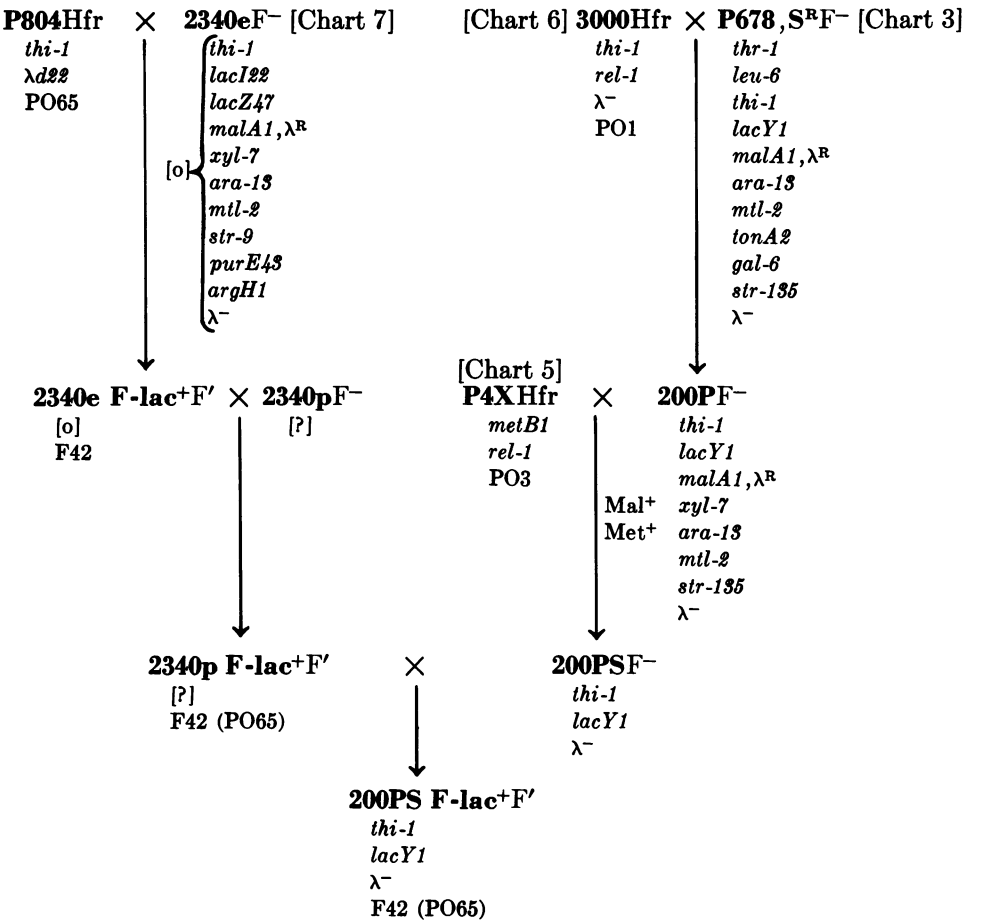

CHART 11. *The Paris F_1-gal and F_2-gal*

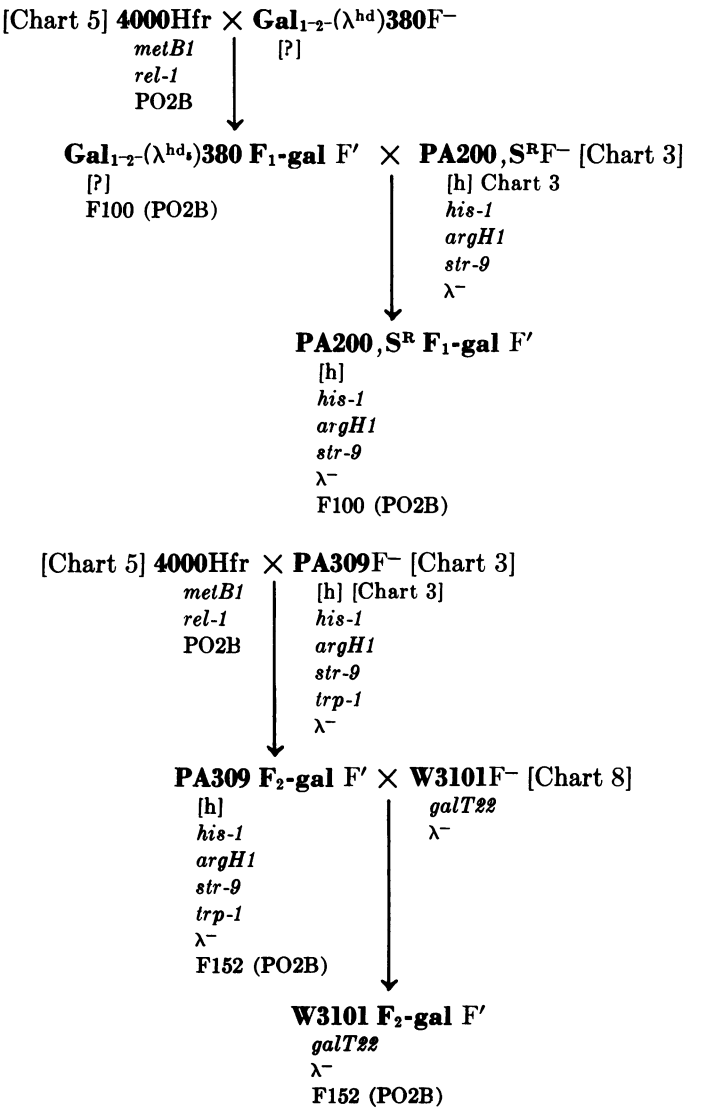

CHART 12. *Derivation of Garen pho⁻ and Su⁺ strains*

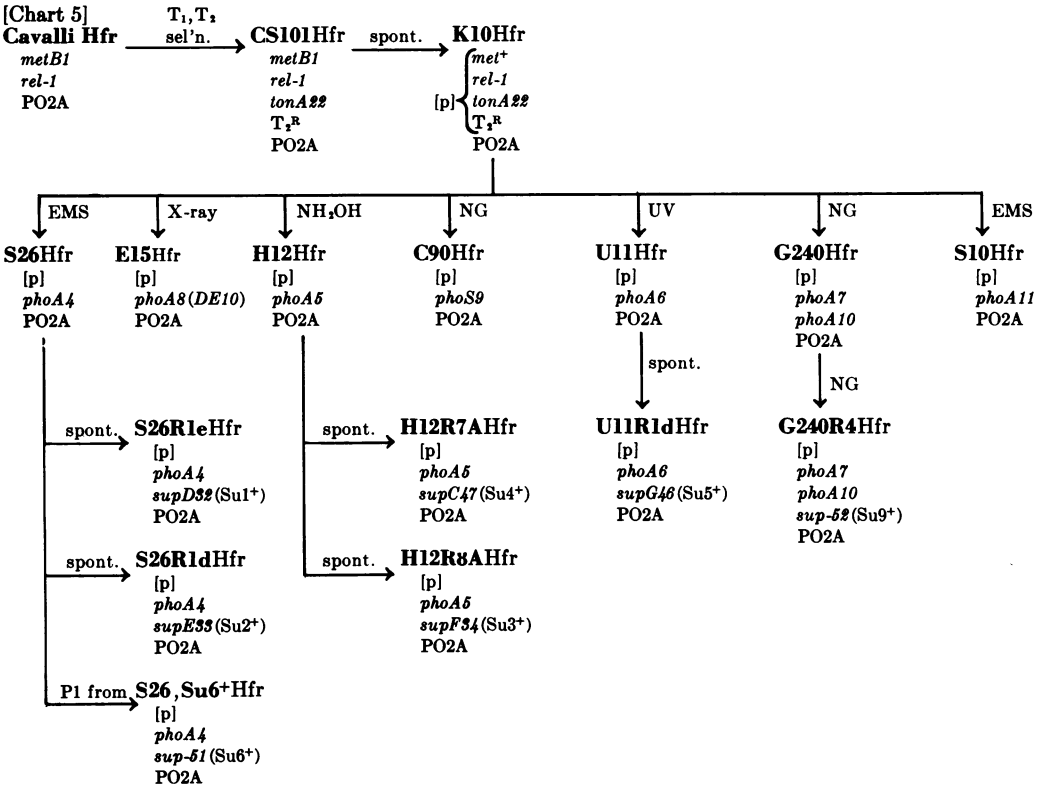

CHART 13. *The derivations of miscellaneous Hfr strains*

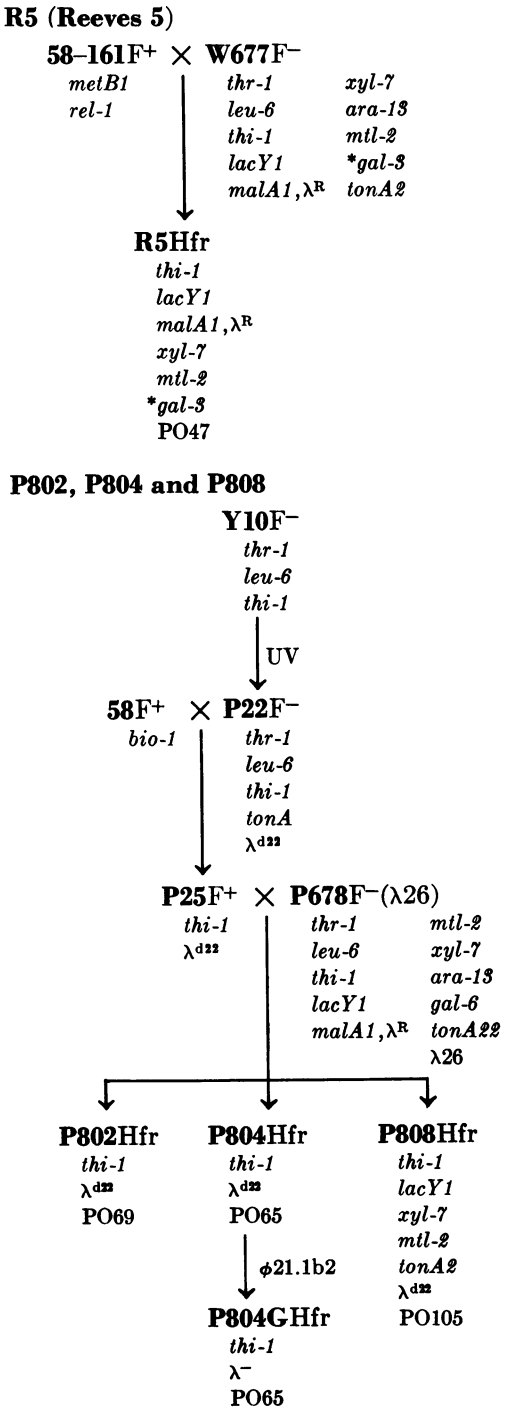

CHART 14. *The strains K12S and 112*

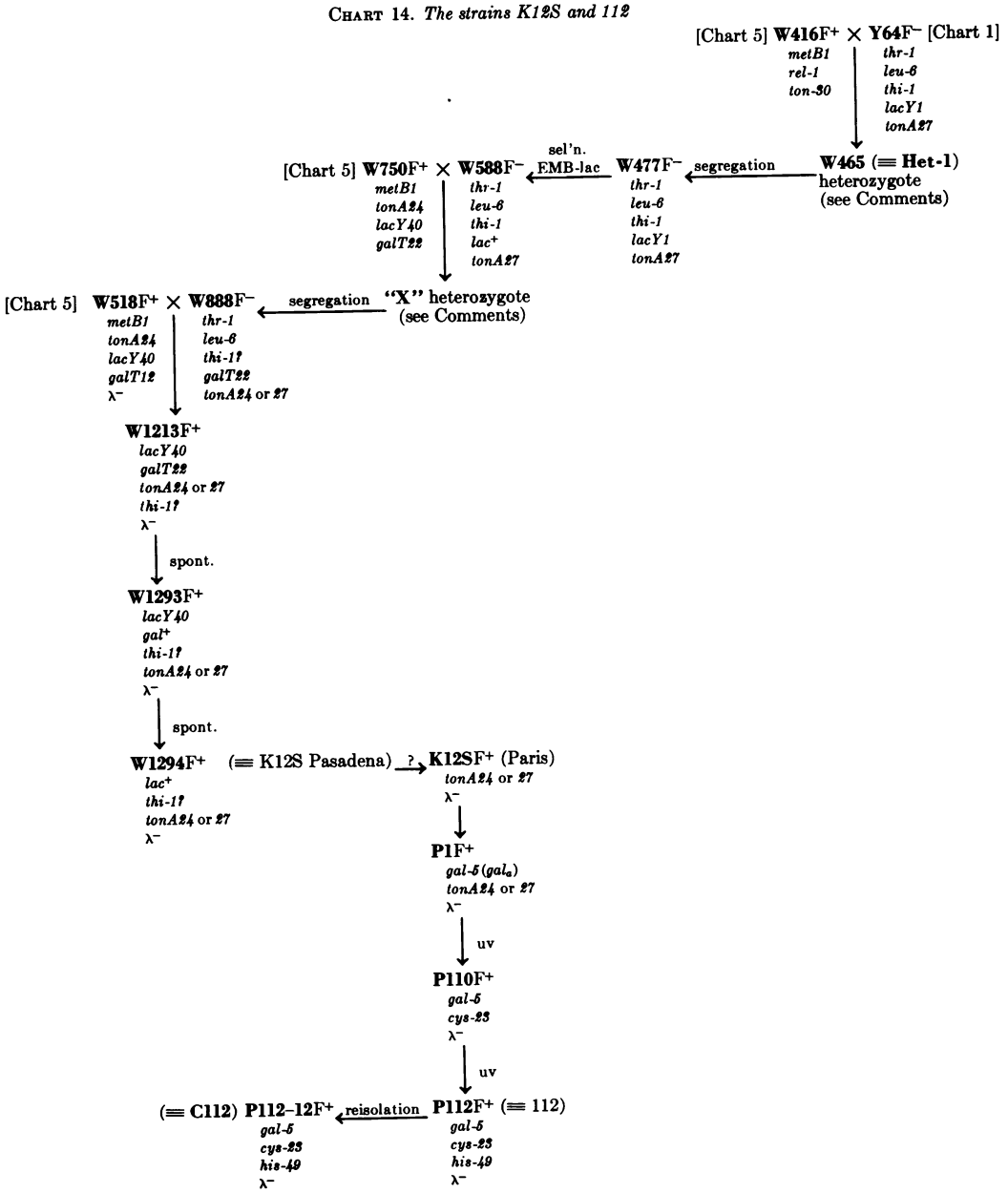

TABLE 1

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
E. coli K-12		35, 64, 98, 99		WG1; see text.
(λ)		57, 58, 60, 61		
F^+		17, 67		
AB250	C			See PA100
AB253	C			See W208, S^R
AB254	C			See W208, S^R F^-
AB257	C			See 4000 Hfr; not Cavalli Hfr!
AB258	C			See W583
AB259	C			See Hayes Hfr, Thi^-, λ^-
AB262	C			See PA100, S^R Pro ; JW1
AB264	C			*E. coli* K-12 wild type; *ara* due to the presence of phage mu-1 in the *ara* cistron.
AB265	C			See W2915
AB266	C			See W2961
AB280	C			"58-161", actually W6
AB281	C			Hayes Hfr, Thi^-, λ^-; see 3000
