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Chromosomal Loci of *Neurospora crassa*

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INTRODUCTION

In this article we bring together information on the phenotypes, genetic characteristics, and map locations of all the known Mendelian gene loci of *Neurospora crassa* and on other chromosomal landmarks such as centromeres, tips, and the nucleolus organizer. Over 500 loci are included. Linkage maps have been revised and augmented. If sites of gene action are known, they are indicated in figures that show biosynthetic or catabolic pathways. Information on wild-type enzymes is included only where necessary to explain the mutant phenotypes. The text is concerned primarily with the organization and function of each locus and only secondarily with allelic variation or properties that distinguish specific alleles. Chromosome rearrangements are not considered except when mapping or analysis of loci depends upon rearrangement break points. (See reference 808 for a review of rearrangements.) Mutations in the mitochondrial genome are not considered except as necessary for describing nuclear genes that interact with mitochondrial mutations. (See reference 394 for a review of mitochondrial genetics and reference 206 for a map of the *Neurospora* mitochondrial genome.)

For a brief general introduction to the biology, genetics, and cytology of *Neurospora*, see the opening sections of reference 808, where references to the most useful sources of more detailed information may be found.

HISTORICAL BACKGROUND

Neurospora was named and described 55 years ago by Shear and Dodge (978), who showed that mating type is determined by a single pair of alleles that show 4:4 segregation among unordered asci which were shot from the perithecia. Morphological differences of spontaneous origin were soon discovered and shown by Dodge to segregate in Mendelian fashion. The linear array of ascospores in the ascus was shown cytologically by M. S. Wilcox to reflect events in meiosis, and genes were shown by Wilcox and by Dodge to segregate sometimes at the first and sometimes at the second division of meiosis. C. C. Lindegren proposed that second-division segregations, as reflected in ascospore order, measured the frequency of crossing-over in the segment between a gene locus and its centromere. Lindegren discovered linked genes and constructed the first linkage maps (eight genes and the centromeres in two linkage groups) (609–611, 613). For references and accounts of the early *Neurospora* work, see reference 808 and *Neurospora Newsletter* (volume 20, 1973).

The predominantly auxotrophic mutants obtained by G. W. Beadle, E. L. Tatum, and their associates, beginning in 1941, were used to construct more complete maps. Six linkage groups were known by 1949 (482), and the seventh was soon added (874).

B. McClintock and J. R. Singleton showed in the 1940s that chromosome morphology and behavior during meiosis and mitosis in the ascus resemble those in higher eucaryotes and that they can be studied cytologically by the methods of plant cytogenetics, using light microscopy. The first genetic evidence of chromosome rearrangements was obtained in this period (see reference 482), and translocations were confirmed and described cytologically (656, 987).

The discovery of biochemical mutants in *Neurospora* in 1941 (67) led to the explosive development of biochemical genetics and molecular biology. Although many problems could be attacked more readily by using bacteria and viruses, *N. crassa* continued to be used as a eucaryotic model, and a succession of fundamental contributions were made using *Neurospora*. Auxotrophic mutants were used to elucidate

biosynthetic pathways (e.g., reference 1010). Complementation between alleles was demonstrated both in vivo and in vitro (344, 393, 1157). Temperature-sensitive conditional mutants that were irreparable by supplementation were obtained (481, 484). Gene conversion was proved (686), and its important characteristics were delineated (e.g., references 143, 362, 720, 907 and 1015). Meiotic recombination within genes was shown to be polarized (720). Genes that regulate the frequency of locally specific recombination events within and between other genes were discovered and characterized (see references 167 and 170). The complete meiotic karyotype was reconstructed in three dimensions for the synaptonemal complex, with its associated recombination nodules (see reference 396). Genetic polymorphisms were shown to be abundant in natural populations of *Neurospora*—not a foregone conclusion for a vegetatively haploid microorganism (601, 730, 1002). Electrophoretic and other variants from wild-collected strains proved a valuable adjunct to the mutants obtained from laboratory strains by conventional means (820) (for examples, see entries below for *leu-5, het, ars, mig, ss*, and *pts*).

An abundance of evidence from *Neurospora* established the basic similarity of genetic mechanisms in fungi, with their small DNA genomes, to those of higher eucaryotes. This applies to meiosis, crossing-over and interference, gene conversion and intragenic recombination, chromosome rearrangements, genome organization, chromosome composition and structure, and the presence of systems of meiotic drive (see references 571, 791, 808 and references therein, and 1092). In the realm of gene action, genetic and biochemical studies with *Neurospora* have contributed basic information on biosynthetic pathways, gene-enzyme relations (see reference 343), regulation (237, 427, 642, 665), transport (921, 1150, 1151), circadian rhythms (328), and the interplay between chromosomal and mitochondrial genomes in specifying organelle structure (see reference 394).

These and other investigations with *Neurospora* have resulted in a vast literature, gathered in bibliographies (36–38, 373); the first two of these have been thoroughly indexed by subject. The present review brings together the widely scattered information on genetic properties, phenotypes, and map relationships of all the known genes.

GENETIC NOMENCLATURE AND CONVENTIONS

Usage in this review reflects current practice of *Neurospora* workers and of the Fungal Genetic Stock Center (FGSC) (43, 44, 52, 807). The basic *Neurospora* conventions antedate bacterial genetic nomenclature and follow those of *Drosophila* more closely. Gene symbols are written in lower-case italics (e.g., *nmr*) unless the mutant allele is known to be dominant; the first letter is then capitalized (e.g., *Ban*). (Mating type alleles, *A* and *a*, are an exception.) A symbol without superscript is used to represent the mutant allele. The same symbol with a superscript plus (+) designates the wild-type allele. Multiple alleles or alleles differing in resistance or sensitivity, or allelic series having no definitive wild type, may be distinguished by other superscripts, e.g., *cyh-1*^{R}, *cyh-1*^{S}, *het-6*^{OR}, *het-6*^{PA}, T^{L}, T^{S}.

Although the basic letter symbols for many auxotrophic loci are the same as for those of bacteria, locus symbols for *Neurospora* often consist of fewer than three letters. Nonallelic "mimic" loci having the same descriptive letter symbol are distinguished from one another by numbers following the symbol rather than by capital letters as for bacteria. A hyphen separates the distinguishing number from the common letter symbol (e.g., *ilv-2, ilv-3*).

Allelic mutations bear identical locus symbols and numbers. Each new mutation is assigned a unique allele number (often called an isolation number) to distinguish it from all allelic mutations of independent origin. (Allele numbers are commonly prefixed by letters indicating the laboratory of origin.) Allele numbers are not usually displayed with the gene symbol, except when necessitated by the use of several alleles, when it is included in parentheses after the full locus symbol, e.g., *pyr-3* (KS43), or when a new mutant gene has not yet been assigned a locus number pending tests for allelism with similar genes at previously established loci. In the latter situation, a mutant gene is temporarily designated by an appropriate letter symbol followed immediately by the allele number in parentheses, e.g., *ilv(STL6)*. When new locus symbols, locus numbers, and allele number prefixes are to be assigned, it is advisable to consult other workers and the latest FGSC stock list to avoid duplication.

Regulatory genes have usually been given the same basic letter symbol as the structural genes they regulate (e.g., *nit-2, leu-3, cys-3*), but this is not always true (e.g., *pcon, pgov, scon, ty*). Suppressors are symbolized by "*su*" followed immediately by the symbol of the suppressed gene in parentheses; locus numbers of nonallelic suppressors of the same gene follow the parentheses [e.g., *su(met-7)-1, su(met-7)-2*]. As for *Drosophila*, "*su*$^{+}$" designates the wild type, and "*su*" designates the mutant suppressor allele. For allele-specific suppressors, the allele number is included as a superscript of the locus symbol, e.g., *su(trp*^{td201}*)-1*. Conventions are

similar for supersuppressors (nonsense suppressors), with the basic symbol *ssu*.

A few symbols that were previously ambiguous or imprecise have been revised here, with the agreement of the investigators concerned.

Alleles at several gene loci originated in other *Neurospora* species and have been introgressed into *N. crassa*. Those species capable of gene exchange by way of sexual crosses all appear to be similar in chromosome sequence (808; E. G. Barry, personal communication). Introgressed markers have, therefore, been treated as though they had arisen as mutations within *N. crassa*.

Genetic linkage groups are designated by Roman numerals, and cytologically defined chromosomes are designated by Arabic numbers. Linkage group arms are conventionally designated as right (R) and left (L). Heterokaryons are represented by genotypes of the component nuclei enclosed in parentheses, e.g., (*al-2 arg-6 A + arg-1 al-1 A*). Mitochondrial variants are designated by italicized symbols enclosed in brackets, e.g., [*mi-3*]. Chromosome rearrangements include translocations (symbol *T*), inversions (*In*), transpositions within the same chromosome (*Tp*), and duplications (*Dp*). This symbol is followed by the linkage groups involved, in parentheses, and an identification number. With reciprocal translocations, the linkage groups are separated by a semicolon (e.g., *T(I;II)4637*, a reciprocal interchange between linkage groups I and II). With insertional or terminal rearrangements, the linkage groups are separated by an arrow indicating which is the donor and which the recipient of the transferred segment [e.g., *T(I→II)39311*, wherein a segment of linkage group I is inserted into II].

For more explicit recommendations regarding genetic nomenclature, see the references mentioned at the beginning of this section.

LINKAGE MAPS

Genetic linkage information on chromosomal loci is summarized in Fig. 1 through 7. Loci whose order is established with reasonable certainty are displayed on the maps. Many loci are not displayed, but are listed below the maps because their order has not been completely established relative to loci shown on the map. The meaning of each locus symbol, data on linkage with nearby markers, and further detailed information will be found in the section Information on Individual Loci, where genes are listed alphabetically by symbol.

Relative distances on the maps are only rough approximations. *rec* genes with local effects differ in many of the strains used for mapping, and recombination values for the same interval have been shown to vary 10-fold or more in crosses of different parentage (167, 169, 170, 174). For this reason, no attempt has been made to correct for undetected multiple crossovers in long intervals by using a mapping function.

Linkage group I is estimated to be at least 200 map units long, and the total for all seven groups probably exceeds 1,000 (808). Because of the genetically determined variability, no map scale is indicated graphically. Interval lengths were estimated as composite or representative values based on all the crosses available and are shown on the scale ca. 1 cm = 7% recombination. It must be stressed that absolute and relative interval lengths shown on the maps possess only limited predictive value, depending on the genotype of a particular cross with respect to genes that determine the frequency of recombination in each local region. In contrast, gene order is constant in the absence of rearrangement.

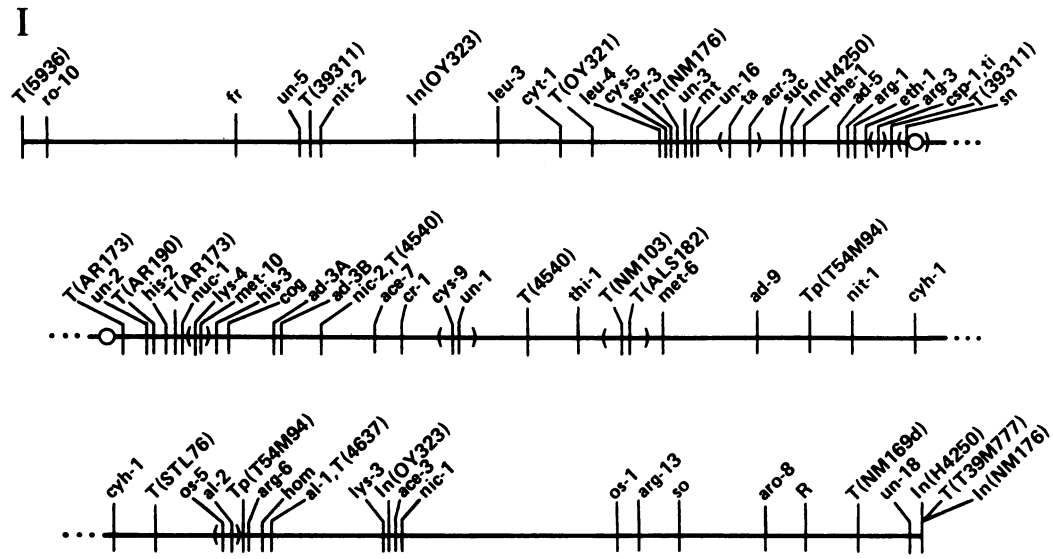
Because of *rec* gene differences, gene order cannot be established reliably by combining two-point recombination values from different crosses. Seriation has, therefore, been based wherever possible either on meiotic crossing-over data from three-point crosses or on duplication coverage. Duplication-producing rearrangements such as insertional or terminal translocations allow a simple test to be performed from which it can be determined whether a locus is to the right or left of a rearrangement breakpoint (see, for example, references 798, 808, and 816). Breakpoints are shown on the maps only for those rearrangements that have been used to establish gene order or to map chromosome tips and centromeres.

Crosses involving strains with a history of transformation have not been used in constructing standard maps because genes may be inserted in abnormal positions, and we recommend that new loci not be defined on the basis of mutations that originated in pedigrees from transformed parents until more is understood about the transformation process. (See the entry for *os-6*.)

INFORMATION ON INDIVIDUAL LOCI

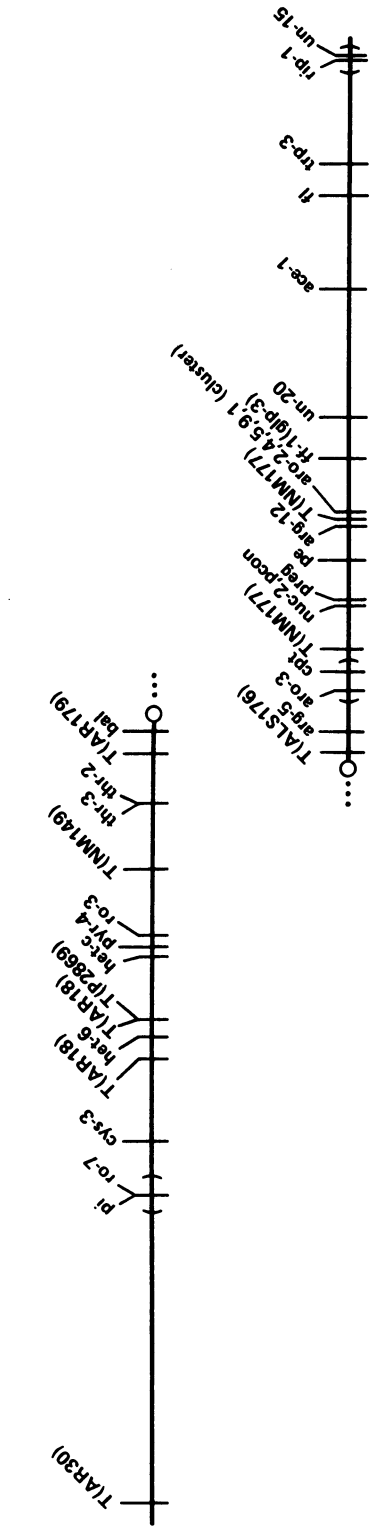
Gathered in this section is information on each established gene locus and on chromosomal features such as centromeres, tips, and the nu-

FIG. 1. Linkage group I of *N. crassa*. The map shows loci whose order is established with reasonable certainty. Order of markers in parentheses is uncertain. The centromere is represented as a circle. The order of genes listed below the map is less well established relative to loci shown on the map; percentages and fractions indicate recombination among random ascospore progeny, unless stated otherwise. For detailed information and documentation, see alphabetical entries under "Information on Individual Loci."



ADDITIONAL LOCI IN LINKAGE GROUP I

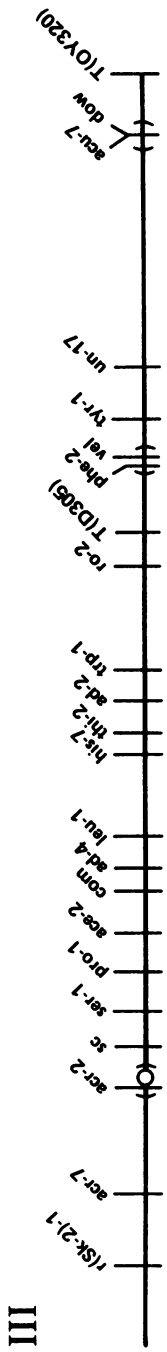
acr-1	Linked to *mt* (8 to 12%)
acr-4	Linked to *mt*, *acr-3* (5%)
acu-4	Right of *arg-1* (5/29)
age-1	Linked to *so* and *aro-8*
age-3	Right of *un-18*
amyc	Between *ad-5* and the centromere
aro-7	Between *arg-1* (4%) and *his-3* (1 to 2%)
atr-1	Between *In(H4250)* and *T(39311)*
aza-1	Left of *mt* (23%)
aza-2	Linked to *mt* (2%) and *aza-1* (39%)
B^{en}	Left of *nit-2* (30%)
Ban	Left of *mt* (14%)
bs-1	Linked to *un-1* (9%), probably to the right
cnr	Linked to *hom* (1%), probably to the right
col-12	Linked to *mt* (17 to 22%)
cr-2	Between *T(NM103)* and *al-2* (18%)
cr-3	Between *T(4540)* and *cr-2*
csh	Between *thi-1* (12 to 20%) and *ad-9* (5%)
cy	Linked to *ad-5* (1/54), probably to the left
cya-1	Linked to *mt* (6%)
cys-11	Linked to *cys-5* (<1%), probably to the right
cys-12	Right of *ad-9* (12%), linked to *al* (0/76)
cys-13	Right of *his-3* (2%)
cyt-4	Between the *T(AR173)* breakpoints
cyt-18	Linked to *al-2* (10%) and *nic-1* (1 to 5%)
dot	Linked to *ad-9* (0/44); right of *thi-1* (2%)
En(pdx)	Linked to *mt* (5%), probably to the left
erg-4	Linked to *al-1* (10%)
ff-3	Right of *os-1* (3%)
fls	Between *nit-1* (5 to 19%) and *al-1* (6 to 19%)
fmf-1	Linked near *arg-1*
fpr-5	Left of *al-2* (25%)
fs-3	Left of *mt* (16%)
Fsp-2	Right of *nic-2* (6%)
glp-1	Linked to *ad-9* (2%), probably to the right
glp-5	Left of *cr-1* (15%)
gsp	Left of *mt* (10%)
gua-1	Probably between *his-2* (3%) and *cr-1* (3%)
het-5	Between *T(NM103)* and the right tip
ipa	Between *mt* (20%) and *arg-1* (1%)
lis-1	Between *ad-3* (6%) and *al-1* (16%)
lys^{R}	Between *his-3* and *nic-2*
mb-2	Between *cyh-1* (5%) and *al-1* (7%)
mb-3	Linked to *cyh-1* (18%), *al-1* (2%), and *mb-2* (6%)
mei-3	Between *arg-1* (3%) and *T(39311)*
mig	Between *tre* (≪1%) and *ad-9*
mo-1	Linked to *mt* (9%)
mo-5	Linked to *mt* (20%)
mus-9	Between *cyh-1* (18%) and *al-2* (6%)
mus(SC28)	Right of *al-1* (18%)
nd	Between the centromere (15%) and *al-2* (20%)
nit-8	Linked to *nit-1* (32%) and *mt* (10%)
nuh-6	Between the centromere (5%) and *nic-2* (4%)
nuh-8	Right of *nic-1*
os-4	Between *T(39311)* and *T(AR173)*
pat	Linked to *mt*, probably to the right
prd-3	Near the centromere
rec-3	Between *acr-3* (1 to 2%) and *arg-3* (2 to 8%)
rg-1	Between *T(AR173)* and *lys-4* (1 to 7%)
rg-2	Linked to *mt* (15%)
ro-6	Between the *T(4540)* breakpoints
sar-1	Near *mt*
sdh-1	Linked to *mt* (0/13)
sf	Linked to *mt* (3%) and *cy* (3%)
slo-1	Between *mt* (14%) and *thi-1* (2 to 5%)
smco-1	Linked to *mt* (1%) and *rg-1* (0/72)
smco-3	Linked to *mt* (10%) and *al-2* (29%)
smco-5	Linked to *mt* (2%)
sor-4	Linked to *phe-1* (<1%)
sor(T9)	Between *mt* (6%) and the centromere (5%)
spco-11	Linked to *mt* (17%) and *mo-5* (18%)
spco-12	Linked to *mt* (20 to 35%) and *mo-5* (5%)
ss	Very near *nit-2*
ssu-2	Linked to *mt* (22%) and *al-2* (26%)
ssu-3	Linked to *mt* (22%) and *al-2* (33%)
st	Between *ad-3B* (5%) and *thi-1* (14%)
su(bal)	Linked to *mt* (13%)
su(col-2)	Linked to *mt* (0/837)
su(met-7)-1	Linked to *al-2* (1%)
su([mi-3])-1	Linked to *al-2* (4%)
su(mtr)-1	Right of *his-2* (2%)
su(trp-3^{id2})-2a	Linked to *al-2* (15%)
T	Between *ad-3A* (18%) and *al-2*
tet	Linked to *acr-3* (2%) and *ad-3B* (1%)
tre	Right of *met-6* (7%)
ty-2	Right of *al-2*
tyr-2	Linked to *cr-1* (2%), probably to the left
tys	Right of *mt* (6%)
uc-2	Linked to *mt* (0/12 asci)
un-7	Between *T(STL76)* and *al-1* (3%)
upr-1	Between *mt* (2%) and *arg-1* (7%)
ure-4	Linked to *his-3* (1%), probably to the right
uvs-6	Linked to *met-6* (<1%)
wc-2	Between *T(ALS182)* and *ad-9* (7 to 20%)
ylo-2	Between *In(H4250)* and *arg-1* (1%)



ADDITIONAL LOCI IN LINKAGE GROUP II

acu-5	Linked to *arg-5* (6%) and *aro-3* (7%)	*het-d*	Right of *fl* (25%)
alc-1	Linked to *pe* (10%)	*lp*	Right of *thr-3* (10%)
aod-2	Linked to *arg-5* (7%) and *thr-3* (16%)	*mus-7*	Between *arg-5* (8 to 12%) and *nuc-2* (11%)
col-10	At or near *pi*	*nuh-5*	Linked to *trp-3* (30%); near *T(4637)*
cot-5	Right of *T(P2869)*; probably left of *pyr-4*	*(pcon)*	(Probably allelic with *nuc-2* [0/854])
cwl	Linked to *arg-5* (3%)	*ro-9*	Linked to *thr-3* (0/63); right of *T(NM149)*
cya-4	Near *thr-3*	*scr*	Linked to *arg-12* (2%), probably to the left
cyb-3	Left of *ro-3* (9%)	*spco-14*	Linked to *arg-5* (7%)
cyt-12	Between *thr-3* (38%) and *trp-3* (18%)	*su([mi-1])-3*	Linked to *fl* (31%)
da	Linked to *thr-3* (3%)	*su([mi-1])-4*	Linked to *fl* (22%) and *arg-5* (40%)
eas	Linked to *rip-1* (1/151), *trp-3* (0/71), and *fl* (1/52)	*su([mi-1])-10*	Linked to *arg-5* (29%) and *fl* (24%)
en(am)-2	Near *pe*	*tng*	Between *T(NM149)* and *arg-5* (6 to 14%)
(fs-2)	(Tentative linkage *cot-5* [14/48])	*tu*	Between *pe* (8%) and *fl* (19%)
Fsp-1	Right of *pe* (4%)	*uc-1*	Linked to *pyr-4* (31%)
glp-2	Between *T(ALS176)* and *T(NM177)*	*ure-3*	Between *arg-12* (7 to 12%) and *ace-1* (14%)
		xdh-1	Linked to *pe* (14%)

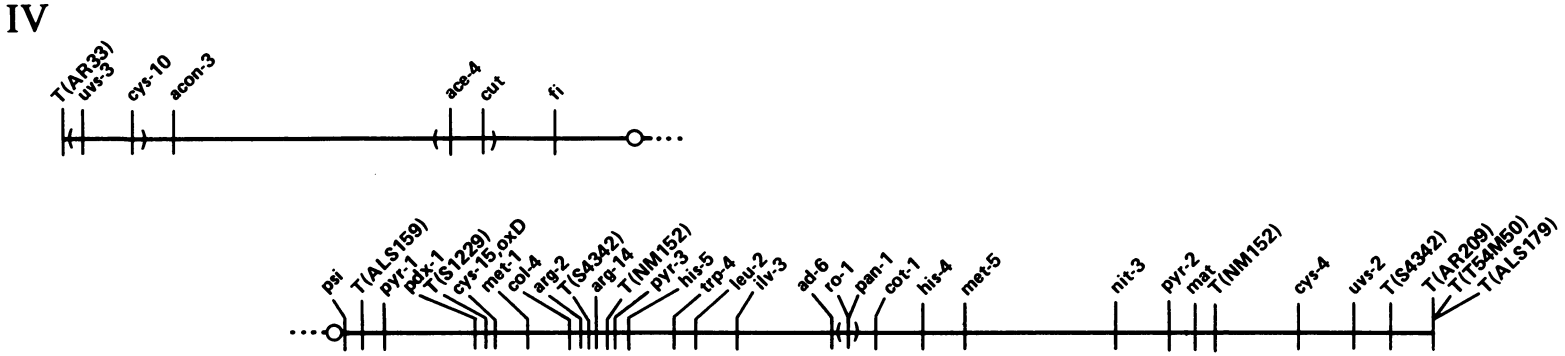
FIG. 2. Linkage group II of *N. crassa*. Scale and conventions as for Fig. 1.



ADDITIONAL LOCI IN LINKAGE GROUP III

acon-2	Linked to *vel* (6%) and *tyr-1* (9 to 14%)	*ota*	Linked to *pro-4* (4%); between *ad-4* (15%) and *tyr-1* (14%)
acr-6	Linked to *shg* (0/368)	*pgov*	Linked to *tyr-1* (1 to 4%), probably to the right
aza-3	Linked to *trp-1* (14%)	*prd-1*	Linked to *acr-2* (5%) and *pro-1* (20%)
col-16	Linked to *leu-1* (1%) and *pro-1* (10%)	*pro-4*	Linked to *thi-2* (0/78) and *ota* (4%)
cum	Linked to *acr-7* (8 to 18%), *acr-2* (18%), and *Sk-2*	*ser-5*	Linked to *trp-1* (1%) and *ser-1* (12%)
cya-7	Linked to *ad-4* (25%)	*shg*	Linked to *trp-1* (7%)
cyt-8	Linked near *ad-4*	*Sk-2*	Left of *his-7* (25%)
erg-3	Linked to *dow* (10%), probably to the left	*Sk-3*	Left of *his-7* (11%)
ff-5	Between *pro-1* (2%) and *mei-8* (1%)	*sor-3*	Linked to *ad-4* (7%)
ff-6	Linked near *ty-1*	*spg*	Linked to *sc* (<1%) and *thi-4* (0/103)
fpr-3	Linked to *trp-1* (1%) and *thi-2* (5%)	*su([mi-1])-1*	Linked to *ad-4* (14%)
gluc-1	Linked to *dow* (10%)	*su(trp-3^{ad})-2*	Linked to *leu-1* (22%)
het-7	Between *T(D305)* and the right tip	*thi-4*	Linked to *acr-2*, *spg*, and *sc* (<1%)
mei-4	Linked to *leu-1* (12%), probably to the left	*trk*	Linked to *leu-1* (0/92)
mei-8	Between *ff-5* (1 to 4%) and *ad-4* (4%)	*ty-1*	Linked to *tyr-1* (0 to 6%) and *dow* (21%)
mo-4	Right of *leu-1* (8%)	*un-6*	Right of *sc* (21%) and *acr-2* (6 to 20%)
nit-7	Between *trp-1* (26 to 32%) and *dow* (45%)	*un-14*	Right of *acr-2* and *thi-4* (8 to 20%); left of *leu-1* (5%)
nuh-1	Between *leu-1* (4%) and *nuh-2* (<1%)	*un-21*	Between *acr-2* and *un-6*
nuh-2	Between *nuh-1* (<1%) and *trp-1* (11%)	*uvs-4*	Left of *ad-4* (4%)
		uvs-5	Linked to *vel* (1%)

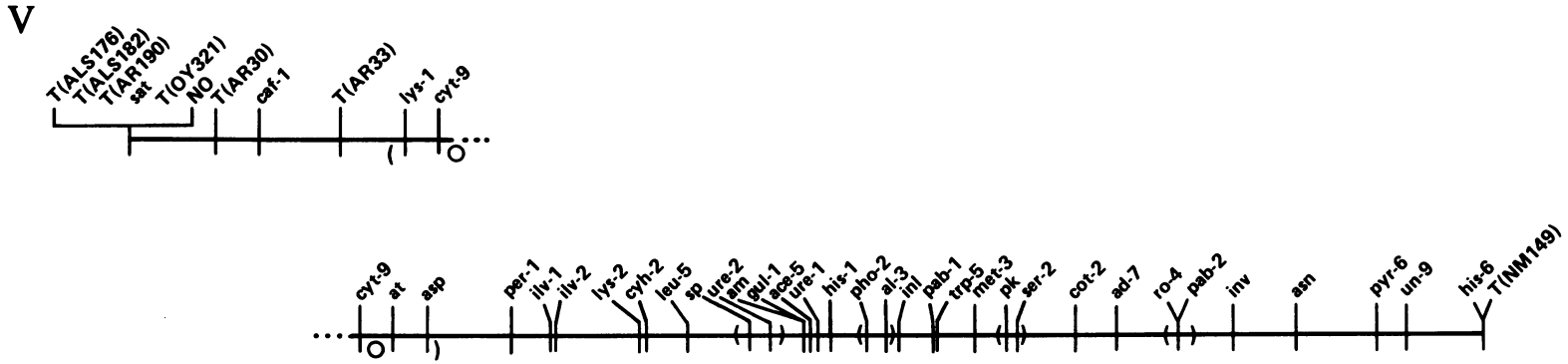
FIG. 3. Linkage group III of *N. crassa*. Scale and conventions as for Fig. 1.



ADDITIONAL LOCI IN LINKAGE GROUP IV

acu-2	Between *leu-2* (11%) and *pan-1* (6%)
ads	Linked to *col-4*
aod-1	Left of *trp-4* (23%)
bd	Right of *pan-1* (2%)
car	Linked to *cys-10* (1%)
cel	Linked to *pan-1* (1%)
chol-1	Linked to *ad-6* (1%), probably to the right
coil	Between *arg-2* (2%) and *leu-2* (12%)
col-1	Linked to *pan-1* (0/47 asci) and *cot-1* (3%)
col-5	Linked to *cot-1* (1%), probably to the right
col-6	Linked to the centromere (0/28 asci) and *pan-1* (21%)
col-8	Linked to *pan-1* (4 to 13%)
cot-3	Left of *pan-1* (16 to 25%); probably right of *arg-2*
crib-1	Linked to *met-1* (6%)
cya-5	Right of *pan-1* (2%)
cya-6	Right of *pan-1* (2%)
cys-14	Linked to *cot-1* (21%)
cyt-5	Left of *trp-4* (9%)
cyt-19	Linked to *cyt-5* (1/201)
dn	Linked to *mat* (1%); right of *pyr-2* (4%)
fdu-2	Right of *cys-4* (2%)
fld	Left of *his-5* (2%)
gpi-1	Linked to *ad-6* (10%)
gua-2	Linked to *cot-1* (5%)
gul-3	Linked to *cot-1* (10%) and *pyr-2* (7%)
int	Linked to *pan-1* (0/50)
le-1	Linked to *pan-1* (1 to 2%); right of *cot-1* (14%)
med	Linked to *met-5* (5%) and *pan-1* (8%)
mei-1	Linked to *arg-2* (<1%), probably to the right
met-2	Linked to *ilv-3* (0/129); between *trp-4* (6%) and *pan-1* (4%)
mod(sc)	Linked to *pan-1* (17%)
moe-3	Left of *pan-1* (17 to 25%) and *bd* (18%)
mtr	Between *pdx-1* (2%) and *col-4* (1%)
mus-8	Linked to *mtr* (1%) and *pdx-1* (6%)
nada	Left of *ad-6* (18%)
nit-4	Linked to *col-4* (2%), probably to the right; left of *pan-1* (6 to 27%)
nit-9	Right of *nit-4* (9%)
os-2	Right of *cot-1* (4%)
pf	Linked to *mat* (3%); right of *pyr-2* (2%)
pho-3	Linked to *pan-1* (<1%)
pmb	Right of *uvs-2* (8%)
pt	Right of *T(S1229)* and *pdx-1* (2%); left of *col-4* (2%)
rib-2	Between *T(S4342)* and *chol-1*
rol-1	Linked to *pdx-1* (0/88)
ser-4	Right of *arg-2* (<1%)
smco-4	Linked to *pan-1* (8%)
smco-8	Linked to *pan-1* (1 to 7%) and *smco-4* (30%)
smco-9	Linked to *pan-1* (5%), *smco-4* (2%), and *smco-8* (13%)
spco-8	Linked to *pan-1* (23%)
su([mi-1])-14	Linked to *arg-2* (14%)
thi-5	Linked to *pan-1* (1%)
tol	Linked to *trp-4* (1%), probably to the left
uc-5	Right of *cot-1* (32%)
un-8	Linked to *pyr-1* (0/47); between *T(ALS159)* and *col-4* (5%)
un-12	Linked to *col-4* (0/73) and *pdx-1* (5%)

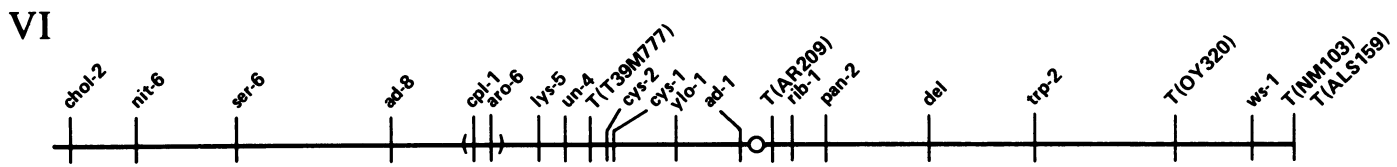
FIG. 4. Linkage group IV of *N. crassa*. Scale and conventions as for Fig. 1.



ADDITIONAL LOCI IN LINKAGE GROUP V

acu-1	Right of *asn* (21%)
acu-3	Between *inl* (7%) and *asn* (20%)
arg-4	Between *sp* (1 to 11%) and *inl* (2 to 4%)
cl	Right of *pk* (<1 to 2%)
col-9	Between *inl* (16%) and *asn* (5%)
cot-4	Between *rol-3* (5%) and *inl* (10%)
cya-2	Linked to *al-3* (3%) (indicated to the right)
cyb-1	Between *al-3* (24%) and *his-6* (10%)
dgr	Linked to *caf-1* (8%)
en(am)-1	Between *am-1* (8%) and *inl* (1%)
erg-1	Between *pk* (2%) and *asn* (9%)
erg-2	Left of *inl* (6%)
fpr-1	Linked to *cyh-2* (<1%)
fpr-4	Right of *inl* (11%)
gln-1	Linked to *inl* (2%), probably to the right
glp-6	Left of *inl* (30%)
gran	Linked to *pl* (0/75); between *pab-2* (1 to 8%) and *his-6* (8 to 27%)
hgu-4	Between *cyh-2* (7%) and *ure-2* (10%)
Iasc	Right arm
lis-3	Right of *inl* (4%)
md	Between *sh* (3%) and *sp* (9%)
Mei-2	Linked near *inl*
mus-11	Linked to *his-6* and *pab-2*
mus(SC17)	Left of *inl* (27%)
nap	Linked to *inl* (15%); right of *ure-2* (32%)
ndc-1	Left of *arg-4* (2%)
nmr-1	Between *am* (3 to 7%) and *gln-1* (4 to 10%)
nuh-3	Between *cyh-2* (4%) and *al-3* (17%)
nuh(23)	Linked to *nuh-3* (6%)
pl	Linked to *gran* (0/75); between *asn* (1 to 9%) and *his-6* (16%)
prd-2	Right arm
pro-3	Linked to *inl* and *pab-1* (0/74); between *his-1* (4%) and *pk* (2 to 6%)
rec-1	Between *ro-4* (7%) and *asn* (5%)
rec-2	Between *sp* and *am*
rol-3	Between *ilv-1* (2%) and *cot-4* (5%)
scon	Probably linked to VR; right of *his-6*
scot	Between *al-3* (7%) and *his-6* (11%)
sh	Between *ilv* (4 to 7%) and *md* (3%)
smco-6	Linked to *asn* (6%); near *pyr-6*
smco-7	Linked to *rol-3* (0/154); right of *ilv-1* (2%)
spco-9	Linked to *asn* (6%); right of *met-3* (18%)
spe-1	Linked to *cyh-2* (2%); left of *inl* (14%)
su(ile-1)	Right of *met-3* (4%)
su([mi-1])-f	Left of *inl* (10%)
ts	Linked to *inl* (4%)
uc-4	Between *inl* (12%) and *his-6* (11%)
udk	Left of *uc-4* (29%)
un-11	Linked to *al-3* (0/48)
un-19	Linked to *al-3* (9%) and *un-11* (14%)
val	Linked to *ilv-2* (0/135)
wa	Linked to *inl* (6%)

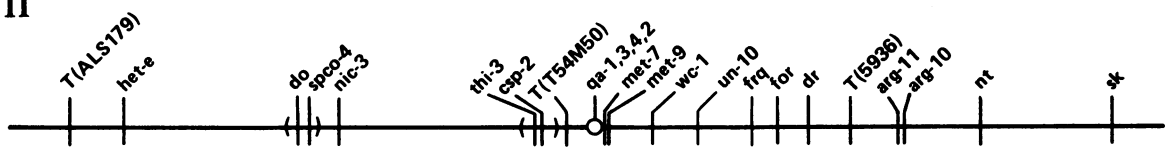
FIG. 5. Linkage group V of *N. crassa*. Scale and conventions as for Fig. 1.