AB284	C	101	2	W208, S^RF^+
AB311	C, D	101	2	AT11
AB312	C, D	101	2, 9	AT12
AB313	C, D	101	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	45	2	
AB1621	C	2	2	
AB1859	C		2	
AB1884	F	44	4	
AB1885	F	44	4	
AB1886	F	43, 44, 45	4	
AB1896	F		4	
AB1899	F	43, 45	4	
AB2301				See 3300
AB2457	F		4	
AB2462	F	46	4	
AB2463	F	43, 44, 46	4	
AB2470	F	28, 43	4	
AB2474	F	43	4	
AB2487	F	109	4	
AB2495	F	28	4	
AB2497	F	28, 44	4	
AB2500	F	44	4	
AB3004	C			See C600 (CR34)
AB3022	F	28	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

TABLE 1—*Continued*

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
AT52	D			See AB352
AB705	D		6	
AT715	D		6	
AT716	D		6	
B1-B12				See Broda 1-12 Hfr's
Broda 1-Broda 12, Hfr's		12, 40, 41, 77	5	
C6		4	2	
C60		4	2	
C90	d	31	12	
C112		3a		See P112
C600	a	4	2	CR34 std. λ indicator
sup E44		27, 91		
Cavalli Hfr		16	5, 12	Hfr C, W1895, Hfr_1 (Lederberg) *not* AB257
CR34	a	4	2	See C600