ADDITIONAL LOCI IN LINKAGE GROUP VI

acu-6	Left of *cys-1* (3%)
age-2	Right of *ws-1*
Bml	Linked to *cys-2* (2%) and *ylo-1* (2 to 3%), probably between
chr	Between *chol-2* (10%) and *pan-2*
cpc-1	Right of *ylo-1* (1–3%)
cya-3	Between *chol-2* (10%) and *cyt-2* (10%)
cys-2	Between *un-4* (4%) and *cys-1* (1 to 3%)
cyt-2	Between *cya-3* (10%) and *lys-5* (6%)
edr-1	Linked to *ad-1* and *pan-2* (0/125)
edr-2	Left of *ad-1* (19%)
fpr-6	Between *pan-2* and *trp-2*
glp-4	Between *ad-1* (0 to 2%) and *rib-1* (3 to 4%)
gul-5	Linked to *trp-2* (10%)
het-8	Between *chol-2* (19%) and *ad-8* (12%)
het-9	Between *T(AR209)* and the right tip
lis-2	Between *chol-2* (11%) and *trp-2* (25%)
mod-5	Linked to *trp-2*, near the centromere (3%)
moe-2	Linked to *trp-2* (14%), probably to the left
mts	Right of *ylo-1* (<1%)
sor-1	Left of *ylo-1* (3%)
spco-7	Linked to *ad-1* (0/65), between *ylo-1* (4%) and *T(AR209)*
spco-13	Linked to *trp-2* (16%) and the centromere (1/10 asci)
ssu-7	Between *ad-8* (8%) and *ylo-1* (14%)
su(mtr^{26})	Linked to *trp-2*, in the right arm
su(trp-5)	Near *aro-6* (allelic?)
un-13	Linked to *ylo-1* (2%) and *lys-5* (4%)
un-23	Right of *trp-2* (5 to 27%); left of *ws-1*
ws-2	Linked to *trp-2* (2%) and *ylo-1* (16%)

FIG. 6. Linkage group VI of *N. crassa*. Scale and conventions as for Fig. 1.



ADDITIONAL LOCI IN LINKAGE GROUP VII

adh	Linked to *do* (0/53), *spco-4* (4%), and *nic-3* (11%)
aga	Between *wc-1* (2%) and *arg-10* (24 to 27%)
aln-1	No data
ars	Right of *thi-3* (2 to 5%); left of *ile-1* (1%) and *met-7* (<1%)
bn	Linked to *thi-3* (2%); right of *T(T54M50)*
col-2	Linked to *met-7* (1%)
col-3	Linked to *met-7* (0/93) and *wc-1* (1%)
col-17	Linked to *nt* (14%) and *spco-5* (6%)
cyt-6	Linked to *wc-1* (2%) (indicated left)
cyt-7	Linked to *nic-3* (18%) (indicated left)
gul-4	Linked to *nic-3* (17%)
het-10	Between *T(5936)* and the right tip
hlp-1	Between *sfo* (1 to 9%) and *nt* (28 to 37%)
hlp-2	Between *hlp-1* (8 to 25%) and *nt* (29%)
ile-1	Between *ars* (1%) and *wc-1* (<1 to 2%)
kyn-1	Linked to *nic-3* (30%) and *wc-1* (20%)
le-2	Linked to *met-7* (7%)
mb-1	Linked to *nic-3* and *wc-1* (23%)
mel-1	Left of *thi-3* (27%)
mo-2	Linked to *nt* (29%)
mus-10	Right of *met-7* (7%)
oli	Linked to *frq* (<2%)
rol-2	Linked to *met-7* (0/298)
sfo	Between *thi-3* (6%) and *hlp-1* (1 to 9%)
slo-2	Left of *met-7* (2%)
sor-2	Linked to *nt* (31%)
spco-5	Linked to *col-17* (6%) and *nt* (20%)
spco-6	Linked to *do* (10%), *spco-5* (8%), and *nt* (20%)
ssu-1	Between *met-7* (14%) and *su(trp-3^{td201})-1* (10%)
ssu-4	Between *nic-3* (28%) and *met-7* (20%)
su([mi-1])-5	Left of *nic-3* (23%)
su(trp-3^{td201})-1	Right of *ssu-1* (10 to 13%); left of *arg-10* (7%)
su(trp-3^{td201})-2	Linked to *su(trp-3^{td201})-1*, probably to the left
ud-1	Between *met-7* (27%) and *arg-10* (10%)
un-22	Linked to *met-7* (1%)
van	Left of *nic-3* (4%)

FIG. 7. Linkage group VII of *N. crassa*. Scale and conventions as for Fig. 1.

cleolus organizer. Entries are arranged alphabetically by symbol. Some categories of genes are prefaced by a generic entry which concerns the entire group (e.g., *al, arg, mus, rec*, Transport mutants). Synonyms or abandoned symbols are cross-referenced to the current or preferred symbol. Chromosome rearrangements are listed only if they have been used to establish gene order or to map chromosome tips and centromeres.

Within each entry for a gene locus, the name follows the symbol. Linkage data are then given, including linkage group, arm (if known), and location relative to other loci. Representative recombination values are given as percent crossovers among random ascospores unless otherwise specified. Ranges are given. Many of these cannot be attributed to sampling error, but are thought to reflect differences in genes having locally specific effects on recombination (see *rec*). The phenotype is described in the second paragraph. Information is given concerning such attributes as enzyme deficiency, dominance, interaction with other genes, fertility, properties of specific alleles, stability, scoring, technical applications, and alternative names.

In the paragraph on linkage we have cited mainly the most recent, definitive datum sources that establish location of a gene relative to its immediate neighbors. In addition, we have cited the earliest publication that originally assigned the locus to its linkage group; this reference is set in italics. Where an italicized reference number is separated from the previous sentence by a period, it signifies that the reference has not contributed to the recombination data given and is cited solely because it first assigned the gene to a linkage group. It is regrettable that all those investigators whose earlier data indicated gene order less precisely but provided intermediate steps that were essential for deriving the current maps cannot be credited. Substantial mapping data have been published in references 47, 156, 158, 219, 482, 633, 692, 698, 789, 812, 814, 816, 818, 991, 1012, and 1036.

Similarly, in the paragraph on phenotype, space limitations preclude citing all the important references for loci that have been studied extensively; the references selected should lead the reader to other significant literature. Some reference numbers in parentheses are set off from the previous sentence by a period. This signifies that their relevance is not limited to the immediately preceding sentence.

When "PB" is used in place of a reference number, the source referred to is D. D. Perkins and M. Björkman, unpublished data.

Allele numbers are given only when there is a question about allelism, when two alleles differ significantly in phenotype, or sometimes when a mutant was referred to only by allele number in the references cited. Allele numbers of other mutant strains can be found in FGSC listings or in the references given. When a particular mutant gene has not yet been definitely assigned to a locus, the allele number may be given in parentheses following an appropriate gene symbol, e.g., *nuh(23), cyt(289-56)*. Where allele numbers are not specified, information in an entry is for alleles used in the references cited. A table relating allele numbers to locus symbols is given on p. 31–33 of reference 47.

A few entries (e.g., *pts-1, sit*) concern chromosomal genes that have not yet been mapped but that probably involve previously unidentified loci.

Theses and abstracts have usually been cited only if they contain pertinent information that has not been published or adequately documented in a published reference.

A/a: mating type alleles

IL. Between *un-3* (0.04 to 0.1%) and *un-16* (<1%) (488, 758; D. D. Perkins, unpublished data). (*609*)

Opposite mating types are essential for a complex of events associated with sexual reproduction and morphogenesis: attraction of trichogyne to cells of opposite mating type (39, 93); pickup and transport of the nucleus to the ascogonium; growth and development of the perithecium; proliferation of heterokaryotic ascogenous hyphae; conjugate nuclear divisions in precrozier and crozier cells; karyogamy.

Mating type alleles also act as vegetative incompatibility genes during the vegetative phase. $A+a$ combinations are unable to form stable heterokaryons (66, 384, 830, 914). Vegetative fusion is usually followed by cell death (384), but some $A+a$ heterokaryons grow slowly (252, 412, 422). Heterozygous *A/a* duplications are highly abnormal, with inhibited growth and spider-like morphology (761, 804). Incompatibility in heterokaryons or duplications is relieved by spontaneous deletion of either allele (252, 756). Vegetative incompatibility is not expressed during the sexual phase after fertilization. Both manifestations of vegetative incompatibility are suppressed by *tol*, but sexual compatibility is not affected (755). The vegetative incompatibility is normally suppressed in *N. tetrasperma* (668) and *N. sitophila* (674, 804). Extensive efforts have failed to separate the sexual and vegetative traits by genetic recombination (758). Null mutants selected by loss of vegetative incompatibility usually lose both sexual and

vegetative functions simultaneously (one exception), and both functions are usually restored simultaneously in revertants selected for restoration of fertility (one null mutant gives atypical revertants) (252, 411, 412).

Only two mating type alleles, *A* and *a*, are known. These are apparently homologous throughout the genus *Neurospora* (820) and perhaps in related genera (770). Nothing is known about the genetics of the five true-homothallic species of *Neurospora*, which closely resemble *N. crassa* in karyotype and meiotic behavior, including the fusion of two haploid nuclei in the penultimate cell of the crozier to form the zygote nucleus (855).

In the early literature, *A* was called + (plus) or A, and *a* was called − (minus) or B (e.g., reference 286). The locus may also be designated *mt*, mating type (e.g., reference 808), and is usually referred to as *mt* in the present paper.

aaf: acetylaminofluorine requirement

Data said to be consistent with one gene.

Complex phenotype. Alternative requirements: 2-acetylaminofluorine, certain azo dyes, or certain single amino acids. Cold sensitive. Originated among progeny of a *rib-1* strain that had become tolerant to 2-acetylaminofluorine. Abstract only: 1069.

_ac_: acetate

Changed to *ace*.

ace: acetate

Acetate mutants *ace-1* through *ace-7* are auxotrophs that grow on 0.3% sodium acetate, as do *suc* mutants (which often grow better on acetate than on succinate). A carbon source is also needed. Most *ace* mutants grow better when the carbon source is maltose rather than sucrose (578). Acetate mutants, except *ace-1* and *ace-5*, can grow on Tweens as the sole carbon source (S. Brody, personal communication). The mutants differ in their ability to grow on complex media. Unlinked genes *ace-2*, *-3*, and *-4* are involved with the pyruvate dehydrogenase complex (769). A separate set of acetate mutations called *ac-1*, *-2*, *-3*, *-4*, and *-5* (1034) were not mapped and are not available for testing for allelism with *ace-1* through *ace-7*. General reference: 578.

ace-1: acetate-1

IIR. Right of *un-20* (15%) and *ure-3* (14%). Left of *fl* (2 to 11%) (*47*, PB).

Requires acetate. Poor growth on complex complete medium. Grows well on acetate (0.1%) aided by ethanol (0.5%) (E. L. Tatum and L. Garnjobst, cited in reference 47; PB). Ascospore maturation and germination are slow. Germination is best on sucrose minimal medium with yeast extract and ethanol (L. Garnjobst, personal communication). Not the same as *ac-1* of reference 1034, which was lost. Called *ac*.

ace-2: acetate-2

IIIR. Right of *pro-1* (1 to 9%). Left of *com* (5%) and *ad-4* (4 to 7%) (578). (*812*)

Requires acetate. Will not use succinate or ethanol (290). Lacks pyruvate dehydrogenase complex activity (769). Good growth on complex complete medium (578). Not the same as *ac-2* of reference 1034.

ace-3: acetate-3

IR. Right of *In(OY323)* and, hence, of *lys-3* (0.3 to 1%). Left of *nic-1* (0.2%). Included in duplications from *In(OY323)* × *In(NM176)*. (2, *57*, *578*, 907)

Requires acetate. Lacks pyruvate dehydrogenase complex activity (769). Poor growth on complex complete medium (578). Conidiation best at 25°C, not 34°C (D. D. Perkins, unpublished data). Not the same as *ac-3* of reference 1034.

ace-4: acetate-4

IVL. Between *cys-10* (19 to 33%) and *fi* (10 to 17%) (*578*).

Requires acetate. Grows on complex complete medium (578). Lacks pyruvate dehydrogenase complex activity. Lipoate acetyltransferase fails to aggregate to form the core of the pyruvate dehydrogenase complex. As a result, there is high activity of the free components pyruvate dehydrogenase and lipoamide reductase (769). Not the same as *ac-4* of reference 1034.

ace-5: acetate-5

VR. Between *gul-1* (<1%) and *ure-1* (<1%) (577, *578*).

Requires acetate. Poor growth on complex complete medium (578). Not the same as *ac-5* of reference 1034.

***ace-6*: acetate-6**

See *suc*.

***ace-7*: acetate-7**

IR. Between *nic-2* (4 to 7%) and *cr-1* (1 to 3%) (*578*).

Requires acetate. Good growth on complex complete medium. Unable to use xylose as a carbon source; resembles *suc* mutants and differs from the wild type and all other *ace* mutants in this respect. Normal pyruvate dehydrogenase and pyruvate carboxylase activities. (578)

***acon(-1)*: aconidiate**

See *fl*.

***acon-2*: aconidiate-2**

IIIR. Linked to *vel* (6%) and *tyr-1* (9 to 14%), probably between them (*648*, PB).

Macroconidiation defective. Allele RS91 is heat sensitive, with macroconidiation blocked at 34°C. Some conidia are formed at 25°C, but growth is subnormal (648). Homozygous fertile (PB).

***acon-3*: aconidiate-3**

IVL. Between *cys-10* (1 to 6%) and *cut* (33%) (PB). Report of VIL linkage not confirmed.

Macroconidiation blocked (648). Female sterile. Some conidia have been observed low in slants at 25°C (PB).

***acp*^{i}: acetate permease (inducible)**

Not mapped. Not allelic with other acetate utilization (*acu*) mutations.

Lacks inducible acetate transport system. (864, 866)

***acr-1*: acriflavine resistant-1**

IL. Linked to *mt* (8 to 12%) (*498*).

Low-level resistance to acriflavine (2 μg/ml) was found for wild-type STA4 compared with that for wild-type Pa, which is more sensitive (498). Difficult to score.

***acr-2*: acriflavine resistant-2**

III. Linked to *thi-4* (0/286). Left of *sc* (3 to 6%) and *spg* (1 to 11%) (*498*, 816). *acr-2* has been shown left of the centromere on published maps but without direct evidence. *acr-2* and *trp-1* (on IIIR) cosegregated at the second division in 1 of 13 asci (H. B. Howe, Jr., personal communication), which would favor a right arm location.

Resistant to acriflavine (494, 495); also resistant to 3-amino-1,2,4-triazole (seven alleles tested) (494). Resistance is probably dominant (heterokaryon tests) (498). Not resistant to malachite green. An excellent stable marker, fully fertile, with unambiguous scoring. Sizable inocula should be used to avoid false-negative tests. Use acriflavine at 50 μg/ml in minimal agar medium (816) (higher concentrations may be used) and aminotriazole at 0.5 mg/ml; both added before autoclaving.

***acr-3*: acriflavine resistant-3**

IL. Between *un-16* (1 to 5%) and *suc* (1 to 5%). Probably right of *ta* (816; PB). (*498*)

Resistant to acriflavine and to malachite green (three alleles tested). Not resistant to 3-aminotriazole. Resistance is probably dominant (heterokaryon tests) (498). Scoring is clear-cut with uniform inocula of appropriate size. False-negative or false-positive scoring may result if test inocula are too small or too large. May show delayed resistance: read tests at 2 and 4 days, 34°C. Use acriflavine at 10 μg/ml in minimal agar at 34°C (816) and malachite green at 2 μg/ml (498).

***acr-4*: acriflavine resistant-4**

I. Linked to *mt* and *acr-3* (5%) (*499*).

Resistant to acriflavine (50 μg/ml) when *acr-4* is combined with morphological mutation *shg* (499).

***acr-5*: acriflavine resistant-5**

I or II. Linked to *T(IR;IIR)4637 al-1* (*499*).

Resistant to acriflavine (50 μg/ml) when *acr-5* is combined with linked morphological mutation *mo(KH161)* (499).

***acr-6*: acriflavine resistant-6**

IIIR. Linked to *shg* (0/368) (*499*).

Resistant to acriflavine (50 μg/ml). Originated in *shg* and not separated by recombination. Strain of origin is acriflavine sensitive. (499)

***acr-7*: acriflavine resistant-7**

IIIL. Left of *sc* (12 to 14%). Linked to *thi-4* (7%) (PB). Right of *r(Sk-2)-1* (7%) (B. C. Turner, personal communication). Report of VI linkage in reference 818 is incorrect.

Resistant to acriflavine (50 μg/ml). Not cross-resistant to 3-aminotriazole or malachite green. Several *acr-7* strains have become female infertile after vegetative transfer. (PB)

***act*: actidione resistant**

Changed to *cyh* (cycloheximide resistant) (807).

***acu*: acetate utilization**

For a diagram of the pathway, see p. 304 in reference 343. Scored on minimal medium without sugar, using ammonium acetate (3 mg/ml) as the carbon source. The wild type shows sparse but clearly positive growth in contrast to clear blanks for *acu* mutants. Selectable by inositol-less death on acetate medium. *acu-1*, *acu-5*, *acu-6*, and *acu-7* do not behave as respiratory mutants in tetrazolium overlay tests on acetate medium (310).

***acu-1*: acetate utilization-1**

VR. Right of *asn* (21%) (*349*).

Unable to use acetate as a carbon source (349, 350). Selected by inositol-less death on acetate medium.

***acu-2*: acetate utilization-2**

IVR. Between *leu-2* (11%) and *pan-1* (6%) (*349*).

Unable to use acetate as a carbon source. Reduced level of oxoglutarate dehydrogenase. Poor recovery from ascospores (349, 350).

***acu-3*: acetate utilization-3**

VR. Between *inl* (7%) and *asn* (20%) (590). (*349*)

Unable to use acetate as a carbon source (349, 350). Affects isocitrate lyase (350, 590). Some revertants produce temperature-sensitive enzyme (590).

***acu-4*: acetate utilization-4**

IR. Right of *arg-1* (5/29) (*349*).

Unable to use acetate as a carbon source (349, 350).

***acu-5*: acetate utilization-5**

II. Linked to *arg-5* (6%) and *aro-3* (7%) (*349*).

Unable to use acetate as a carbon source. Affects acetyl coenzyme A synthetase (350).

***acu-6*: acetate utilization-6**

VIL. Left of *cys-1* (3%) (76, *349*).

Unable to use acetate as a carbon source (349). Structural gene for phosphoenolpyruvate carboxykinase (76, 350). Strains with some complementing alleles possess protein that is electrophoretically similar to the enzyme; a temperature-sensitive partial revertant allele specifying an abnormally thermolabile enzyme maps at the original locus (76). Interallelic complementation (349).

***acu-7*: acetate utilization-7**

IIIR. Linked to *dow* (0/72) (*PB*).

Unable to use acetate as a carbon source. Poor recovery from ascospores (~25%) (349). Reduced level of oxoglutarate dehydrogenase (350).