CR34, Thy^-		80, 81	2	
CR63		5	8	λ host range indicator
sup D60		11, 27, 113		
CR67		5	8	$\lambda^{vir.}$ indicator
CS2		93	5	
CS11		92	2	
CS19		92	1	
CS100		93	2	
CS101		93	5, 12	
E15	d	37, 56	12	
G240	d	19	12	
G24OR4	d	19	12	
H12	d	30, 32, 105	12	
H12R7A	d	30, 32	12	
H12R8A	d	32, 106	12	
Hayes Hfr	e, A, B	39	5, 6	HfrH; W2323 Hfr_2 (Lederberg); Hfr2 or Hfr_2 (Paris)
Hayes Hfr, Thi^-	e, k, A, B	50	6	Hfr4 or Hfr_4 (Paris)
Hayes Hfr, Thi^-, λ^- (Paris)	e, k, A, B, C	50	6, 7, 10	HfrH, AB259, HfrC, 3000 and Hfr H (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)				See W2945
Hfr_4 (Paris)	B	50		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
Hfr 7-4	g	26*a*	9	MA1048
JIF^+	c		8	
$J4F^+$	c	24	8	
$J4\text{-}5F^+$	c	24	8	J45
$J5F^+$	c	24	8	
$J5\text{-}3F^+$	c	24	8	J53
$J5\text{-}10F^+$	c	24	8	J510
$J6\text{-}2F^+$	c	24	8	J62
J1Hfr	B			See P1
J2Hfr	B			See P4X
J3Hfr	B			See P3, 4000
J4Hfr	B			See P10
J5Hfr	B			See P72
J6Hfr	B			See P13