***ad*: adenine**

For the purine biosynthetic pathway, see Fig. 8. (For the abbreviations used below, see the legend to Fig. 8.) For the interrelationship of purine, histidine, and tryptophan pathways, see reference 786. The growth of mutants in the terminal (post-AICAR) steps of the adenine biosynthetic pathway is aided by histidine, which has a sparing effect on *ad-1*, *ad-4*, and *ad-8* (661; M. E. Case, personal communication). Mutants carrying some *ad-5* alleles are aided by histidine; others are inhibited (M. E. Case, personal communication). Indole may strongly inhibit adenine mutants (595).

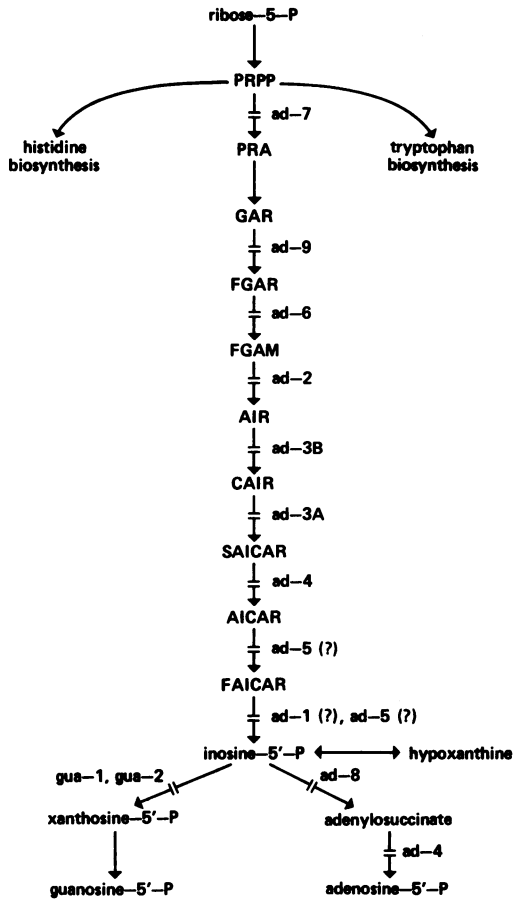


FIG. 8. Purine biosynthetic pathway and sites of action of *ad* and *gua* genes (81, 120, 348, 393, 405a, 511, 525). PRPP, 5-phosphoribosyl pyrophosphate; PRA, 5-phosphoribosylamine; GAR, 5'-phosphoribosyl-glycineamide; FGAR, 5'-phosphoribosyl-formylglycineamide; FGAM, 5'-phosphoribosyl-formylglycineamidine; AIR, 5'-phosphoribosyl-5-aminoimidazole; CAIR, 5'-phosphoribosyl-5-aminoimidazole-4-carboxylate; SAICAR, 5'-phosphoribosyl-5-aminoimidazole-4-*N*-succinocarboxamide; AICAR, 5'-phosphoribosyl-5-aminoimidazole-4-carboxamide; FAICAR, 5'-phosphoribosyl-5-formamidoimidazole-4-carboxamide.

Strains carrying mutations at loci *ad-3A* and *ad-3B* accumulate purple (red) pigment when adenine is limiting; see the *ad-3B* entry. Smaller amounts of the pigment may be seen in strains carrying other post-AIR genes such as *ad-4* and *ad-5* (526, 682). Pigment may be polymerized AIR (786). Mutant genes affecting earlier biosynthetic steps are epistatic to *ad-3* and later mutant genes with respect to pigment production (526). *ad-3B* (and presumably also *ad-3A*) stocks accumulate spontaneous mutations at other *ad* loci, which prevent pigment production and improve the growth rate (691).

ad-3B, *ad-4*, *ad-8*, and *ad-9* mutants have been used to study the effect of histidine on purine pool utilization (786). Regulation of purine catabolism reviewed in reference 642. Regulation of purine biosynthesis (405a, 788). Adenine mutations at the various loci were assigned to complementation groups designated by capital letters (526). The relationships of most of these groups to steps of the biosynthetic pathway are given in Fig. 10 of reference 120.

ad-1: adenine-1

VII. Right of *ylo-1* (6%). Left of the centromere (<1 to 2%), *T(AR209)*, and *rib-1* (3 to 5%) (1102, 1012). (*482*)

Uses adenine or hypoxanthine (682, 824). Accumulates AICAR (81, 904) and SAICAR (81). May affect inosine 5'-monophosphate cyclohydrolase (902, 904) (Fig. 8). Used to study purine transport (903 and references therein). Ascospores are white in homozygous *ad-1* × *ad-1* crosses, and *ad-1* ascospores may be white in heterozygous crosses (D. D. Perkins, unpublished data). Called complementation group M.

ad-2: adenine-2

IIIR. Between *thi-2* (1%) and *trp-1* (1 to 7%) (11, 219). (*482*)

Requires adenine or hypoxanthine (682). Controls conversion of phosphoribosylformylglycineamidine to AIR (120) (Fig. 8). Strains carrying allele 70004(t) are heat sensitive (34°C versus 25°C) (682) and osmotic remediable (636). Called complementation group H.

ad-3A: adenine-3A

IR. Between *his-3* (1 to 2%) and *ad-3B* (0.1 to 0.7%) (271). Right of *ure-4* (78). (*482*)

Requires adenine or hypoxanthine (682). Blocked in interconversion of CAIR plus aspartate to SAICAR (348) (Fig. 8). Produces purple pigment, permitting direct visual selection (276, 682); see the *ad-3B* entry. Reduced interallelic fertility (407). No interallelic complementation (267; F. J. de Serres, personal communication).

ad-3A and *ad-3B* are two genetically and functionally distinct loci separated by a short but functionally complex region of unknown but essential function (271, 407). They have been used intensively for quantitative genetic and molecular studies of mutation (for a review, see reference 35). Either forward mutation (e.g., reference 277) or reverse mutation (e.g., refer-

ence 772) can be measured precisely; the former is detected visually by purple pigment. Purple pigment has also been used to assess the effect of histidine and tryptophan on purine nucleotide synthesis (786). Alleles N23 and N24 have been used as mutagen testers. N23 reverts with agents that cause base pair substitutions; N24 reverts with agents that cause frameshifts (772).

SK(ad-3A) is at or near *ad-3A* and may be a cryptic *ad-3A* allele. Does not require adenine. In *SK(ad-3A)* × *ad-3A* crosses, the *ad-3A* progeny die; possibly *SK(ad-3A)* mutants fail to make enough adenine to support their growth (251). Translocations *Y155M64 ad-3A* (272; PB) and *Y112M15 ad-3A* (413) each have one breakpoint that is inseparable from *ad-3A*. Called complementation group A (264). "*A*" in the locus symbol does not refer to mating type.

***ad-3B*: adenine-3B**

IR. Between *ad-3A* (0.1 to 0.7%) and *nic-2* (3%) (271). (*482*)

Uses adenine or hypoxanthine (682). Blocked in interconversion of AIR to CAIR (348) (Fig. 8). Produces purple pigment, permitting direct visual selection (276, 682). Pigment is secreted with low concentrations of adenine (e.g., 0.1 mM), not with high concentrations (2 mM) (276, 682, 785). Pigment production used to assess effect of histidine and tryptophan on purine nucleotide synthesis (786). Reduced interallelic fertility (264, 407). Complementation maps (268, 274). Relation of mutagens to complementation patterns (269). Mutants with nonpolarized complementation patterns on the right side of the complementation map grow on minimal medium if supplied with CO_2; other mutants do not respond to CO_2 (270). Used extensively for mutagenesis (see *ad-3A*). Rearrangement *T(I→III)Y112M4i ad-3B*, which has a breakpoint inseparable from *ad-3B*, was the first insertional translocation to be reported for fungi (266). Allele 7-017-0137 shows "fixed instability," mutating to an unstable prototrophic allele (41). Alleles 2-17-126, 12-21-28, and numerous others are supersuppressible (408, 749, 955). Called complementation group B.

***ad-4*: adenine-4**

IIIR. Right of *met-8* (1 to 4%) and *com* (0 to 5%). Left of *leu-1* (1 to 3%) (219, 815). (*482*)

Requires adenine. Cannot use hypoxanthine or inosine (661). Growth on adenine (at least for strains carrying allele 44206) is improved by the addition of histidine and still more by histidine plus methionine (661). Structural gene for adenylosuccinase, which controls two reactions in adenine synthesis (393, 1158). (See Fig. 8.) Accumulates a small amount of purple pigment when adenine is limiting (682). Used for the first demonstration of complementation between alleles in vivo (393) (simultaneous with independent demonstration in *am*) and in vitro (1157). Enzyme in revertants (1158). Used to study purine transport (787). Strains carrying alleles 44206 and 44415 are heat sensitive (34°C versus 25°C) (482, 682) and are osmotic remediable at 30°C (636); enzyme synthesized at 30°C by heat-sensitive strains has altered properties (636). Called complementation group F.

***ad-5*: adenine-5**

IL. Between *phe-1* and *arg-1* (1%) (816; H. B. Howe, Jr., personal communication). (*482*)

Uses adenine or hypoxanthine (682) (Fig. 8). Accumulates AICAR (81, 904) and SAICAR (81). Some mutants are stimulated by histidine and may not grow on hypoxanthine unless histidine is present; others may be inhibited by histidine (393; M. E. Case, personal communication). Produces some purple pigment, but less than *ad-3A* and *ad-3B* mutants (526). Called complementation group J. Evidence, apparently enzymatic, given in reference 120 suggests that some *ad-5* mutants lack both AICAR formyltransferase and inosine 5′-monophosphate cyclohydrolase, but apparently other *ad-5* mutants lack only the formyltransferase. Indirect evidence (902, 904) suggests that strains carrying *ad-5* allele Y112M192 are blocked at the formyltransferase step.

***ad-6*: adenine-6**

IVR. Right of *ilv-3* (9%) (579). Left of *pan-1* (1 to 2%), *chol-1* (1%), and *cot-1* (2 to 6%) (633). (*692*)

Uses adenine or hypoxanthine (682). Blocked in conversion of phosphoribosylformylglycineamide to phosphoribosylformylglycineamidine (120) (Fig. 8). Inhibited by caffeine in the presence of adenine (1172). Called complementation group I.

***ad-7*: adenine-7**

VR. Right of *cot-2* (4%). Left of *ro-4* (4%) and *pab-2* (8%). (158, 156). (*687*)

Uses adenine or hypoxanthine (682). Lacks phosphoribosylpyrophosphate amidotransfer-

ase, the first enzyme in de novo purine biosynthesis (525) (Fig. 8). Ascospores from homozygous *ad-7* × *ad-7* crosses are white (allele Y175M256). Strains carrying allele P73B171(t) are temperature sensitive.

***ad-8*: adenine-8**

VII. Right of *ser-6* (15%) and *het-8* (12%). Left of *aro-6* (8%) and *cpl-1* (6 to 11%) (437, *510*, 730, PB).

Requires adenine; cannot use hypoxanthine (526). Lacks adenylosuccinate synthase (511) (Fig. 8). Fine-structure mapping and intralocus complementation (510–512). Has little hypoxanthine uptake and little hypoxanthine phosphoribosyltransferase; both these effects are partly counteracted in *ad-1 ad-8* double mutants (903). Little hypoxanthine phosphoribosyltransferase is also found in *mep(3)* and *mep(10)* mutants, q.v. Used to study purine transport (787, 903, and references therein). Called complementation group E.

***ad-9*: adenine-9**

IR. Right of *met-6* (9 to 16%). Left of *Tp(T54M94)* and *nit-1* (3 to 15%) (466, 816). (*815*)

Uses adenine or hypoxanthine (526). Controls conversion of phosphoribosylglycineamide to phosphoribosylformylglycineamide (120) (Fig. 8). Called complementation group D.

***adg*: adenine-arginine**

See *arg-11*.

***adh*: adherent**

VIIL. Linked to *do* (0/53), *spco-4* (4%), and *nic-3* (11%) (*816*, PB).

Abnormal morphology. Conidia not powdery; do not shake loose. Complements *spco-4*. Morphologically distinct from *do* and *spco-4* mutants. (816, PB).

***ads*: adenine sensitive**

VI. Linked to *col-4* (*513*).

Growth completely inhibited at 35°C by 10 μM adenine; high concentrations inhibitory at 25°C. Poor growth on minimal medium at 35°C as compared with that at 25°C. Inhibition not relieved by vitamins, amino acids, guanine, guanosine, or guanylic acid; no growth response to guanosine in the absence of adenine (513; T. Ishikawa, personal communication).

***aga*: arginase**

VIIR. Between *wc-1* (2%) and *arg-10* (24 to 27%) (*240, 697*).

Presumed structural gene for arginase (240, 697) (see Fig. 10). Unable to form ornithine from arginine; arginine is thus unable to satisfy the proline requirement of *pro-3* in a *pro-3 aga* double mutant. Prototrophic single mutants develop polyamine requirement in the presence of arginine. This is due to feedback inhibition of ornithine biosynthesis by arginine, combined with a catabolic block in ornithine formation from arginine (240). Siderophore production is severely reduced in the absence of ornithine in the triple mutant *aga arg-5 ota*, which has been used to study iron transport (1146, 1147) and to obtain mutants defective in siderophore uptake (G. W. Charlang and N. P. Williams, personal communication); see *sit*.

***age-1*: aging of conidia-1**

IR. Symbol used for a series of many linked loci distal to *nit-1*, possibly redundant complexes. Individual loci symbolized as 1.3, 1.5, etc. Prototype *age* 1.5 is right of *so* (14%) and left of *aro-8* (7.6%); *age* 1.3 maps at same site as *so*, q.v. (*K. D. Munkres, personal communication*).

Reduced conidial longevity in the light. Not expressed in the dark, or in the light with vitamin E or reduced glutathione. Deficient in an isozyme of catalase, in mitochondrial superoxide dismutase, and in other enzymes involved in destroying free radicals and peroxides. Scored by plating efficiency after incubation of mature slant cultures at 30°C, 100% relative humidity, in continuous white fluorescent light, 24 J m^{-2}. Also scored by a defect in conidiophorogenesis on Vogel sorbose-sucrose plates. Initial mutants selected as spontaneous variants from f_1 of Oak Ridge wild types; variants with increased conidial longevity can also be selected. High spontaneous mutation rate. Longer life span correlated with slower growth. (702, 704, 705, 708; K. D. Munkres, personal communication)

***age-2*: aging of conidia-2**

VIR. Right of *ws-1* (8%). (*K. D. Munkres, personal communication*)

Phenotype similar to that of *age-1* (702, 704, 708).

***age-3*: aging of conidia-3**

IR. Right of *age-1* and *un-18*. (*705*, K. D. Munkres, personal communication)

Reduced conidial longevity. Differs from *age-1* and *age-2* mutants in having yellow conidia and normal genesis of conidiophores (705).

***al*: albino**

Mutants designated as albino impair carotenoid synthesis. These affect only vegetative cells (mycelia and conidia) and are without known effect on the perithecia or ascospores, where the pigment is melanin. The albino mutants vary in amount and color of carotenoids. Different alleles result in conidia and mycelia that are white, yellow, pink, purple, or white with traces of color or in white mycelia with pigment in the peripheral conidia. See, for example, reference 1042. Carotenoid synthesis is also affected by *ylo*, *wc*, and *age-3*, q.v., and by modifiers of intensity (982).

Albino mutants have been used to study the role of carotenoids in photoprotection (984, 1071, and references therein). Rapid development of carotenoids is induced by light; the action spectrum is described in references 250 and 1181, and mechanism of photocontrol is considered in reference 444. However, carotenoid synthesis can proceed slowly in complete darkness. Maximum carotenoid production results if incubation is at 6°C immediately after exposure to inducing light (442). Albino mutants can be scored in submerged colonies from plated ascospores by transfer of sorbose plates to 4°C under light after colonies have grown at 25°C in the dark (154, 500). An unstable constitutive variant has been described (587). Most *al* mutations map in a short region of IR where *al-1* and *al-2* were previously thought to be contiguous but are now known to be separated by other loci (797; D. D. Perkins, unpublished data).

***al-1*: albino-1**

IR. Right of *hom* (<1%), *arg-6* (<1 to 4%), *T(T54M94)*, and *al-2*. Left of *lys-3* (9%). (797, 808; D. D. Perkins, unpublished data). (*482*)

Carotenoids abnormal. Strains carrying the various alleles differ widely in phenotype, ranging from white (e.g., 4637) and "aurescent" (pigment in peripheral conidia and conidiophores, 34508) to yellow mycelia and conidia (e.g., ALS4 and RES-25). See, for example, reference 1042. Strains carrying alleles ALS-14, RES-6, 34508, and RES-25 contain large amounts of phytoene (99 to 100% of the total neutral carotenoids), suggesting a lesion that affects phytoene dehydrogenase (398, 1039) (see Fig. 9). Strains carrying allele RWT-ylo accumulate zeta carotene and smaller amounts of neurosporene, suggesting a leaky block of the step between these intermediates (1071). It is not known whether phytoene dehydrogenase catalyzes the whole series of dehydrogenations or whether leakiness of this enzyme accounts for the different mutant phenotypes. For complementation tests, see references 500, 1039, and 1041. Fine-structure mapping (500, 1042). Translocation *T(4637)*, inseparable from *al-1*, was the first albino mutation and one of the first chromosome rearrangements in *Neurospora* to be identified and studied (656). Allele 34508 called *aur*: aurescent.

***al-2*: albino-2**

IR. Right of *os-5* (<1%) and *T(STL76)*. Left of *arg-6* (1%) and *al-1* (797, 802, 808, 816, 818). Included in duplications from *Tp(T54M94)*, confirming location left of *arg-6* (808). (*482*)

Carotenoids absent or abnormal, but steroids produced (398). Blocked in microsomal fraction and defective in phytoene synthetase (445), a particulate enzyme (445 and references cited therein) (Fig. 9). Tracer experiments indicate a lesion between prephytoene pyrophosphate and phytoene (572). Alleles include those resulting in white and pale rose-white, e.g., 15300 and Y254M165 (1042), and purple, e.g., MN58a (154). For complementation, see references 500 and 1041. Fine-structure mapping (500, 1042) needs reevaluation because of new information on the location of the *arg-6* marker (797).

***al-3*: albino-3**

VR. Between *his-1* and *inl* (1%) (*1119*, PB).

Carotenoids deficient (398). Reported to lack geranylgeranyl pyrophosphate synthetase activity and is blocked in soluble fraction, consistent with lesion between isopentenyl pyrophosphate and geranylgeranyl pyrophosphate (445), but can still produce farnesyl pyrophosphate (445) and steroids (398). (See Fig. 9.) This evidence contradicts in vivo labeling results that indicate a lesion between prephytoene pyrophosphate and phytoene (572). Strains carrying allele Y234M470 (*al-3^{ros}*), formerly called rosy (49), become partially pigmented but are readily distinguished from the wild type. *ylo-1* can be scored in combination with *al-3^{ros}* (Y234M470) (PB). Strains carrying other alleles (e.g., RP100) (1119) are white with a trace of pink pigment.