TABLE 1—*Continued*

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
J7Hfr	B			See P808
J45F$^+$				See J4-5
J53F$^+$				See J5-3
J62F$^+$				See J6-2
J510F$^+$				See J5-10
JC12	b	20	9	AB815
JC158	b	20	9	AB856
JC182	b	20, 22	9	AB862, "double male"
JC355	b		9	AB869
JC411	b	22,23	9	
sup-59		36		
JC500	b		9	
JC590	b	22	9	
JC1552	b		9	
JC1553	b	21, 23	9	
JC1554	b	21, 23	9	
JC1557	b	14	9	
JC1569	b	14	9	
JC2915	b	108	4	
JC2926	b		4	
JC4474	b		6	
JC4490	b		6	
JC5029	b	21, 109	6	
JC5088	b	21, 109	6	
JC5401	j		6	
JC5408	j	108	4	
JC5412	j	21, 108, 110	6	
JC5421	j	108	4, 6	
JC5422	j	108	4	
JC5426	j	108, 110	6	
JC5474	j	108	4	
JC5489	j	6	4	
JC5491	j	108	6	
JC5495	j	108	6	
JC5519	j	108	4	
JC5544	j	108	6	
JC5547	j	108	6	
JC5743	b	6	4	
JW1	B			See PA100, S^R, *Pro*$^-$
K10		56, 93	5, 12	
K12, wild type				See first entry
K12S		4, 71*a*, 106*b*	14	See W1294
K15			5	
KL14	f	74	6	
KL16	f	73, 74	6	
KL16-99	f	73	6	
KL20	f		6	
KL25	f	73, 74	8	
KL96	f	73, 74	6	
KL98	f	73	8	
KL99	f	74	6	
KL110	f		9	
KL161	f		6	
KL162	f		6	
KL166	f		6	
KL168	f		6	
KL169	f		6	
KL173	f		6	
KL174	f		6	
KL211	f		9	
KL252	f		4	
M series	B		6	Mutants of Hfr3300 (Paris)

TABLE 1—*Continued*

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
P1	B		14	
P1, Hfr	B	52, 54 (p. 162)	5	HfrP1, Hfr Type 1, HfrJ1
P2	B		5	HfrP2, HfrP21
P3	B	49, 52, 54 (p. 162)	5, 11	HfrP3, HfrP31, Hfr Type 3, 4000, HfrJ3, AB257
P4X	B	49, 52, 54 (p. 162)	5, 9, 10	HfrP4X, Hfr Type 2, HfrJ2
P5	B		5	HfrP5
P6	B		5	HfrP6
P8	B		5	HfrP8
P10	B	49, 52, 54 (p. 162)	2	HfrP10, Hfr Type 4, HfrJ4, AB673
P13	B	49, 54 (p. 162)	5	HfrP13, Hfr Type 6
P21	B			See P2
P22	B	27	13	
P25	B		13	
P31	B			See P3
P72	B	49, 52, 54 (p. 162)	5	Hfr P72, Hfr Type 5
P110	B		14	
P112	B		14	
P112-12	B, k		14	
P676	B	50, 54 (p. 60), 112	2	
P678	B	54 (p. 60) 111, 112	2, 3, 13	
P678, S^R	B		3, 7, 10	2001w
P678, F^+	B		3	
P678, λ^-	B	54 (p. 60), 112	3	
P697	B			See PA100
P697, S^R	B			See PA100, S^R
P802	B		13	
P804	B		10, 13	
P804G	B		13	
P808	B	49, 54 (p. 162)	13	Hfr P808, Hfr Type 7
P3478		26	8	
PA100	B		3, 9	P697
PA100, S^R	B		3	P697, S^R
PA100, S^R, Pro^-	B		3	JW1
PA100-PA125	B		3	Series of auxotrophic mutants
PA200	B		3	
PA200-PA266	B		3	Series of auxotrophic mutants
PA265	B		9	
PA200, S^R	B		3, 11	
PA200, S^R F_1-*gal*	B		11	
PA300-PA394	B		3	Series of auxotrophic mutants
PA309	B		11	
PA309 F_1-*gal*	B		11	
PA351	B		3, 7	
PA601-PA640	B		3	Series of auxotrophic mutants
PA610	B		3	
PA641-PA644	B		3	Series of auxotrophic mutants
R1-R5	h			See Reeves 1-5
Ra-1	f	55, 72	8	
Ra-2	f	55, 72, 73	8	
Reeves 1	h	40, 41, 83	5	HfrR1
Reeves 2	h	83	2	HfrR2
Reeves 3	h	83	2	HfrR3
Reeves 4	h	40, 41, 83	5	HfrR4
Reeves 5	h	83	13	HfrR5
S10	d	30, 79, 97	12	
S26	d	32, 33, 97	12	
S26R1d	d	32, 33	12	
S26R1e	d	32	12	

TABLE 1—*Continued*

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
S26, *Su6*$^+$	d		12	
T94A	i			See 58-278M
U series	B		6	UV mutants of HfrH 3000 (Paris)
U11	d	30	12	
U11R1d	d	30	12	
W1	A		2	
supE44		33, 106		
W6	A	34, 67	1, 5	58-161, bio$^-$; AB280
bio$^+$		34, 67, 89, 104		
rel$^-$		3, 9, 10, 96		
W13	A	34	1, 5	Y40, bio$^+$
W14	A	34	1, 5	Y87, bio$^+$
W45	A		5	
W67	A	65, 68	5	
W102	A	107	2	
W112	A	59	2	
W133	A	59	2	
W208	A		2	
W208, S^R	C		2	AB253
W416	A		5, 14	
W435		61	5	
W465	A	62*a*	14	Unstable heterozygote
W477	A		14	
W480	A		2	
W516		34, 59	5	
W518	A	34, 59, 61, 78	5, 14	
W566	A		2	
W582	A		2	
W583	A	78	2	AB258
W588	A		14	
W595	A		2	
W620	A		5	
W660	A		2	
W677	A	18, 63, 66	2, 6, 13	AB781
gal-3		78		
W677, F$^+$	e		2, 6	
W750	A	59, 78	5, 14	
W888	A	59	14	
W894	A		2	
W902	A	59, 78	2	
W904	A		2	
W922	A		2	
W945	A	104	2	
W1163	A		5	
W1177	A	63, 66	2	W677, S^R
W1210	A	78	5	
W1213	A		14	
W1293	A		14	
W1294	A		14	
W1394	A		2	
W1485	A	61, 102	8	
supE42		102, 113		
W1603	A	61	2	
W1654	A		8	
W1655	A	61	5	
W1655, F$^-$		89*a*	5	
W1655, F$^+$, λ^R		12	5	
W1673	A		8	
W1872	A	61	8	
W1895	A			Hfr_2, stable clone selected from Cavalli Hfr