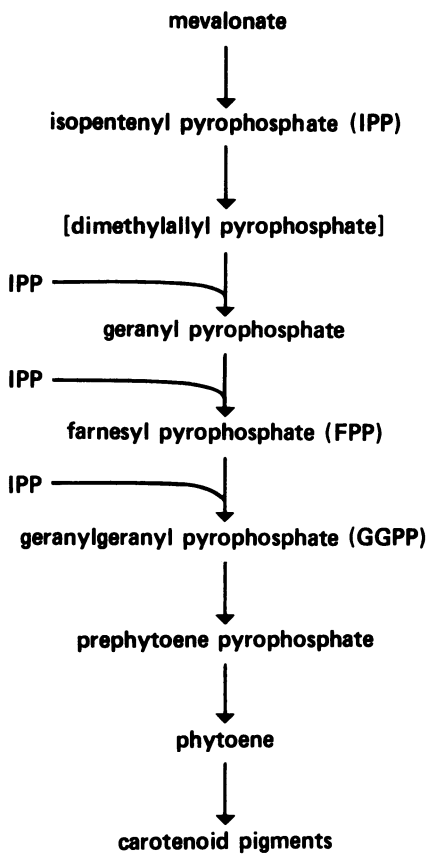


FIG. 9. Biosynthetic pathway for carotenoids. It is thought that the same prenyl transferase catalyzes all the steps from dimethylallyl pyrophosphate to geranylgeranyl pyrophosphate (444; R. W. Harding, personal communication), and it has been proposed that a separate prenyl transferase converts dimethylallyl pyrophosphate to farnesyl pyrophosphate for sterol synthesis (445). The conversion of phytoene to the various carotenoid pigments involves a series of dehydrogenations, cyclizations, and other reactions. There must also be a *cis/trans* isomerization analogous to that found in tomato (842). The sequence of some of these steps is still uncertain; the pathway must branch, and there may be alternate routes to some of the products. See references 228, 443, 444, 842 and citations therein for proposed sequences. *al-1* is probably blocked in phytoene dehydrogenase (398). It is not known whether this enzyme catalyzes the whole series of dehydrogenations. *al-2* is reported blocked between geranylgeranyl pyrophosphate and phytoene (445) and between prephytoene pyrophosphate and phytoene (572). *al-3* is alternately reported blocked between isopentenyl pyrophosphate and geranylgeranyl pyrophosphate (445) and between prephytoene pyrophosphate and phytoene (572), but it is not blocked in the production of farnesyl pyrophosphate or sterols (398, 445). *ylo-1* is evidently blocked in a late step, probably either in the conversion of lycopene to 3,4-dehydrolycopene or in the conversion of either torulene or γ-carotene to neurosporaxanthin (see citations in reference 398).

***alc-1*: allantoicase-1**

II. Linked to *pe* (10%), probably on the opposite side of *pe* from *xdh-1* (24%) (*872*).

Defective in purine catabolism. Unable to use allantoic acid as the sole nitrogen source. Lacks only allantoicase (872) (see Fig. 24).

alcoy

Genotype: *T(IR;IIR)4637 al-1; T(IVR;VR) R2355, cot-1; T(IIIR;VI)1, ylo-1* (816).

Linkage tester containing three unlinked reciprocal translocations, each tagged with a visibly scorable marker, marking linkage groups I through VI. Linkage of a gene to *al-1*, *cot-1*, or *ylo-1* in a cross to *alcoy* allows assignment to a linkage group by a single follow-up cross. A majority of new point mutations are linked to one of the *alcoy* markers (816). An improved version, *alcoy; csp-2*, carries the VII marker *csp-2* in addition to the three original markers (811). *alcoy* has been used cytologically to study the synaptonemal complex and recombination nodules (396).

***aln-1*: allantoinase-1**

VII. (*872*)

Defective in purine catabolism. Unable to use allantoin or any purine intermediate before it as the sole nitrogen source. Lacks allantoinase (872) (see Fig. 24).

alx-1

See ANT-1.

***alx-2*: alternate oxidase-2**

Unmapped.

Lacks inducible cyanide-insensitive respiration. Cannot grow on antimycin A. Complements alx-1 (ANT-1) (308).

***am*: amination deficient**

VR. Right of *ure-2* (2%) and *sp* (4 to 8%). Left of *gul-1* (≪1%) and *ace-5* (<1%) (122, 570, 579, 998). (*R. W. Barratt, cited in reference 1036*)

Structural gene for nicotinamide adenine dinucleotide phosphate (NADP)-glutamate dehydrogenase (336) (see Fig. 19), for which a com-

plete 452-residue amino acid sequence has been obtained (465). Requires a source of α-amino nitrogen for growth, alanine being a good supplement (e.g., reference 997). Readily scorable at 25°C; leaky at 34°C (42). Leaky growth and adaptation on minimal medium are prevented by 0.02 M glycine (782, 783) or by *en(am)-1, en(am)-2*, or *nit-2*, q.v. The *am* mutants show abnormal regulation of reduced nicotinamide adenine dinucleotide (NADH)-glutamate dehydrogenase and are synergistic with *nit-2* in this effect (226). Some *am* alleles (e.g., RU1) suppress the pyrimidine requirement caused by *pyr-3* (CPS^- ACT^+) mutations (1137). Used for the first demonstration of complementation between alleles in vivo (344) (simultaneous with independent demonstration with *ad-4*). In vitro complementation (342). Used for studies of complementation mechanism (199, 200, 1120). Used for fine-structure mapping (337, 338). Control of intralocus recombination by *rec-3* (996–998). Used to study colinearity of the gene and gene product, internal suppressors (105, 340, 465), and the action of supersuppressors (954, 955). The functional defects in several mutant enzymes with single amino acid replacements have been defined: am^1 mutant enzymes fail to bind NADPH (1120); am^2, am^3, am^{19}, am^{130}, and am^{131} enzymes are stabilized in the inactive conformational form (30, 200, 336, 556, 1044), and all are complementable by am^1; am^{14} is osmotically reparable and is thought to have unstable quaternary structure (340). Used in a study showing glutamine to have a role as corepressor of uricase synthesis (1118). Used to study nitrogen assimilation and metabolism (503) and nitrogen metabolite repression (186, 291). Efficient procedure for selecting new *am* mutants (551). Spectrum of ultraviolet irradiation (UV)- and nitrous acid-induced mutants (554). Allele am^{17} has a chain-terminating codon of either the amber or ochre type at residue 313 of glutamate dehydrogenase, based on amino acid replacements in revertants and by *ssu-1* (956). Allele 6 is a frameshift mutation with an insertion in the Ser5 codon (985). Allele 126 is highly unstable (553). Allele 132 is a deletion (1162). The am^+ gene has been cloned in *Escherichia coli* (J. R. S. Fincham, personal communication) and transformed back into *Neurospora* (J. A. Kinsey, personal communication).

***amr*: ammonium regulation**

See *nit-2*.

***amy-1*: amylase**

See *sor(T9)*.

***amy(SF26)*: amylase**

See *exo-1*.

***amyc*: amycelial**

IL. Between *ad-5* and the centromere (H. B. Howe, Jr., personal communication). (*K. C. Atwood, cited in reference 789*)

Conditional morphological mutant. Recessive. On sucrose media, it forms dotlike granular colonies of irregular budding vesicular elements. On permissive media, made either with acetate (plus α-ketoglutarate, succinate, malate, or certain amino acids) or with amino acids as carbon and nitrogen sources, it forms hyphae and macroconidia but is apparently still colonial (281; for a review, see reference 1088). Adenosine 3',5'-monophosphate induces conidiation even on sucrose (281). Photographs (774, 1088). Low oxygen consumption and depressed amino acid pools (281). Abnormal mitochondria (773). Surface glycopeptide (281). Wall composition (207). Recovery of the antigenic arc representing the isozyme of malate dehydrogenase associated with conidiation (784). Ultrastructure (773, 1088, 1089 and references cited therein). Used extensively in balanced heterokaryons to detect lethal recessive mutations (e.g., reference 32) and to evaluate nuclear distribution (33); techniques described (34).

***an*: anaerobic**

Segregates 1:1. Not mapped.

Reported to be facultatively anaerobic, growing weakly anaerobically on enriched medium. Prototrophic and indistinguishable from the wild type under aerobic conditions. Not glucose repressed. The anaerobic culture is aconidial, with reduced cytochrome oxidase and malate dehydrogenase activities and mitochondrial changes. Ethanol is produced. Obtained by filtration enrichment with recycling. (492, 493) The symbol An^+ has been used in reference 493 to specify the mutant phenotype, and An^- has been used to specify the wild phenotype.

ANT-1: (antimycin-sensitive strain)

Symbol used to designate double-mutant strain *azs; has* (311). Not a locus designation. (Also called alx-1.) See *azs* and *has*.

***aod-1*: alternate oxidase deficient-1**

IV. Left of *trp-4* (23%) (*1051*).

The alternate oxidase system is not induced when cytochrome-mediated, cyanide-sensitive respiration is inhibited. In four *aod-1* mutants, a mitochondrial peptide of M_r = 35,000 is induced as in the wild type; in a fifth mutant, it is absent. Recessive. Strains carrying *aod-1* plus either [*mi-1*] or [*mi-3*] are viable. (82, 1051) Called *aod-B* (82). Three alleles originally called *aod-1*, *aod-2*, and *aod-3* are now called *aod-1-1*, *aod-1-2*, and *aod-1-3*, respectively (1051).

aod-2: alternate oxidase deficient-2

II. Linked to *arg-5* (7%), *thr-3* (16%), and *trp-3* (36%) (82, *1051*).

The alternate oxidase system is not induced when cytochrome-mediated, cyanide-sensitive respiration is inhibited. A mitochondrial polypeptide (M_r, 35,000) is not induced as in the wild type. Recessive. Strains carrying *aod-2* plus either [*mi-1*] or [*mi-3*] are viable (82, 1051). Called *aod-A* (82). The allele originally called *aod-4* is now called *aod-2-1* (1051).

apu: accumulation of purines

Not linked to *ad-7* (VR), *mt*, or *aza-1* (IL).

Excretes purines. Obtained among prototrophic revertants of the mutant *ad-7* (*ad-7* blocks the first step in de novo purine synthesis). Secretion assayed by cross-feeding on plates seeded with *ad-3A* conidia. Purine secretion by the mutant *apu* occurs later, and the colony size is larger than with strains carrying *aza-1* allele 67-12 (4 days versus 36 h; 25°C). (525).

arg: arginine

For details of the arginine biosynthetic pathway, see Fig. 10. The most comprehensive reference on biosynthetic pathway mutants is 238. Arginine mutants have been used extensively for studies of compartmentation (see references 223, 233, 242, and 245) and for studies of control of flux through the arginine pathway (356 and references therein; also see the *ota* entry). Crossing is inhibited by high arginine levels; 0.1 or 0.2 mg of arginine per ml of crossing medium is satisfactory. Lysine and arginine show competitive inhibition, and all arginine auxotrophs are inhibited by lysine. Lysine resistance is conferred on *arg-1* mutants by probable transport mutation lys^R, q.v. Crosses of strains involving both requirements can usually be handled by adjusting the ratio. Medium containing 0.8 mg of L-arginine hydrochloride and 1.6 mg of L-lysine hydrochloride per ml is recommended for crosses (P. St. Lawrence, personal communication). Leaky arginine mutants (e.g., *arg-2*, *arg-3*, *arg-13*) are less leaky on nitrate medium (238) or canavanine plus lysine (876). Leakiness of germinating ascospores of *arg-1* and *arg-3* strains is prevented by 0.05 mg of lysine per ml with no canavanine (D. Newmeyer, unpublished data). Some *arg* genes were originally called *cit* or *orn*. For degradative or related steps in arginine metabolism, see *aga*, *ota*, *spe*, and *ure*.

Arginine biosynthesis and catabolism are controlled in a major way by compartmentation (reviewed in references 237 and 245). Acetyl glutamate kinase and acetyl glutamate synthase are feedback regulated by arginine (1148; C. P. Chang and R. L. Weiss, personal communication). With one exception, the enzymes of arginine biosynthesis are not repressed below levels that occur in minimal medium when arginine is added to cultures. The exception is carbamyl-phosphate synthetase A, the small subunit of which is repressed fivefold. When cultures are limited for arginine, all but one biosynthetic enzyme increase concomitantly by about three- to fivefold; the carbamyl-phosphate synthetase A small subunit increases as much as 20-fold (223, 242). These "derepressions" can also be brought about by starvation for other amino acids such as tryptophan, lysine, and histidine (137, 1131; reviewed in reference 642); they require the normal product of the *cpc-1* locus (61). See *cpc-1*. The catabolic enzymes arginase and ornithine aminotransferase are present without induction and are elevated only two- to fivefold in response to nitrogen limitation or the addition of arginine (but not ornithine) to the medium. These enzymes are not affected by mutations at the *nit-2* or *cpc-1* locus.

arg-1: arginine-1

IL. Between *ad-5* (1%) and *eth-1* (<1%) (816). (*751*).

Uses arginine but not precursors (1010). Lacks argininosuccinate synthetase (752) (Fig. 10). Interallelic crosses produce perithecia, but most ascospores are white and inviable (751). Leaky *arg-1* mutants are frequent among those selected as citrulline-resistant variants of $arg\text{-}12^s$; *pyr-3*. Most of these show interallelic complementation, and many are transport deficient (1075). *arg-1* mutants do not grow well on some complex complete media unless extra arginine is added.

arg-2: arginine-2

IVR. Right of *col-4* (<1 to 2%). Between *T(S1229)* breakpoints, but not between *T(S4342)*

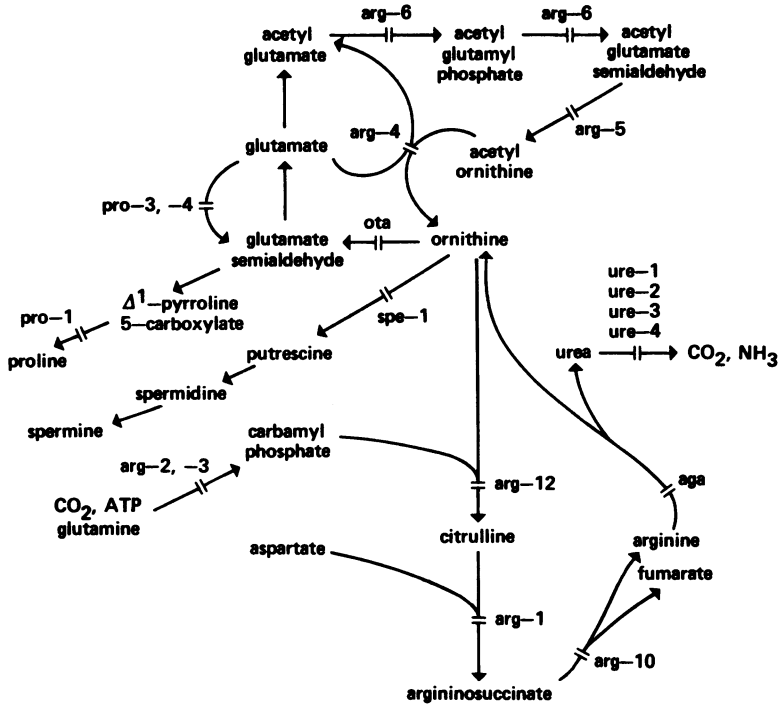


FIG. 10. Biosynthetic pathways for arginine, proline, polyamines, and associated intermediary metabolism, showing sites of gene action in biosynthesis and arginine catabolism (238, 241, 452, 569, 657, 696, 697, 1009, 1105, 1177, and references therein). Carbamyl phosphate for pyrimidine synthesis is made as a separate pool by a distinct enzyme (see *pyr-3*, Fig. 20). Interchange between the two pools occurs only in certain mutant combinations. ATP, Adenosine triphosphate.

breakpoints; hence, left of *arg-14* and *pyr-3* (1 to 3%). (101, *692, 695*, 808, 876, 991).

Uses citrulline or arginine (1010). Specifies the small component of arginine-specific carbamyl-phosphate synthetase A, a two-component enzyme (242). This component enables the enzyme to use glutamine as a nitrogen donor (233, 243). (The large component is specified by *arg-3*.) (See Fig. 10.) For regulation and compartmentation, see reference 242. The arginine/citrulline requirement can be suppressed by *pyr-3* (CPS^+ ACT^-) mutations (236, 876); see *pyr-3*. Leakiness is prevented by canavanine; lysine overcomes the side effects of canavanine (876). Strains with some (all?) of the alleles can grow on minimal medium in 30% CO_2 (108). Leakiness is decreased if CO_2 is removed or if uridine is added (880). Translocation *T(IL;IVR)MEP24* is inseparable from *arg-2* (R. H. Davis, personal communication).

arg-3: arginine-3

IL. Between *eth-1* (1%) and *csp-1* (1%) (816, 972). Between the *T(39311)* breakpoints; hence, left of the centromere and of *sn* (1 to 6%) (174, 798). (*1005*).

Uses citrulline or arginine (1010). Structural gene for the large component of arginine-specific carbamyl-phosphate synthetase A, a two-component enzyme (242, 243). This component can form carbamyl phosphate in vitro, using ammonia as the nitrogen donor (231, 243). (The small component is specified by *arg-2*.) (See Fig. 10.) For regulation and compartmentation, see reference 242. The arginine/citrulline requirement can be suppressed by *pyr-3* (CPS^+ ACT^-) (236, 658); see *pyr-3*. Strains carrying allele 30300 can grow on minimal media in 40% CO_2 (108, 191). Translocation *MEP35* is inseparable from *arg-3* (R. H. Davis, personal communication; PB) Called *cit-1*.

arg-4: arginine-4

VR. Between *sp* (1 to 11%) and *inl* (2 to 4%) (812, 976). (*D. G. Catcheside, cited in reference 812*).

Uses ornithine, citrulline, or arginine (1010). Lacks acetylornithine-glutamate transacetylase (acetylornithine acetyltransferase) (249, 1106) (Fig. 10). Weakly suppresses CPS^- ATC^+ *pyr-3*

mutants (see reference 660). Alleles 21502 and 34105 (later called *arg-4* and *arg-7*) were originally thought to be genetically distinct because they complemented each other (1005), but an intercross produced no recombinants (R. W. Barratt, cited in reference 660). Both lack the same enzyme (249, 1106).

***arg-5*: arginine-5**

IIR. Right of *T(ALS176)*; hence, right of the centromere. Left of *aro-3* (428, 808; D. G. Catcheside, cited in reference 812). (*789*). Listed incorrectly in I by Houlahan et al. (482) because linkage of *arg-5* to albino in *T(I;II)4637 al-1* had been shown previously (1005).

Uses ornithine, citrulline, or arginine (1010). Structural gene for acetylornithine transaminase (696) (Fig. 10). Sideramine production is completely blocked in the triple mutant *arg-5; ota; aga* when ornithine is absent. Used to study iron transport (1146, 1147). Called *orn-1*.

***arg-6*: arginine-6**

IR. Right of *T(T54M94)* and *al-2* (1 to 2%). Left of *hom* (1%), *al-1* (<1 to 4%), and *T(4637)* (797, 808). (*1005*).