TABLE 1—*Continued*

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
W2070	A	78	8	
W2071	A		5	
W2207	A		5	
W2252	A		5	
W2323	A			See HfrH; Hfr_1
W2324	A			See HfrH, Thi^-, λ^+
W2637	A		8	
W2660	A	87	2	
W2817	A	87	2	
W2914	A		2	
W2915	A		2	
W2924	A	85, 86, 87	2	Hfr_3
W2945		84	2	Hfr_4
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, 11	
W3101 F_2-gal	B		11	
W3102	E		8	
W3104	E		8	
W3106	E		8	
W3107	E		8	
W3108	A		8	
W3109	A		8	
W3110	A, E		8	
W3110, Thy^-			8	
W3135	A	87	5	
W3201	A		5	See text
W3208	A	42, 77, 95	5	See text
W3213	A	13, 77	5	Hfr_{13}
W3236	A		5	
W3634	A		6	HfrH, Thi^-, λ^-, *not* Paris strain 3000
W3787	A	25	5	
W3807	A		5	Hfr_6, MA1040
W4354	A		5	
"X" series	B	8	6	X-ray mutants of HfrH 3000 (Paris)
Y10		59, 62, 70, 99	1, 2, 13	
supE44		27		
Y24		62, 70, 99	1	
Y40		59, 62	1, 5	
bio^+		34		
Y46		62	1	
Y53		59, 62	1, 2	
Y64		62	1, 14	
Y70		59	1, 2	
Y80		62	1	
Y86		62	1	
Y87	A	59, 62	1, 5	
bio^+		34		
Y91		62	1	
Y94		62	1	
Y100		62	1	
Ymel		88, 102	1	
supE57	l	113		
supF58	l	91		

TABLE 1—*Continued*

Strain designation	Sources of data	Published references	Chart	Synonyms and comments
"YA" series	B	7	6	N-mustard mutants of HfrH 3000 (Paris)
58		35, 62, 98, 99	1, 5, 13	W3301
58-161		62, 98, 99	1, 5, 13	See also W6
Reversion to bio$^+$		34, 67, 89, 104		
Mutation to rel$^-$		3, 9, 10, 96		
58-161F$^-$		38, 39, 94	5	58-161F$^-$ Spicer; W6 derivative
58-161F$^-$, S^R		38, 39	5	58-161F$^-$ Hayes; W6 derivative
58-161, F$^-$, S^R, Az^R		38, 39	5	W6 derivative
58-161, F$^+$, S^R, Az^R		38, 39	5	W6 derivative
58-278		62	1	
58-278M		103	1	T94A; Treffers mutator strain
58-309		98	1	
58-336		98	1	
58-580		98	1	
58-593		98	1	
58-610		98	1	
58-741		98	1	
58-2651		98	1	
112	B			See P112
112-12	B, k			See P112-12
200P	B		10	
200PS	B	49	10	
200PS F-*lac*$^+$	B	49	10	
679		35, 62, 98, 99	1, 2	
679-183		98, 99	1	
679-440		98	1	
679-680		62, 98, 99	1, 2	
F$^-$		67		
2000	B	29	7	
2001w				See P678, S^R
2001d	B		7	
20SO	B	15, 29, 76	7	
2300	B		7	
2310	B		7	
2310e			7	
2320	B	11, 29	7	
2340e		29, 76	7, 10	
2340p		29, 76	10	
3000	B	29, 76, 82	6, 7, 10	HfrH, Thi$^-$, λ$^-$, Hfr"C" (Paris), AB259, HfrH
3000X74			6	
30SO	B	7, 76	6, 7	
30SOU1-U7	B	7, 15	6	Series of pyr$^-$ mutants
3300	B	29, 76, 82	6, 7, 9	
3310	B	29, 82	6, 7	
3320	B	82	6, 7	
3340	B	82	6, 7	
4000	B	82	5, 11	P3, P31, Hfr Type 3, AB257

nyms are given in Table 1 and are cross-indexed to the original designations. The alphabetical prefixes used in the original strain designations may, in addition to identifying the strains, indicate the laboratories in which the strains were made and 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 strains; non-K-12 strains were designated by **AC** numbers. P. Howard-Flanders A. J. Pittard, A. L. Taylor and others have had **AB** "number blocks" and designated their strains and mu-

tant alleles according to this system. Now, however, Howard-Flanders and Taylor use their own systems. The **AT** prefix is used by A. L. Taylor, University of Colorado Medical Center. The **C** prefix was used at the California Institute of Technology. **CR** was used by R. Appleyard after he moved from the last-named institution to Chalk River, Canada. **CS** was used by P. D. Skaar, and others, for strains isolated in the Cold Spring Harbor Laboratory of Quantitative Biology.

The prefix **J** was used by B. D. Davis, Cornell University Medical College, for some K-12 derivatives. However, Davis worked extensively with the "W," or Waksman, strain of *E. coli* [American Type Culture Collection strain number 9637], not to be confused with the Wisconsin **W** strains of K12.) **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, Berkeley. **KL** is used by K. B. Low of Yale University (formerly at New York University School of Medicine).

P was used by Jacob, Wollman, and others at the Pasteur Institute for F^+ and Hfr strains, while **PA** was used to designate their F^- straints (with the exception of a few early F^- strains that had **P** designations). The large number of synonymous strain designations applied to some of the Paris Hfr's has led to considerable confusion, which we have attempted to resolve in Table 2.

The **W** prefix was used by J. Lederberg and collaborators at the University of Wisconsin to designate mutant strains. They used the prefix **WG** (Wisconsin Genetics) to designate wild-type strains: *E. coli* K-12 was designated "WG1," and all other WG numbers referred to non-K-12 strains in their system. The prefix **Y** was used in the laboratory of E. L. Tatum at Yale University in the 1940s. The very earliest strains, those produced by Gray and Tatum at Stanford University, were given only number designations.

The genetic symbols. The genetic symbols used throughout are those of Taylor (100), with the following exceptions and additions. The older symbols *malA* and *malB* are used for Mal^- mutations originally mapped in terms of these loci. The symbol *thyR* is used to refer to the mutation involved in producing the phenotype "thymine low requirement" when it is not known whether the locus affected is the *drm* or *dra* locus. The symbol PO is used to designate the points or origin of Hfr strains, each individual mutation to the Hfr state being assigned a unique number; the Hfr descendants of, as well as the episomes derived from, an Hfr strain are assumed to have inherited the point of origin of their Hfr ancestor. The symbol *sfa* is used to designate sex factor affinity sites as defined by Adelberg and Burns (1). The phenotypic symbols T_2^R chloroacetate^R, and glycerol$^-$ are used in the descriptions of some of the early strains to designate resistance to bacteriophage T_2 and chloroacetate, and the inability to ferment glycerol, respectively.

The mutant allele designations used throughout are those assigned for used in the *E. coli* Genetic Stock Center and do not necessarily correspond to those used in any other laboratory. The Stock Center numbering system is built on the system employed by E. A. Adelberg in his strain records; some of these mutant allele designations have thus been in use for many years and may be recognizable from their frequent appearance in the literature. There are three sets of mutant allele designations that are sufficiently widely recognized that we have felt it necessary to include them alongside the Stock Center designations in the strain descriptions. These are the *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 *Su* designations used by Garen and co-workers for suppressor alleles. The Wisconsin *gal* designation if given for a gal^- allele the first time it appears on a pedigree chart and in the descriptions of the λ HFT *gal* lysates in Chart 8. The gal_b symbol is indicated on Charts 2 and 3 in the descriptions of strain P678. A few of the Paris lac^- designations are given in Charts 6 and 7.

The symbol F1 is used throughout 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 λ is not noted in strain descriptions as this is the wild-type state. Resistance to λ is symbolized by λ^R without any indication as to the locus involved because the locus of this resistance is not known for many of the early strains.

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 methane sulfonate; irrad. = radiation of unknown character; NA = nitrous acid; NG = *N*-methyl-*N*,-nitro-*N*-nitrosoguanidine; N-mustard = nitrogen mustard; spont. = mutation occurring in absence of deliberate mutagenic treatment;

UV = ultraviolet 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 = selection for or against utilization of the indicated sugar (lactose here) on eosin-methylene blue agar plates; λ"x" = selection with indicated type of bacteriophage λ; motility agar = selection on basis of motility in semisolid agar; nal = nalidixic acid; spc = spectinomycin; str = streptomycin; T_1, T_2, T_6 = bacteriophages T_1, T_2, and T_6, respectively; TRIM = trimethoprim. The standard abbreviations for sugars are used: ara = arabinose; gal = galactose; lac = lactose; mal = maltose; mtl = mannitol; and xyl = xylose.

The treatment of suppressors. In our experience it has proved very difficult to track suppressors through the strain pedigrees. This is true not only because the presence or absence of suppressors was seldom noted knowledgeably for the early strains, but also because their expression seems to be affected by so many other factors. For these reasons we have in most cases noted the presence of suppressors only in those strains in which they have been reported and have note noted the likelihood of their presence in ancestors or offspring of these strains. The one exception is the *supE*$^-$ allele of the Y10 line, which will be discussed in the comments on Charts 1, 2, 3 and 4.

Comments on Charts

Chart 1. Some early Stanford and Yale strains. As can be seen from this chart, many of the early strains were isolated after the rather drastic treatment of X-irradiation. An appreciation of this fact has led to their being abandoned by many in later years as ancestors for the construction of new stocks. Nevertheless, the majority, by far, of the strains that have come to our attention can be traced back to these early lines.

The strains W6, W13, W17, and CS19 are included here to emphasize the instability of the *bio-1* mutation, which reverted very early on in several important ancestral stocks (34). This allele will be discussed at more length in the comments on Chart 5.

The suppressor mutation *supE44* was detected in strain Y10 in 1966 (27) and may be present in most or all of its direct descendants (i.e., strains descended by mutational rather than recombinational events).

Chart 2. Some derivatives of strain Y10. *supE*$^-$ mutations have been reported in strains Y10 (27), C600 (91), and W1 (106). We have assumed that these are all the same mutant allele (designated *supE44* by us) which resulted from a mutation in strain Y10 or one of its ancestors and which may be in all of its descendants. The uncharacterized amber mutation in strain W208 (H. Hoffman-Berling, *personal communication*) may be *supE44*, but we have assigned it the unique mutant allele designation *sup-49* until more is known about it.

The strain designations CR34 and C600 are synonymous. The 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 rechristened CR34 at that time (R. Appleyard, *personal communication*). Considerable confusion has arisen from this renaming and from the unfortunate fact that when Okada, Yanagisawa, and Ryan (80, 81) made a Thy$^-$ derivative of this strain they did not give this derivative a new strain designation. The only name for the latter is now 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 the chart, the sequence of events involved three *gal*$^-$ mutations and two reversions, which may have been due to suppressor mutations. Morse, Lederberg, and Lederberg in 1953 (78) recognized that their gal_5 (our *gal*-3) marker in W677 was complex. The gal_b marker (our *gal*-6) in P678 and its derivatives have been observed to give a variety of Gal phenotypes upon recombination (E. A. Adelberg, *personal communication*) and may even involve a chromosome rearrangement (R. Curtiss, III, personal communication).

The *malB5* mutation in the Wisconsin Hfr_3 (W2924) and the mutation *malB16* in the Paris Hfr J4 (P10) both involve the integration of the sex factor in the *malB* locus (Hfr_3 [85, 86, 87]; J4 [90]).

The strain W208S^R, ancestor of AB283, was acquired by E. A. Adelberg in the Paris laboratory and is probably not identical with the Wisconsin strain W2325, which is also a S^R derivative of W208.

Chart 3. Some derivatives of strain P678. The widespread early derivatives of the Paris strain P678 consist, for the most part, of five series of auxotrophic strains, each series being produced from a single strain from the previous series. All were descended from P678 cured of bacteriophage lambda. The strain PA100 was, in Adelberg's collection, called

P697, a designation used only briefly in Paris. Similarly, the designation JW1 for the Str^R, Pro^- derivative of PA100 has been used in the Adelberg collection for some years but was apparently evanescent in Paris. The gal_b constellation is in all of these strains. The suppressor from the Y10 line may be here also.

Chart 4. Some derivatives of strain AB1157. After discovering the complexity of the gal_b marker, Adelberg used the Wisconsin strain W2915 in the construction of many of his strains. AB1157, a derivative of W2915, was then used extensively by P. Howard-Flanders and A. J. Clark, as is shown in this chart. Again, the suppressor from strain Y10 may be in some or all of these stocks.