Uses ornithine, citrulline, or arginine (1010). Probably bifunctional, specifying arginine-sensitive acetylglutamate kinase (238) and *N*-acetylglutamyl-phosphate reductase (J. Cybis and R. H. Davis, cited in reference 238). Probably the structural gene for both enzymes (R. H. Davis, E. Wolf, and R. L. Weiss, personal communication) (Fig. 10). L-Methionine may inhibit (D. D. Perkins, unpublished data). Possible allele: *su(pro-3)* (1129). Called *orn-2*.

***arg-7*: arginine-7**

Same as *arg-4*, q.v. Called *orn-3*.

***arg-8*: arginine-8**

See *pro-3*.

***arg-9*: arginine-9**

See *pro-4*.

***arg-10*: arginine-10**

VIIR. Between *arg-11* (1 to 2%) and *nt* (1 to 12%) (*751*, 789).

Uses arginine but not precursors (751). Lacks argininosuccinate lyase (341) (Fig. 10). Accumulates argininosuccinate on limiting arginine (341). Viable ascospores from interallelic crosses are rare, but the viable ones are often arg^+, whereas most arg^- ascospores remain colorless and inviable (751). All *arg-10* mutants tested showed spasmodic growth in growth tubes at low arginine concentrations (O. J. Gillie, personal communication). *arg-10* mutants do not grow well on some complete media unless extra arginine is added.

***arg-11*: arginine-11**

VIIR. Left of *arg-10* (1 to 2%) (*789*).

Requires arginine or citrulline, plus low levels of a purine and a pyrimidine (290, 754, 1006). Inhibited by guanidine, sarcosine, and serocyamine (1005). Complements *arg-10* fully in heterokaryons (754). The relation of this mutation to arginine biosynthesis or metabolism is obscure. Growth requirements vary markedly with CO_2 concentration and inoculum size. At 0% CO_2 or with small inocula, the requirement for all three supplements is absolute; with increasing CO_2 concentration or inoculum size, pyrimidine and then purine can be omitted; at 30% CO_2, all three supplements can be omitted. (108, 192, 754) Growth rate and morphology are highly variable among progeny from *arg-11* × wild type crosses (754). Grows spasmodically in growth tubes (O. J. Gillie, personal communication). Allele 44601 formerly called *un* and *adg* (290, 482).

***arg-12*: arginine-12**

IIR. Right of *pe* (1 to 5%). Left of the *T(NM177)* right breakpoint and of *aro-1* (<1%) (389, 808, 1052). (*1160*).

Uses citrulline or arginine. Structural gene for ornithine carbamyl transferase (230, 244, 1160) (Fig. 10). Leaky allele $arg\text{-}12^s$ was discovered as a suppressor of a *pyr-3* mutant and initially called *s* (483). It reduces ornithine carbamyl transferase activity over 98% without imposing any arginine requirement. $arg\text{-}12^s$ suppresses the pyrimidine requirement of *pyr-3* strains that lack only pyrimidine-specific carbamyl-phosphate synthetase. This is because $arg\text{-}12^s$ strains accumulate arginine-specific carbamyl phosphate, which can then be used for pyrimidine synthesis (236); see *pyr-3*. Nonleaky *arg-12* alleles cannot cause such suppression because the exogenous arginine that is required for growth results in repression of the arginine-specific car-

bamyl-phosphate synthetase (236). Mutations at all other arginine biosynthesis loci can be obtained efficiently as tight double mutations, using *arg-12*^{s} as starting material (238). Double mutants *pro-4; arg-12*^{s} and *pro-3; arg-12*^{s} are prototrophic. The double mutant *arg-5 arg-12*^{s} cannot use exogenous ornithine (see references 234, 241).

***arg-13*: arginine-13**

IR. Between *os-1* (1%) and *so* (2 to 12%) (816). (*660*)

Responds well to arginine or citrulline and poorly to ornithine (238, 660). Acts as a suppressor of the pyrimidine requirement of CPS$^-$ACT$^+$ mutations of *pyr-3* (660). Leaky on minimal medium; scoring cleared by addition of lysine. Interallelic crosses are sterile. Formerly called *arg(RU3)*.

***arg-14*: arginine-14**

IVR. Right of the *T(S4342)* left breakpoint and of *arg-2* (1%). Left of *T(NM152)* and *pyr-3* (1%) (*238*).

Uses arginine, citrulline, or ornithine. Point mutants selected as tight double mutants by using *arg-12*^{s} (238). Allele S1229 is inseparable from translocation *T(S1229)*. (54, 55, 808)

arg(CD-15), arg(CD-55)

See *cpc-1*.

arg(RU1)

Allelic with *am*, q.v.

***arg*^{R}: arginine resistant.**

IVR. Right of *pyr-2* (14%) (*566*). Probably allelic with *pmb* (565, 566).

Growth of the double mutant *lys-1; arg*^{R} is resistant to the normal inhibition by L-arginine (566).

***aro*: aromatic**

Used for genes concerned with biosynthesis of aromatic amino acids and *p*-aminobenzoic acid. All *aro* strains except *aro-6*, *aro-7*, and *aro-8* are auxotrophs requiring a mixture of *p*-aminobenzoic acid, tyrosine, tryptophan, and phenylalanine. The first step in the pathway is catalyzed by three isozymes subject to feedback inhibition by different end products of the branched pathway. These isozymes are specified by different widely separated genes (*aro-6*, *aro-7*, *aro-8*). The second, third, fourth, fifth, and sixth steps are specified by a cluster gene that produces a single transcript (for reviews, see references 387 and 1130). The final step before branching is specified by a unifunctional gene (*aro-3*) which is separate from the *aro* cluster gene, although linked to it at a distance. See Fig. 11 for the pathway and sites of gene action. The third and fourth steps are paralleled by similar reactions in the quinate catabolic pathway (see Fig. 21). Thus, the *aro-9* enzyme can be replaced by the *qa-2* enzyme and, under appropriate conditions, the *aro-1* enzyme can be replaced by the *qa-3* enzyme. Supplement levels: 40 to 80 mg each of tyrosine, tryptophan, and phenylalanine per liter and 0.25 mg of *p*-aminobenzoic acid per liter (178, 428). Also called *arom*.

***aro-1, -9, -5, -4, -2*: aromatic cluster gene**

IIR. Right of *T(NM177)* and *arg-12* (<1%). Left of *ff-1* (4 to 6%) (808, 1052; A. Kruszewska, personal communication). (*47*) For intracluster map see references 387 and 885.

Structural gene for the aromatic biosynthetic pathway leading to tryptophan, tyrosine, phenylalanine, and *p*-aminobenzoic acid (Fig. 11). Multifunctional cluster gene (370) specifying five enzymes (370, 389, 1130). Clustering of functions discovered by Gross and Fein (428). For reviews, see references 387 and 665. The order of regions that specify the five functions is still conveniently represented by the symbols established when it was thought that five separate genes were involved:

(arg-12) aro-1 aro-9 aro-5 aro-4 aro-2 (ace-1)

aro-1 specifies dehydroshikimate reductase; *aro-2*, dehydroquinate synthetase; *aro-4*, 3-enolpyruvyl shikimic acid-5-phosphate synthetase; *aro-5*, shikimate kinase; and *aro-9*, biosynthetic dehydroquinase. These symbols actually represent five domains of the pentafunctional polypeptide, which may be separated in purification, owing to proteolysis. The native enzyme is a dimer of the pentafunctional chains (370, 627). In some contexts, it may be preferable to designate the entire cluster gene as a single locus, *aro*. Mutations exist that block individual steps; in addition, there are polar mutations that eliminate more than one function. There are some

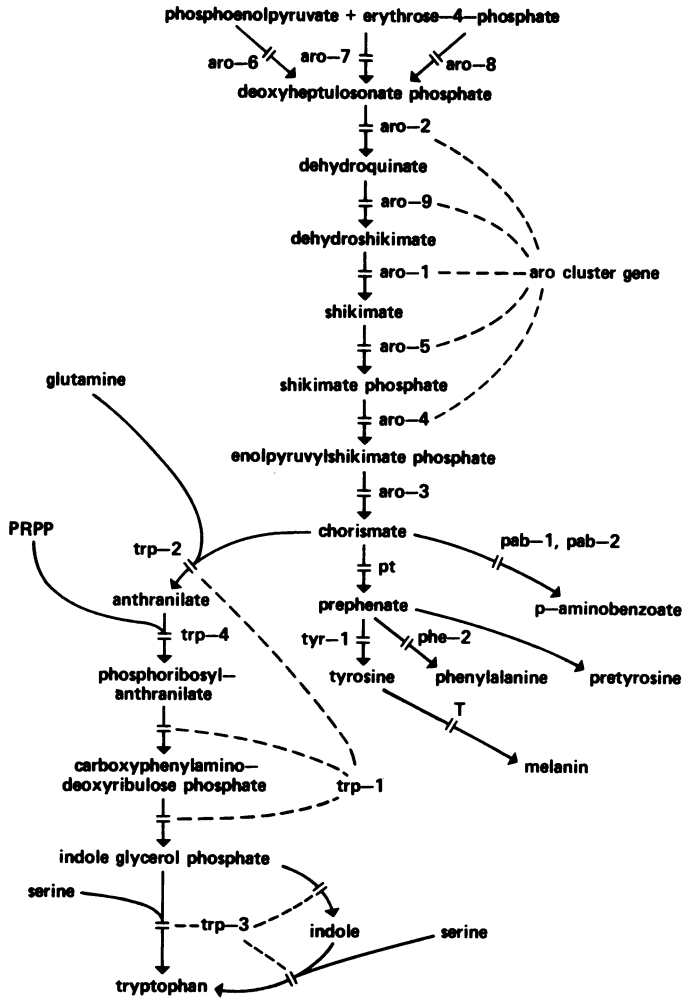


FIG. 11. Biosynthetic pathways of the aromatic amino acids, showing sites of action to the *aro*, *trp*, *pt*, *phe*, *tyr*, and *T* genes (98, 147, 201, 259, 316, 387, 437, 473, 519, 546, 1126, 1167). The conversion of chorismate to *p*-aminobenzoate has not been demonstrated in *Neurospora*. In the conversion of tyrosine to melanin, the later steps are nonenzymatic. The gene products of *trp-1* and *trp-2* form an enzyme aggregate with three properties; a given *trp-1* mutation may block one or more of the reactions. *aro-9*$^+$ activity (biosynthetic dehydroquinase) can be replaced by the product of *qa-2*$^+$, the equivalent gene in the catabolic pathway (885). Pretyrosine accumulates when other pathways are blocked (see *phe-2*). PRPP, 5-Phosphoribosyl pyrophosphate.

discrepancies between genetic mapping and mapping by polarity (reviewed in reference 665). However, various kinds of evidence agree that transcription begins at the *aro-2* end (145, 387). *aro-1*, *aro-2*, and *aro-9* mutants can use shikimate (0.3 mg/ml) as an alternative to the mixture of four aromatic amino acids (shown for *aro-1* by Tatum [1055]). Mutations that block different individual steps complement with each other (389). Complementation between alleles that block the same single step has been detected only for *aro-2* and *aro-1* (139, 389). Polar mutants are divided into six classes (A through F) based chiefly on their complementation behavior (389); types D, E, and F are semicolonial and have yellowish-orange conidia (144). Single-function *aro-9* mutants were obtained by selecting in a strain of genotype *qa-1*, which is noninducible for catabolic dehydroquinase activity (885). Translocation *T(II;III)C161 aro* (called *arom-2*) is inseparable from the *aro-1* cluster, and *T(II;III)C161* strains lack several activities (428). *aro(p)* indicates polar mutations in the *aro* cluster. Noncomplementing alleles M26, M1039, M1065, M1108, M1162, M1172, and Y306M54 (abbreviated M54) are suppressible by nonsense suppressors ("supersuppressors") (144, 145, 957).

***aro-1*: aromatic-1**

Part of the *aro* cluster gene in IIR. See *aro* cluster gene.

Specifies dehydroshikimate reductase (Fig. 11) (389, 428). Accumulates dehydroshikimate, which induces dehydroshikimate dehydrase in the catabolic pathway (428). Suppressed by the mutant *qa-4*, which lacks dehydroshikimate dehydrase; this allows induction of the catabolic enzyme quinate (shikimate) dehydrogenase, which substitutes for the biosynthetic enzyme dehydroshikimate reductase (147). A lag in growth of the mutant *aro-1* on shikimate occurs with sucrose or glucose as the carbon source. This is overcome by substituting 1% glutamate for the sugar (505).

***aro-2*: aromatic-2**

Part of the *aro* cluster gene in IIR. See *aro* cluster gene.

Specifies dehydroquinate synthetase (389) (Fig. 11). *aro-2* point mutants should not be confused with strain C161 (*arom-2* in reference 428), which lacks several activities specified by the *aro* cluster, including the *aro-2* function (428); the C161 mutation is inseparable from translocation *T(IIR;III)C161* (808).

***aro-3*: aromatic-3**

IIR. Right of *arg-5* (1 to 3%). Left of *T(NM177)* and of *nuc-2* (*428*, 671; L. Garnjobst, personal communication). Not closely linked to the *aro* cluster gene.

Specifies chorismate synthetase (369, 389) (Fig. 11). Requires a mixture of *p*-aminobenzoic acid, tyrosine, tryptophan, and phenylalanine for growth. Shows interallelic complementation (389). Leaky, giving hazy growth on minimal medium at 4 days, 34°C; tests should be scored promptly (D. D. Perkins, unpublished data).

***aro-4*: aromatic-4**

Part of the *aro* cluster gene in IIR. See *aro* cluster gene.

Specifies 3-enolpyruvate shikimic acid-5-phosphate synthetase (389) (Fig. 11).

***aro-5*: aromatic-5**

Part of the *aro* cluster gene in IIR. See *aro* cluster gene.

Specifies shikimate kinase (389) (Fig. 11).

***aro-6*: aromatic-6**

VIL. Between *ad-8* (8%) and *lys-5* (3%) (*437*).

Grows on minimal medium except when both tryptophan and phenylalanine are present to inhibit the alternate synthases (437). Structural gene for 3-deoxy-D-arabinoheptulosonic acid-7-phosphate synthase (Tyr), one of the three isozymes inhibitable by tyrosine, phenylalanine, and tryptophan, respectively (Fig. 11). Both activity-negative and allosteric inhibition-negative alleles have been found (436).

***aro-7*: aromatic-7**

I. Between *arg-1* (4%) and *his-3* (1 to 2%) (*437*).

Grows on minimal medium except when both tyrosine and tryptophan are present (437). Structural gene for 3-deoxy-D-arabinoheptulosonic acid-7-phosphate synthase (Phe), one of the three isozymes inhibitable by tyrosine, phenylalanine, and tryptophan, respectively (Fig. 11). Both activity-negative and allosteric inhibition-negative alleles have been found (436).

***aro-8*: aromatic-8**

IR. Between *so* (7 to 11%) and *R* (4%) (*437*, 1093).

Grows on minimal medium except when both phenylalanine and tyrosine are present (437). Probably the structural gene for 3-deoxy-D-arabinoheptulosonic acid-7-phosphate synthase (Trp), one of three isozymes inhibitable by tyrosine, phenylalanine, and tryptophan, respectively (Fig. 11). Both activity-negative and allosteric inhibition-negative alleles have been found (436).

***aro-9*: aromatic-9**

Part of the *aro* cluster gene in IIR. See *aro* cluster gene.

Specifies biosynthetic dehydroquinase (885) (Fig. 11). Requires shikimic acid or a mixture of four aromatic acid products when a *qa* mutation is present that eliminates catabolic dehydroquinase. The single mutant *aro-9; qa*$^+$ grows on minimal medium without supplement. Single-function *aro-9* mutants were first obtained by selecting for *aro* auxotrophs in a strain carrying

qa-1, a regulatory mutant which lacks catabolic dehydroquinase activity (885) (Fig. 11). Conversely, *aro-9* is used to select *qa-1* and *qa-2* mutants (883, 885).

aro(p)

Symbol used for polar mutations that affect several enzymes of the *aro* cluster gene. See *aro-1*, *-9*, *-5*, *-4*, and *-2*.

***arom*: aromatic**

Changed to *aro*.

***ars*: aryl sulfatase**

VII. Right of *thi-3* (2 to 5%). Left of *met-7* (<1%) and *ile-1* (1%) (*666*, 725).

Aryl sulfatase structural gene (667). Scored by color reaction with *p*-nitrophenyl sulfate. Because the enzyme is repressed in the wild type by traces of inorganic sulfate and other compounds present in all normal agars, screening on plates is carried out with an *eth-1*^{R} *cys-11* background, in which *ars*$^+$ colonies have detectable, derepressed activity (666). Scoring of crosses does not require this special background if the germinated spores are grown with cysteic acid (1 mM) as the sole sulfur source ($MgCl_2$ replacing $MgSO_4$). Scoring method (725). Reversion (699). Mutants lacking aryl sulfatase were first isolated in *N. crassa* (666), and the gene was later shown to be allelic with a "natural" aryl sulfataseless gene introgressed into *N. crassa* from one isolate of *N. tetrasperma* and with a gene which codes for an electrophoretic variant enzyme in another natural isolate of *N. tetrasperma* (667). Regulation reviewed (642).

***asc*: ascus development**

Symbol used by (253, 254) for recessive mutations affecting ascus (or ascospore) development. Numerous mutations have been given this symbol. Only those mapped are listed here. Five remaining unmapped recessive mutations complement each other and *mei-1*. Some are barren; others result in much ascospore abortion. See also *mei*.

asc(DL95)

IVR. Possibly allelic with *mei-1*, q.v.

asc(DL243)

IVR. Possibly allelic with *mei-1*, q.v.

***asc(DL879)*: ascus development**

II. Linked to *arg-5* (3%) (*253*).

Impaired meiosis in homozygous crosses. Recessive. Seventy percent of the ascospores abort and the total ascospore number is reduced, as are pachytene pairing and recombination (254). Viable ascospores are usually disomic for one or more linkage groups. Not tested for allelism with *mus-7*, which maps in the same region. Crosses homozygous for *mus-7* are barren.

***asco*: ascospore maturation**

An allele of *lys-5*, q.v. Photograph (1012).

Ascospore color mutants, autonomous

See *asco*, *bs*, *per*, *ts*, *ws*, *cys-3*, *lys-5*, and *pan-2*. For examples of applications, see references 314, 529, 586, 737, 822, 1013. *cys-3* may be best for demonstrating patterns in asci (858; D. D. Perkins, unpublished data).

***asn*: asparagine**

VR. Right of *inv* (4 to 9%). Left of *gran*, *pl* (1 to 9%), and *pyr-6* (6%) (156, 158, 698, 1036). (*1054*)

Requires asparagine for growth; no response to aspartic acid (1054). Lacks asparagine synthetase. Complementation between alleles (K. G. MacPhee, R. E. Nelson, and S. M. Schuster, personal communication via 1980 Neurospora Information Conference). May be inhibited by histidine (D. D. Perkins, unpublished data). Symbol changed from *asp* (807).