Chart 5. Some derivatives of strains 58-161 and W6. Most of the widely used Hfr's are to be found on this chart. They were thought to be derived from 58-161 (*bio-1*, *metB1*) at the time they were made, but it was later noted (34, 67, 89, 104) that the *bio-1* marker had reverted, apparently rather shortly after 58-161 was made. Later still, it was discovered that the spontaneous mutation *rel-1* (to the relaxed state with respect to RNA synthesis) had appeared in the strain, also very early in its history (3, 9, 10, 96). The result is that most of the well-known Hfr's are bio^+ and carry the *rel-1* marker.

The *bio-1* marker reverted in the strains Y40 and Y87 also. The strains that are described in the literature as having come from Y87 most likely came from the revertant, designated W14 (34).

The Hayes Hfr is a Str^R, Azi^R derivative of W6. The derivation of the widely used Thi^- derivative, a recombinant, is given in Chart 6.

So many synonymous strain designations have been used to refer to the more widely used Paris Hfr's that considerable confusion has arisen concerning their nature and derivations. Table 2 is designed to clarify some of these points, as a supplement to the charts. Concerning the Paris Hfr P3 (more widely known as Hfr4000), it should be pointed out that this is not the Cavalli Hfr, as has been erroneously believed by many workers in America. The Cavalli Hfr was isolated by L. L. Cavalli (16) after treatment of "58-161" (actually W6) with nitrogen mustard. The Hfr P3 (which we shall call 4000 and which is called Hfr Type 3 in the references immediately following) arose spontaneously from 58-161 (W6) in the hands of Jacob and Wollman (49, 51, 52; 54, p. 162). This strain was assumed by E. A. Adelberg to be the Cavalli Hfr. It was designated AB257 in his collection and has been widely disseminated as "the Cavalli Hfr" or "HfrC," which it is not (82). The point of origin of this

TABLE 2. *The Paris Hfr's*

Type[a]	Synonyms	Point of origin	Derivation	Genotype	Comments
Hfr H	Hfr 2; original Hayes Hfr	PO1 thi thr ←	Spont. from 58-161 F^+, S^R, Azi^R	*metB1, rel-1, str-100, azi-7*	
	Hfr 4; Hfr_4	PO1 thi thr ←	Recombination: HfrH × W677 F^+	*thi-1, rel-1*	
	Hfr 3000; Hfr "C"	PO1 thi thr ←	Hfr 4, cured of λ^r by UV.	*thi-1, rel-1,* λ^-	
Type 1	P1	PO103 leu azi →	Spont. from 58-161 (W6)	*metB1, rel-1*	Lost very early
Type 2	P4 × 6; J2	PO3 pro lac →	Spont. from 58-161 (W6)	*metB1, rel-1*	
Type 3	P3; P31, 4000	PO2B purE lip →	Spont. from 58-161 (W6)	*metB1, rel-1*	Source of F_1-*gal* and F_2-*gal* of Paris (F100 and F152)
Type 4	P10; J4	PO18 thi malB thr →	Spont. from C6000 F^+	*thr-1, leu-6, thi-1, lacY1, tonA21, malB16,* λ	F is integrated in *malB* locus
Type 5	P72; J5	PO102 ilv met ←	Spont. from 58-161 (W6)	*metB1, rel-1*	
Type 6	P13	PO104 mtl ilv ←	Spont. from 112, S^R	*his-49, cys-23, gal-5, str-58,* λ^-, T_1^R, T_3^R	
Type 7	P808	PO105 tonA pro →	Spont. from cross: P25F^+ × P678	*Thi-1, lacY1, xyl-7, mtl-2, tonA2*	
	P2; P21	PO106 lac purE →	Spont. from 58-161 (W6)	*metB1, rel-1*	
	P804	PO65 pro lac →	Spont. from cross: P25F^+ × P678	*thi-1*	Source of F'-*lac* of Jacob and Adelberg, (F42)

[a] As listed in references 49, 51, 52, 53; 54, p. 162.

strain was designated PO2, the number assigned to the point of origin of the Cavalli Hfr. The Stock Center inherited this nomenclature, unfortunately, and has been perpetuating this error until quite recently. No one has found, to our knowledge, any difference between the points of origin of strain 4000 and the Cavalli Hfr. However, it would not be surprising if these two strains differed in genetic background considering the different treatment they received. We have assigned them distinctive PO designations, as follows. The Cavalli Hfr now has PO2A, in our nomenclature, and the point of origin of Hfr 4000 is PO2B. In cases where it is impossible to decide which of the two strains is involved, the point of origin will be called simply PO2.

The parent of Cook and Lederberg's (25) extensive series of several hundred *lac*$^-$ mutants (W3787) is given on this chart.

The Wisconsin strains W3208 and W3201, sometimes referred to as Hfr_8 and Hfr_{15}, respectively, require special comment. When the strains W3208 and W3201 were isolated, it was assumed that they were Hfr's because of their ability to transfer chromosomal markers at high frequency. When these two strains were examined in greater detail later on, it was found that both were F′ strains, harboring F8 and F15, respectively (42, 47, 95). Thus nothing is known of the Hfr strains that may have been the immediate ancestors of these two F′ strains (J. Lederberg, *personal communication*).

Chart 6. HfrH Thi$^-$, λ$^-$ and some of its derivatives. The most widely used version of the Hayes Hfr, HfrH Thi$^-$, λ$^-$, is sometimes thought to be the original strain isolated by Hayes. It, is, however, a recombinant strain, resulting from a cross between a phenocopy of HfrH and the heavily marked Wisconsin strain W677, F$^+$. The strain isolated from this cross was Thi$^-$ and carried bacteriophage lambda. Oddly enough, considering its ancestry, it appears to be suppressor-free. Both the Paris and Wisconsin laboratories then cured this strain of lambda phage. The Paris HfrH, Thi$^-$, λ$^-$ is the one that is widely used: it is equally well known as strain 3000. Some confusion has resulted from the fact that it was also called "HfrC" within the Pasteur Institute (see Table 2).