***asp*: aspartate**

V. Between *at* (0 to 3%) and *per-1* (16 to 26%) (819, PB). (*812*)

Growth aided by aspartate or glutamate. Some response also to homoserine or leucine (290). Grows adaptively on minimal medium; adapts more rapidly at 25°C than at 36°C. Inhibited by alanine; 0.5 mg of alanine per ml of test medium aids scoring. Symbol changed from *aspt* (807). The symbol *asp* was originally used for asparagine. In 1973, new symbols were adopted to conform to bacterial usage for amino acid auxotrophs (807), with *asn* used for asparagine and *asp* used for aspartate.

***aspt*: aspartate**

Changed to *asp*.

***at*: attenuated**

V. Right of *cyt-9* (5%) and *lys-1* (2 to 10%). Left of *asp* (0 to 3%) (*819*, PB).

Conidia formed in small flecks or granular clumps on the agar surface, especially in the crescent at top of the slant (819). Growth and pigmentation slower than those of the wild type, but cover slant. Good marker. Most easily scored on minimal medium, on which conidiation is less profuse than on complete medium. Called *morph(M111)* in reference 819.

***atr-1*: aminotriazole resistant**

IL. Right of *In(H4250)* and of *suc* (0/39). Left of the *T(39311)* right breakpoint (PB). (*818*)

Resistant to 0.5 mg of 3-amino-1,2,4-triazole per ml of solid medium (added before autoclaving). Not resistant to acriflavine. Abnormal vegetative morphology. Female sterile. Resistance is recessive in heterozygous duplications from *T(39311)* (PB). Allele RC2 obtained by M. L. Pall. Histidine in test media neutralizes the toxicity of aminotriazole. The mutants *acr-2* (498), *cpc* (61), *leu-1*, and *leu-2* (PB) are also resistant to 3-amino-1,2,4-triazole.

***aur*: aurescent**

Name widely used for *al-1* allele 34508 (IR); with strains carrying this allele only mature peripheral conidia and conidiophores become visibly pigmented. Pigment of *al-1* mutants is said to be pale yellow (1039), but orange has been observed in 34508 strains (PB).

***aza*: azapurine resistant**

The wild type is resistant to azapurines, but certain strains are sensitive. These sensitive strains were used for selecting azapurine-resistant mutants. Three loci are known. *aza-1* and *-2* mutants have been lost, but their described characteristics and map locations of the genes should allow recurrences to be recognized. They were selected by using azaadenine, but at least one allele at each locus resulted in resistance also to azaguanine. The *aza-3* mutant was selected for resistance to azaguanine by a different procedure, and resistance to azaadenine has not been determined. The designation azapurine is proposed for all three loci.

***aza-1*: azapurine resistant-1**

IL. Left of mating type (23%) (*524*).

Resistant to purine analogs 8-azaadenine and 2-6-diaminopurine. (One of four alleles also resulted in resistance to 8-azaguanine.) Obtained by selection in the mutant *mts*, which is inhibited by the analogs. Selected and scored by using 1 mg of 8-azaadenine per ml of medium. Resistance is recessive in heterokaryons. At least one allele results in purine secretion. Called azaadenine resistant. (524) Strains lost (K. K. Jha, personal communication).

***aza-2*: azapurine resistant-2**

IL. Linked to mating type (2%), *aza-1* (39%) (*524*).

Resistant to purine analogs 8-azaadenine and 8-azaguanine. (One of the alleles does not confer resistance to 2-6-diaminopurine.) Obtained and scored as described for *aza-1*. Resistance is recessive in heterokaryons. Called azaadenine resistant. (524) Strains lost (K. K. Jha, personal communication).

***aza-3*: azapurine resistant-3**

III. Linked to *trp-1* (14%) (*462*).

Resistant to purine analogs 8-azaguanine and 6-mercaptopurine. Obtained by selection on limiting adenine in an adenine auxotroph which is inhibited by the analogs. Selected and scored by using *ad-3A ad-3B; ad-2* on medium with 2 μg of adenine sulfate and 200 μg of azaguanine per ml. Relative resistance of $aza\text{-}3^R$ and $aza\text{-}3^S$ strains to 8-azaadenine not determined. Resistance is recessive in heterokaryons. Hypoxanthine can be used as the sole purine supply (461, 462). Called azaguanine resistant.

***azs*: azide sensitive**

Not mapped. Unlinked to *has* (311).

Cannot produce the inducible azide-sensitive respiratory pathway when grown in the presence of chloramphenicol (311). The double mutant *azs; has* has been used to obtain oligomycin-resistant (312) and succinic dehydrogenase-deficient (307) mutants. Obtained from strain ANT-1 (antimycin sensitive; also called alx-1), which segregates for *has* and *azs* (305, 308, 311).

***B^m*: mauve**

IL. Left of *nit-2* (30%) (*395*).

Colonies are mauve on special dye medium where the wild type is blue (395).

***bal*: balloon**

II. Right of *T(AR179)* and, hence, of *thr-2* and *thr-3*. Left of *T(ALS176)* and *arg-5* (1 to 7%) (*789*, 808, 812, PB). Probably left of the centromere (428; L. Garnjobst, personal communication).

Forms a smooth, slow-growing hemispherical colony (789). Glucose-6-phosphate dehydrogenase deficiency (948, 949, 952). (*col-2* and *fr* mutants are also deficient in glucose-6-phosphate dehydrogenase.) Reduced NADPH level (110). Reduced linolenate level (115). Reduced amount of peptides in the cell wall (1165). Fully female fertile, which is uncommon for colonial mutants having such restricted growth. Morphology is subject to alteration by modifiers that are commonly present in laboratory stocks, resulting in spreading growth and conidiation. See *su(bal)*. Photograph (112, 946, 948). Allele C-1405 formerly called *mel-2* (717, 812).

***Ban*: Banana**

IL. Left of *mt* (14%); probably left of *leu-3* (*859*).

Each ascus delimits a single giant ascospore that encloses all four meiotic products and their mitotic derivatives. Dominant and almost completely penetrant. Mature giant ascospores are germinable and usually give rise to mixed cultures. In older perithecia, the prefusion nuclei in the croziers revert to mitosis, which is synchronized and favorable for cytological observation. Vegetative morphology is abnormal. Female sterile with no protoperithecia (859). Used in the study of *Sk* (Spore killer) (857).

***bas*: basic amino acid transport**

Possibly allelic *pmb*, q.v. Called bas^a.

***bat*: basic amino acid transport**

Allelic *pmb*, q.v. (248; R. Sadler and S. Ogilvie-Villa, unpublished data).

***bd*: band**

IVR. Right of *pan-1* (2%) (*918*).

Dense bands of conidia produced on appropriate solid medium (917, 918) at intervals of about 24 h. Conidiation enhanced even on slants (S. Brody, unpublished data). Used extensively to study circadian rhythms (114, 329, 918). *bd* has no effect on underlying clock mechanism, but allows visible expression of rhythm (330). Grows at about 70% of the wild-type rate (S. Brody, unpublished data). CO_2 inhibits conidiation and thus inhibits banding; *bd* mutants are much less sensitive than the wild type to this effect of CO_2 (917). Biotin starvation leads to *bd* phenocopy in the wild type and increased persistence of banding in *bd* mutants (1132). Originally identified in a *bd; inv* strain called "timex" (916). *bd* alone is sufficient to cause banding (918). Used to study conidiation under nonstarvation conditions (928). The double mutant with *csp* eliminates conidial scatter (example: reference 114). Conveniently scored by conidial banding on agar in long tubes or large plates at 25°C in constant dark or in a dark-light cycle, but not in constant light (916).

ben

See *Bml*.

***bis*: biscuit**

Name changed to *pk* (peak), q.v. For nomenclature, see p. 270 of reference 816.

bm-1

Probably allelic *pmb*, q.v.

***Bml*: Benomyl resistant**

VIL. Linked to *cys-2* (2%) and *ylo-1* (2 to 3%); probably between them (*103*, PB).

Resistant to the fungicide benomyl [methyl-1-(butylcarbamol)benzimidazol-2-yl carbamate] (102, 103). Resistance appears dominant in forced heterokaryons (103). Readily scored on 1 μg of benomyl per ml added before autoclaving (PB) or on less in filter-sterilized medium, where 0.2 μg/ml inhibits wild type (O. C. Yoder, personal communication). Called *Ben* (103); *mbic* (49). Renamed to avoid confusion with symbol used for benzene resistance (strain now lost).

***bn*: button**

VII. Right of *T(T54M50)* and, hence, of *thi-3* (2%) (*789*; D. D. Perkins, unpublished data).

Nonconidiating, restricted colonial growth (789). Germination may be better on minimal than on complex complete medium (D. D. Perkins, unpublished data).

***bs-1*: brown spore-1**

IR. Linked to *un-1* (9%), probably to the right (*818*).

Ascospores brown rather than black at maturity and viable. Expressed autonomously, allowing visual scoring in heterozygous asci (818). Used to study factors affecting second-division segregation frequencies (586). Translocation *T(I;IV)NM139 bs* has a similar, inseparable phenotype; the translocation-associated *bs* mutation is not allelic with *bs-1*, although one breakpoint is in IR proximal to *al-2* (808).

c

Used as a symbol for *het-c*, *cy*, and *col-4*.

***caf-1*: caffeine resistant**

VL. Right of *T(OY321)* (11%), *T(AR30)* (19%), and, hence, of *NO*. Left of *T(AR33)* (5 to 12%) and *lys-1* (4 to 14%). (817, PB; *K. S. Hsu, unpublished data*)

Resistant to caffeine (494). Resistance is dominant in duplications from *T(AR33)* (817). Scoring is clear at 25°C and poor at 34°C on slants with 2 or 2.5 mg of caffeine per ml of minimal medium without sorbose (R. L. Metzenberg, personal communication). Also readily scorable by using conidial suspensions spotted on plates containing 2 mg of caffeine per ml of minimal sorbose medium (PB).

***can*: canavanine resistant**

See *cnr*.

***car*: carbohydrate**

IVL. Linked to *cys-10* (1%) (*435*).

Altered morphological rhythm associated with a deficiency in the low-affinity glucose transport system. On glucose, the mutant *car* produces dense and sparse mycelia in cycles (period, ca. 50 h). Originated from a cross between *pat* and *acu-7*, but *acu-7* is not necessary for the phenotype. Periodicity is affected by composition of the medium rather than time, so that the cycle is not circadian as in *bd* or *pat* mutants. On acetate, the mutant *car* is insensitive to the light/dark cycle and has a normal conidiation cycle with a period of about 24 h. Called *LPcar*: long-period carbohydrate (435). The symbol *car* was also used formerly for some carotenoid mutations, at least one of which is an *al-2* allele (1041), and for a carbohydrate mutation (1030) (strain not available for testing).

***cel*: chain elongation**

IVR. Linked to *pan-1* (1%) and *cot-1* (0/17) (*812*).

Requires saturated fatty acids. Defect attributed to impaired chain elongation (455). Deficient in the fatty acid synthetase complex (317). Increased sensitivity to oligomycin (L. R. Forman and S. Brody, cited in reference 283). Tween 20 provides a convenient supplement. Requirement is "leaky" at 21 to 22°C (317) but not at 34°C (812). Used to change fatty acid composition (113) and make the circadian clock sensitive to fatty acids and temperature (114, 649). Temperature compensation of the clock is lacking in *cel* mutants (650). Used to study membrane lipid-phase transitions and electrical properties (363, 364). Used to incorporate photolabile azido fatty acid probes for membrane studies (176). Called *ol* (oleic acid) (812) and *fas* (fatty acid synthesis) (317). Initial report of oleic acid utilization (812) was incorrect (455), probably because of impurities.

***cell-1*: cellobiase/cellulase**

Unmapped. Segregates as a single gene, independent of *gluc-1*.

Constitutive production of both cellulase and cellobiase. Does not affect levels of aryl-β-glucosidase. Recessive to the wild type in heterokaryons (728). Isolated by using *gluc-1* and selecting for high activity in destroying the β-glucoside esculin (300). (Cellulase, cellobiase, and aryl-β-glucosidase are normally induced simultaneously by cellobiose.)

Centromeres

Three methods are available for mapping centromeres relative to flanking gene loci: (i) tetrad analysis with ordered asci (or in unordered asci having other known centromere markers); (ii) duplication coverage of the flanking gene in a segment carrying gene loci known to be located in a given arm; (iii) cotranslocation of the flanking gene, together with other loci whose arm is known, to the arm of a known centric chromosome. (See reference 808.) Method ii is the least laborious when appropriate rearrangements are available for a "left-right" test.

Linkage group arms were defined as left (L) and right (R) (47), using *mt*, *pe*, *ser-1*, *pdx-1*, *ilv-1*, *rib-1*, and *nt* as reference markers.

Centromere I

Right of *T(39311)* and *arg-3* (2%). Left of *T(AR173)* and *his-2* (<1%). (391, 808, 1005, PB; P. St. Lawrence, cited in reference 47, 789, or 812) (Map distance between *arg-3* and *his-2* varies from 3 to 18 units, depending on *rec* genes [174], so that the *arg-3* and *his-2* centromere distances can be larger than indicated.) *sn* and *os-4* also lie between these translocations and are, therefore, the gene markers closest to the centromere.

Centromere II

Probably between *bal* and *arg-5*, from ordered asci (428; L. Garnjobst, personal communication). Right of *T(AR179)* and left of *T(ALS176)* (808, PB); *bal* also lies between these translocations.

Centromere III

Left of *sc* and near *thi-4* by ordered asci (47). Order is uncertain relative to *acr-2*, q.v.

Centromere IV

Left of *psi* (D. R. Stadler, A. M. Towe, and M. Loo, cited in reference 619) and *T(ALS159)* (808). Right of *cut* and *fi* by inference from recombination distances, but no direct evidence.

Centromere V

Right of *T(AR33)* and, hence, of *caf-1*. Right of *T(AR30)* (817). Near and perhaps right of *lys-1* by ordered asci (47). Left of *per-1* (18%) by ordered asci (489).

Centromere VI

Right of *ad-1* (1 to 2%) and *ylo-1* (1 to 6%). Left of *T(AR209)*, *rib-1* (1 to 4%), and *pan-2* (2%) (138, 808, 1012). (*glp-4* is also between *ad-1* and *rib-1* [1102].)

Centromere VII

Right of *T(T54M50)* and, hence, of *csp-2* and *thi-3* (808). Left of *met-7* (<1%, one critical ascus) (M. E. Case, personal communication). Near *sfo* (<1%) and *qa* (no recombination) by ordered asci (318; M. E. Case, personal communication).

***cfs(OY305)*: caffeine sensitive**

IR. Between mating type and *al-2* (*1172*).

Growth inhibited by caffeine (0.2 mg/ml) on minimal medium. Growth stimulated by adenine. Not sensitive to caffeine in the presence of adenine. Slow growth on minimal medium. Morphologically abnormal. Probably UV sensitive by spot testing. Not tested for allelism with *ad-3A*, *ad-3B*, or *ad-9* (1172).

***cfs(OY306)*: caffeine sensitive**

IR. Near *al-2*, probably to the right (*1172*).

Growth inhibited by caffeine (0.2 mg/ml) and by adenine. Slow growth on minimal medium. Not stimulated by complete medium. Morphologically abnormal. Complements *cfs(OY305)* and *cfs(OY307)*. (1172)

***cfs(OY307)*: caffeine sensitive**

IR. Near *cfs-1* between mating type and *al-2* (*1172*).

Growth inhibited by caffeine (0.2 mg/ml) and by adenine. Slow growth on minimal medium. Not stimulated by complete medium. Morphologically abnormal. Complements *cfs(OY305)* and *cfs(OY306)*. (1172)

***chol-1*: choline-1**

IVR. Linked to *ad-6* (1%), probably to the right (633). (482)

Requires choline (470). Also uses mono- or dimethylaminoethanol (468) (Fig. 12). Deficient in *S*-adenosylmethionine:phosphatidylethanolamine-*N*-methyl transferase (222, 924). Abnormal colonial morphology on limiting choline (222). Colonies from single conidia on minimal agar medium resemble inhibited *A/a* duplications, with swollen hyphae and darkening in the presence of phenylalanine plus tyrosine (D. D. Perkins, unpublished data). Abnormal phospholipid composition on limited concentrations of supplement (501). Best scored late on minimal medium. Grows slightly and then stops (D. D. Perkins, unpublished data). Used to study inhibition of cytochrome-mediated respiration and of conidiation when lecithin is depleted by choline starvation (534, 535). Initial allele called 34486.

***chol-2*: choline-2**

VIL. Left of *nit-6* (6 to 8%) (*812*, PB).

Requires choline (471). Also uses di- but not monomethylaminoethanol (468) (Fig. 12). Defi-

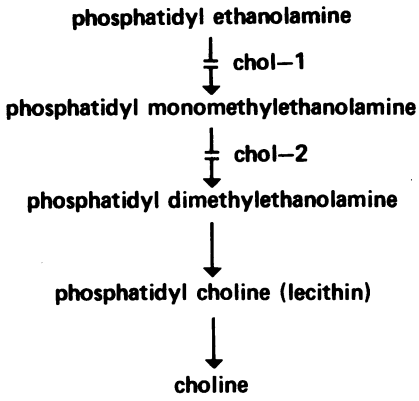


FIG. 12. Biosynthetic pathway of choline, showing sites of gene action (222, 924).

cient in *S*-adenosylmethionine:phosphatidylmonomethylethanolamine methyltransferase (222, 923, 924). Strains carrying the only allele, 47904t, are leaky on minimal medium at 22°C but not at 34°C (501). Phospholipid composition is abnormal on limiting choline (501). Growth is colonial on limiting supplement at 34°C and on minimal medium at 25°C.

***chr*: chrono**

VI. Between *chol-2* (10%) and *pan-2* (*376*).

Altered period of circadian conidiation rhythm. The one allele known is incompletely dominant, specifying a 23.5-h period at 25°C in a csp^+ genetic background. Temperature compensation good above 30°C (375, 377).

***cl*: clock**

VR. Right of *pk* (<1%, 2%) (*296*, 1007).

Spreading flat colonies, forming dense bands. Noncircadian periodicity. Mycelium becomes increasingly dense until growth ceases in all but a few hyphae, which reinitiate the cycle. Grows 1 cm per day. Band size and period modifiable. Photographs (296, 1046). Asci from *cl* × wild type crosses are normal. Homozygous *cl* × *cl* crosses (with backcrossed derivatives) give flaccid asci with unordered ascospores similar to those from *pk* × *pk* crosses. *cl* × *pk* crosses do also, suggesting allelism (1007). *pk* (*bis*) mutants sometimes form growth bands (296). However, substantial crossing-over frequencies and the recovery of *pk cl* double mutants (296) indicate that *pk* and *cl* are at separate loci. Increased activity of L-glutamine:D-fructose-6-phosphate amidotransferase was found in crude extracts of *cl* and five other morphological mutants (899).

***cni-1*: cyanide insensitive-1**

Unmapped chromosomal gene. Not in V (309).