Several important series of mutant strains were produced from strain 3000 in the Paris laboratory. The series produced by X-irradiation included some of the widely used *lac* deletions, such as those in strains 3000 X74 and 3000 X111. Another series was isolated after U1–U488, etc. A third series, isolated after treatment with nitrogen mustard, consisted of some 297 auxotrophic mutants called YA1–YA297. (It is important to note that strains bearing higher YA numbers arose in a different manner.) From the strain 30SO, a *lacZ*$^-$ derivative of strain 3000, there were isolated, after UV-irradiation, seven pyrimidine-requiring auxotrophs, designated 30SOU 1–7, respectively.

From the strain 3300, a *lacI*$^-$ derivative of strain 3000, were isolated three widely used strains carrying *lacZ* mutations, 3310, 3320, and 3340, and a series of strains carrying auxotrophic mutations which were designated as HfrH "M" strains 1–30.

From F$^+$ revertants of strain 3000, K. B. Low and A. L. Taylor produced some of their widely used Hfr's, and Taylor produced several series of auxotrophic strains used in his mapping studies. (The cluster of markers *gltS7*, *gadS1* and *gadR2* may or may not be present in AT705. They were found by Lupo and Halpern (75) in a recombinational derivative of AT705 and it is thought that they may have arisen along with the *rbs-1* mutation, when strain 3000 was treated with nitrosoguanidine to produce strain AT705). A number of widely used *rec*$^-$ Hfr strains have been produced from Hfr KL16 by Low and A. J. Clark.

Chart 7. Some of the early Paris lac strains. This chart is designed to show the rather complex relationships between the widely used 3300 series and 2300 series of strains from the Paris laboratory. There is, however, some doubt about the parentage of strain 2300, which was made by J. Monod.

Chart 8. Other lines derived from wild type. The strains W1485 and W3110 have been used extensively as ancestral stocks in an effort to get away from the heavily mutagenized early Stanford strains. The amber suppressor, *supE42*, found in W1485 is not in W3110, which is either suppressor-free or carries a weak ochre suppressor (C. Yanofsky, *personal communication*).

The genetic step involved in the isolation of W3110 was the selection of a strongly fermenting colony on EMB-gal, as W2367 appeared to be "a weak gal fermenter."

Chart 9. The derivation of JC12 and JC411 and some of their derivatives. These recombinant strains are included here because they have been used widely for the construction of stocks. Note that the *gal-6* (gal_b) constellation is in most of these strains.

Chart 10. Paris strain 200PS and Paris F-lac$^+$. There is a widespread misconception that the designation 200PS refers to a particular F′ strain, carrying a particular episomal

element. This is not the case. 200PS is an F^- strain, which is the host strain to a number of episomes in various Parisian F' strains. In this chart it is the host to the Jacob and Adelberg F-*lac*$^+$ episome (48), our F42, derived from Paris Hfr P804 (Chart 13).

Chart 11. The Paris F_1-gal and F_2-gal. These two widely used episomes arose from the Hfr 4000, not the Hfr Cavalli. The point of origin is, however, similar to, if not the same as, that of the Cavalli Hfr (see notes to Chart 5). The F_1-*gal* is also called the "long" F-*gal* or F-*gal*, λ^{att}, *bio*. At least one widely used version of this episome (in strain M57 of Meselson) carries a mutant suppressor allele, probably at the *supE*$^-$ locus (M. Meselson, *personal communication*).

Chart 12. Derivation of Garen Pho$^-$ and Su$^+$ strains. This chart gives the derivation of the set of strains used most often as sources of these *pho*$^-$ and *sup*$^-$ (Su$^+$) markers. Strain C90 produces alkaline phosphatase constitutively. Strain E10 carries a *phoA* deletion.

Chart 13. The derivations of miscellaneous Hfr strains. The Hfr strain known as R5 or Reeves 5 arose spontaneously during a cross between 58-161F$^+$ (W6) and W677F$^-$ (P. Reeves, *personal communication*). It is a recombinant of these two strains.

The Hfr P804 is the source of the Paris F-*lac*$^+$ episome (our F42: Chart 10).

Chart 14. The strains K12S and 112. One of the first λ^s (i.e., λ^+) strains isolated by the Lederbergs was the strain W518 (61). A derivative of W518, designated W1294, was made by the Lederbergs as shown in Chart 14. The strain W465 which was involved in this pedigree, and which was described by J. Lederberg in 1949 (62*a*) as "H-1", is still not fully understood. It was a heterozygote, an unstable diploid, which segregated out large portions of the genomes of both of the parent strains. It cannot be explained by simply assuming that it was an F-prime strain. The strain W477 was a stable segregant from W465. The strain "X," indicated in this pedigree, which was neither saved nor given a "W" number, was also a heterozygote, and strain W888 was a segregant from this unstable diploid.

The strain W1294, in which the presence of the *thi-1* marker was questionable, was sent to J. Weigle at the California Institute of Technology in Pasadena by E. Lederberg in 1950 (106*a*; and J. Lederberg, *personal communication*). This strain was called in Pasadena simply "S" (106a) or, later, K12S (4, 71*a*, 106*b*). This strain was sent by Weigle to the Paris laboratory, where it was found not to require thiamine (110a).

Monod and Wollman induced in strain K12S the gal_a mutation (our *gal-5*) (110*a*), creating strain P1. From strain P1, Wollman produced strain 112 (110*a*), later called P112, by a series of UV-induced auxotrophic mutations. A reisolate of strain 112, picked by Wollman on one occasion from a colony #12, led to the strains being called 112-12 (later P112-12) in Paris (E. Wollman, *personal communication*). This strain was then sent to Pasadena, where it was called C112 (3*a*).

The data books of the late J. J. Weigle were not consulted by the author. The above reconstruction of the pedigree of strain 112 is the most plausible one that could be reached on the basis of information supplied by the sources cited above. Some question arises due to the fact that Weigle apparently referred to more than one strain as K12S (106*b*).

DISCUSSION

It is now possible to trace the derivation of almost all of the strains held by the Stock Center and to apply a uniform and unambiguous system of strain designations and mutant allele designations to our stocks. Unfortunately, almost all of the strain descriptions that have been drawn up (and sent out) by the Stock Center previously must now be revised and corrected in the light of information gained through tracing the pedigrees.

Once this task is completed, we hope to be able to provide for investigators in the field of *E. coli* genetics strain descriptions and pedigrees that will make it possible to compare the genetic backgrounds of most of the important lines of mutant strains now in use.

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