No cyanide-sensitive or antimycin A-sensitive respiration in the first 24 h of growth. Also, initially insensitive to salicyl hydroxamic acid. But salicyl hydroxamic acid and cyanide together inhibit (309). Cyanide-insensitive respiration and the cytochrome *c* level decrease markedly in the late log phase and stationary phase, whereas the cytochrome aa_3 level increases rapidly (559). As shown by electron microscopy, mitochondria are defective in the early log phase, but later resemble the wild type (558). Behaves as a cold-sensitive ribosome assembly mutant. Small subunits are not assembled at low temperatures; the respiratory differences between early and late growth noted above were found at 30°C, and normal aa_3 production occurred throughout growth at 37°C (548). Electron spin resonance data (308). Selected by failure to reduce tetrazolium in overlay after inositol-less death enrichment (310).

***cnr*: canavanine resistant**

IR. Linked to *hom* (1%) and *nic-1* (11%), probably between them (812). (*1061*)

Resistance to canavanine (480, 1061, 1062) is due to a constitutive enzyme that cleaves L-canavanine to hydroxyguanidine plus a compound which can be converted to L-homoserine (617). Resistance is altered secondarily by modifiers that affect rate of uptake (617). Many laboratory strains are resistant, but a few are sensitive (790). Best scored on 0.2 mg of L-canavanine H_2SO_4 per ml (autoclaved in the medium) at 2 days, 34°C (812; D. D. Perkins, unpublished data). Sensitive strain used to select resistant mutants defective in basic amino acid transport (889, 913, 1152); see *pmb*. Called *can*.

***cog*: recognition**

IR. Between *his-3* (1 to 3%) and *ad-3A* (2 to 7%) (*27*, 171).

The postulated site for initiating local recombination in meiosis. Affects recombination in adjoining regions, in the absence of $rec\text{-}2^+$. Presence of cog^+ then increases recombination within *his-3* (27) and crossing over between *his-3* and *ad-3* (171). The allele for high recombination is completely dominant, and its effect is specifically on the *his-3* alleles in chromatids that contain cog^+, as evidenced by crosses heterozy-

gous for reciprocal translocation TM429, which has a breakpoint between sites within the *his-3* locus (171). See *rec* and *rec-2*; see Fig. 22.

coil

IVR. Between *arg-2* (2%) and *leu-2* (12%) (*74*).

Hyphae grow in clockwise coils on the surface of agar medium. Scorable in young cultures microscopically under low magnification (74).

***col*: colonial**

Name used primarily for mutants having restricted mycelial growth that is self-limiting on agar medium. Usually, radial growth of each colony does not exceed a few millimeters. Colonial mutants vary widely in texture, density, conidiation, pigment, and fertility. Mutations of the series *col-5* to *col-17*, described by Garnjobst and Tatum (382), were in some instances assigned new locus names without having been tested for allelism with already named morphological mutations having similar map locations. Some were shown subsequently to be recurrences (e.g., *col-7* of *rg-1; col-14* of *sc*); others may be recurrences but have never been tested (possible examples: *col-5* and *col-8* with *col-1; col-13* and *col-15* with *vel*). Two colonial mutants with nongerminating ascospores are symbolized *le-1* and *le-2*. For reviews covering morphological mutants and morphogenesis, see references 112, 642, 675, 942, 946, 1088. Growth rates and hyphal diameters of numerous colonial mutants are given in reference 197.

***col-1*: colonial-1**

IVR. Linked to *pan-1* (0/47 asci) and *cot-1* (3%) (*46*, 374).

Colonial morphology, with no macroconidiation. Growth cyclic at moderate temperatures; steady at 38°C. Double mutant *col-1 cot-1* grows better than *col-1* at 24°C and better than *cot-1* at 38°C (374). The double mutant *pe; col-1* forms microconidia (46) and was used in early mutation studies, but *col-4; pe fl* was found to be better (386). It was found (416) that *pe; col-1* strains form microconidia at 25°C but form macroconidia at 35°C. Cell wall analysis; photograph (278). *col-5* and *col-8* not tested for allelism.

***col-2*: colonial-2**

VII. Linked to *met-7* and *met-9* (1%), probably to the left (*812*, 816). Right of *T(T54M50)* (D. D. Perkins, unpublished data).

Colonial morphology (46). Photographs (112, 946, 948). Altered structure of NADP-specific glucose-6-phosphate dehydrogenase (116, 949). (*bal* and *fr* mutants are also deficient in glucose-6-phosphate dehydrogenase.) Reduced NADPH level (110). Reduced linolenic level (115). Accumulates much neutral lipid (765). Pyridine nucleotide levels (111). Suppressor *su(col-2)* increases the linear growth rate and influences the electrofocusing pattern of *col-2* glucose-6-phosphate dehydrogenase (948). Homozygous *col-2* × *col-2* crosses fail to mature. Immature asci are frequently nonlinear and occasionally show dichotomization (1007). Hyphae swell with age to diameters of 20 μm (197).

***col-3*: colonial-3**

VII. Linked to *met-7* (0/93) and *wc-1* (1%) (*812*). Complements *col-2* in heterokaryons (K. Wilson, cited in reference 46). Right of *T(T54M50)* (D. D. Perkins, unpublished data).

Colonial morphology (46). Altered 6-phosphogluconate dehydrogenase (947, 949). (*col-10* also affects 6-phosphogluconate dehydrogenase.) Reduced NADPH level (110). Reduced linolenic acid level (115).

***col-4*: colonial-4**

IVR. Between *met-1* (4%) and *arg-2* (<1 to 2%) (*692*, 876, 991). (*695*)

Spreading colonial morphology, forming dense balls of conidia high in slants (47). Probably dominant in heterozygous duplications from *T(S1229)* (E. G. Barry, personal communication). Cell wall-autolyzing enzyme (631). Reduced amount of cell wall peptides (1165). Used in combination with *pe fl* to produce microconidiating colonial growth suitable for reversion experiments (386). Called *spco-1* (382); called *c* (386).

***col-5*: colonial-5**

IVR. Linked to *cot-1* (1%), probably to the right (819). (*382*)

Dense, nonconidiating, poorly pigmenting, slow spreading, colonial morphology (382, 819). Cell wall analysis and photograph (278). Reverts readily (382). Not tested for allelism with *col-1*, which it resembles and which maps in same region. Called *col(B28)* (819).

***col-6*: colonial-6**

IV. Linked to the centromere (0/28 asci) and *pan-1* (21%) (*382*).

Colonial morphology. Slow ascospore germination (382).

***col-7*: colonial-7**

Allelic with *rg-1*, q.v. (675).

***col-8*: colonial-8**

IVR. Linked to *pan-1* (4 to 13%) (*382*).

Colonial morphology, with fluffs of hyphae at top of slants. *col-8* × *col-5* crosses not fertile. Not tested for allelism with *col-1*. (382) Reduced amount of peptides in cell wall (1165).

***col-9*: colonial-9**

VR. Between *inl* (16%) and *asn* (5%) (*698*).

Small, slow-growing colony. Reverts readily (382).

***col-10*: colonial-10**

IIL. Linked to *cys-3* (14%), near or at *pi*. One wild type in 81 progeny from a *col-10* × *pi* cross may have been a revertant (*382*, 816).

Slow growing; dense, compact morphology without conidia (382). Altered 6-phosphogluconate dehydrogenase (947, 949). (*col-3* also affects 6-phosphogluconate dehydrogenase.) *col-10* (R2438) × *pi* (B101) crosses resemble R2438 × R2438 and B101 × B101 crosses in producing abnormal asci with flaccid walls and unordered ascospores, suggesting allelism (1007). However, R2438 and B101 mutants are distinctly different in morphology. Mutant B101 has not been tested for 6-phosphogluconate dehydrogenase. As a marker, *pi* (B101) is preferable to R2438 because of growth rate, stability, and ease of handling; *ro-7* in the same region is preferable to both (PB).

***col-12*: colonial-12**

I. Linked to *mt* (17 to 22%) (*382*).

Colonial morphology. Slow growth from ascospores (382).

***col-13*: colonial-13**

IIIR. Putative *vel* allele. Linked to *tyr-1* (4%) and *col-16* (0/181) (*382*). No direct intercross with *vel*.

Resembles *col-15* and *vel*. Usually nonconidiating, but occasionally forms puffs of aerial conidia at tops of slants; these are not due to reversion (PB). Photograph. (382)

***col-14*: colonial-14**

Allelic with *sc*, q.v. (PB).

***col-15*: colonial-15**

IIIR. Putative *vel* allele (0/26) (PB). Linked to *tyr-1* (5%) and *col-13* (0/181) (*382*).

Resembles *col-13* and *vel*. Occasionally forms puffs of aerial conidia at tops of slants; these are not due to reversion (PB). Reported to complement *col-13* in heterokaryons (382), but this may have been due to misinterpretation of erratic conidiation.

***col-16*: colonial-16**

IIIR. Linked to *leu-1* (1%) and *pro-1* (10%) (*382*; PB).

Colonial morphology (382). Forms balls of powdery conidia at top of slants of glycerol complete medium. A good marker, preferable to *com*; strains carrying the latter grow more slowly and do not conidiate (PB). Complements *mo-4* and *spco-15* (382).

***col-17*: colonial-17**

VII. Linked to *nt* (14%) and *spco-5* (6%) (*382*).

Colonies grow very slowly (382).

***col-le*: colonial, lethal ascospore**

See *le-1*.

***com*: compact**

IIIR. Between *ace-2* (5%) and *ad-4* (<1 to 5%) (578, *814*).

Forms small, slow-growing colonies (278, 789). Grows better on complete medium than on minimal medium (PB). Cell wall analysis; photograph (278). Called B54.

***con-1*: recombination control-1**

Element postulated to lie near *nit-2* (IL) and also proximal to *his-1* (VR), interacting specifi-

cally with the *rec-1* gene product to regulate recombination. No genetic variants are known. (170)

con-2: recombination control-2

Element postulated to lie between *his-3* (IR) and *ad-3*, in or left of the interval *arg-3* to *sn* (I) and also between *pyr-3* and *his-5* (IVR), interacting specifically with the *rec-2* gene product to regulate recombination. No genetic variants are known. (170)

con-3: recombination control-3

Element postulated to lie proximal to *am-1* (VR) and also between *sn* and *his-2* (IR), interacting specifically with the *rec-3* gene product to regulate recombination. No genetic variants are known. (170)

con: (conidiation)

See *cr-1*.

cot-1: colonial temperature sensitive-1

IVR. Between *pan-1* (2%) and *his-4* (1 to 6%) (*692*, 812, 816).

Extremely colonial at 34°C, but completely normal growth, morphology, and fertility at 25°C and below. Linear growth is maximum at 24°C (374). Becomes colonial at 32°C; colonies from ascospores or conidia are viable and continue to grow slowly with dense branching, but do not conidiate. They quickly resume normal growth when shifted to a permissive temperature (692, 1068). Recessive in duplications (808); apparent dominance in heterokaryons (374) may have resulted from a shift in nuclear ratios. Used in studies of septation and branching (202), growth-inhibiting mucopolysaccharide (878, 879), and sulfate transport (641). Cell wall analysis (374). Growth is stimulated by lysine or arginine (0.1 mM) on glucose media at high temperatures (615).

Because of high viability and tightly restricted growth at restrictive temperatures and normality at 25°C, *cot-1* mutants have valuable technical applications. For example, crosses homozygous for *cot-1* have been used in combination with sorbose for experiments with *rec* genes, where high-density ascospore platings are required for precise quantitative analysis of intralocus recombination (e.g., references 165, 997, and 1070). In another application, when shifted up after initial growth at the permissive low temperature, *cot-1* hyphae assume a "bottle brush" appearance with small side branches (692). This has been used to select *uvs* mutants by subsurface survival on UV-irradiated plates containing *p*-aminobenzoic acid (938; D. E. A. Catcheside, personal communication). *cot-1* conidia or ascospores from *cot-1* × *cot-1* crosses are used for replication in a protocol involving transfer by filter paper (615). For suppressors of *cot-1*, see *gul*.

cot-2: colonial temperature sensitive-2

VR. Right of *pk* (8%) and *ser-2* (5%). Left of *ad-7* (4%) (156, 818). (*698*) Recombines with *inv* (5%) (315).

Small colonies at 34°C but fully viable. Growth and morphology nearly normal at 25°C but not completely so (382). Makes altered invertase; it is not clear whether *cot-2* is the structural gene for a second subunit or whether *cot-2* affects structure indirectly, e.g., by altering the carbohydrate moiety (315). See *inv*. Reduced amount of cell wall peptides (1165). Ascospores are normal in heterozygous crosses, but are round in homozygous *cot-2* × *cot-2* crosses (59). Some *cot-2* strains carry *mei-3*, which was found in the original *cot-2* strain (757); however, most strains used by Eggerding et al. (315) are free of it, and *mei-3* cannot be responsible for the effects on invertase (D. Newmeyer, unpublished data).

cot-3: colonial temperature sensitive-3

IV. Left of *pan-1* (16 to 25%). Linked to *arg-2*, probably to the right. (*382*, PB)

Small colonies at 34°C, but fully viable. Growth and morphology normal at 25°C (382).

cot-4: colonial temperature sensitive-4

VR. Right of *ilv-1* (8%) and *rol-3* (5%). Left of *inl* (10%) (*698*). Not allelic with *sp* (11%) (PB).

Small colonies at 34°C, spreading at 25°C (382). Morphology at 25°C resembles that of the mutant *sp*, with late-forming blooms of conidia on aerial hyphae, but *sp* is not heat sensitive. Good female fertility, but no perithecia in homozygous *cot-4* crosses (PB).

cot-5: colonial temperature sensitive-5

IIL. Right of *T(P2869)* and *T(B18)*. Probably left of *pyr-4* (0/39). Linked to *fs-1* (29%) (PB). (*382*)

Little or no growth at 34°C; colonial at 30°C. Morphology still not normal at 25°C, a temperature at which older colonies form short aerial hyphae (382). Female sterile. Morphology distinct from that of the mutant *fs-1* (PB).

***cpc-1*: crosspathway control-1**

VI. Right of *ylo-1* (1 to 3%) (*238*).

Affects simultaneously both ability to derepress and basal levels of enzymes in arginine and other amino acid biosynthetic pathways. *cpc-1* mutations interfere with cross-pathway control of amino acid biosynthetic enzymes. Sensitive to 3-amino-1,2,4-triazole. Isolated as arginine auxotrophs by selection in the mutant *arg-12*^{s}, q.v. The *cpc-1; arg-12*$^{+}$ single mutant is prototrophic. Delayed growth after ascospore germination; the delay is not alleviated by arginine or precursors. Scorable by delayed growth. (61, 238) Exemplified by alleles CD-15 and CD-55 in reference 238 and j-2, j-5, and j-9 in reference 61.

***cpl-1*: chloramphenicol sensitive-1**

VIL. Between *ad-8* (6 to 11%) and *lys-5* (6%) (*180*, PB).

Sensitive to chloramphenicol (<0.5 mg/ml added to autoclaved medium) and to antimycin A (1 μg/ml). (The wild type is resistant to 4 mg of chloramphenicol per ml.) Protein synthesis is not grossly altered. Cyanide-insensitive and azide-insensitive respiratory systems are still present. Cytochrome spectrum normal on minimal medium. Obtained by inositol death enrichment and replica-plating, using a strain of genotype *inl; trp-3; sn cr-1* (180, 182). Scorable on 0.5 mg of chloramphenicol per ml autoclaved in medium (PB).

***cpt*: carpet**

IIR. Right of *arg-5* (3%). Left of *T(NM177)* and *pe* (6%) (*812*, 808).

Flat, slow-growing mycelium with no macroconidia. Produces microconidia (812; S. R. Gross, personal communication), but much less abundantly than do the double mutants *fl; dn* and *pe fl* (PB). Homozygous fertile.

***cr-1*: crisp-1**

IR. Right of *ace-7* (1 to 3%) and *nic-2* (4 to 7%). Left of *cys-9* (3%) and *un-1* (5%) (721, 816). Included in duplications from *T(4540)*, which do not include *cr-2* or *cr-3* (PB). (*610*)

Rapid conidiation close to surface of agar. Produces very short conidiophores, bearing conidia in tight clusters (610, 611). Photographs (533, 634). Recessive. Deficient in adenylate cyclase (1066); has little or no endogenous adenosine 3′,5′-phosphate (1065, 779). Abnormal morphology partially corrected by exogenous adenosine 3′,5′-phosphate (891, 892, 1065, 1066). Guanosine 3′,5′-phosphate also stimulates mycelial elongation (892). Cyclic nucleotide levels differ in mycelia and conidia (891, 892). NAD(P) glycohydrolase is overproduced and excreted; this is normalized by adenosine 3′,5′-phosphate (533). Induction and localization of β-glucosidase is altered; induction is normalized by adenosine 3′,5′-phosphate (906). Inability to use glycerol and certain other carbon sources is also overcome by adenosine 3′,5′-phosphate (598, 1067). Phosphodiesterase inhibitors do not counteract the morphological effect of *cr-1* (892). Increased lactate dehydrogenase activity (92). Used to determine what functions are controlled by adenosine 3′,5′-phosphate (779). Used to study adenosine 3′,5′-phosphate binding protein (1082).

Strains carrying the various alleles vary in growth habit (B123 strains are flat, restricted; allele L strains are spreading, but morphology may vary on different media). Modifier mutations which alter morphology and the ability of *cr-1* to use glycerol occur frequently (383, 905). Crosses homozygous for allele B123 exude intact linear asci (634). Double mutants *sn cr* and *cr rg* form small conidiating colonies suitable for replica plating with velvet (182, 634, 796, 932, 1020). The triple mutant *sn cr; csp-2* can be overlayered (744; photograph 747). The single mutant (B123) can be replicated by using a needle replicator (634). Scorability and viability are good. Excellent as a marker. Carotenoids formed normally. *cr-1* ascospores may require longer to mature than *cr*$^{+}$ ascospores. Allele C-E4-11-67 called *con* (716, 717).

***cr-2*: crisp-2**

IR. Right of *cr-3* (11%) and *T(NM103)*; hence, right of *thi-1*. Left of *al-2* (18%) (*383*, PB).

Conidiation delayed. Fine, pale-pigmented conidia produced in clumps over the agar surface (383). Recessive. Reduced amount of cell wall peptides (1165). Overproduces and excretes NAD(P) glycohydrolase, but this is not cured by exogenous adenosine 3′,5′-phosphate (533).

***cr-3*: crisp-3**

IR. Right of *cr-1* (13%) and of *T(4540)*; hence, right of *cys-9* and *un-1*. Left of *cr-2* (11%) (*383*, PB).

Delayed conidiation; ultimately producing fine, pale conidia uniformly over the agar surface (383). Recessive. Reduced amount of cell wall peptides (1165). Overproduces and excretes NAD(P) glycohydrolase; this is not cured by exogenous adenosine 3',5'-phosphate (533).

***crib-1*: cold-sensitive ribosome biosynthesis**

IV. Linked to *met-1* (6%) (*927*).

Defective ribosome biosynthesis below 20°C; attributed to a defect in ribosomal ribonucleic acid (rRNA) processing (897). Grows at 6% the wild-type rate at 10°C and 79% at 25°C. 37S cytosolic ribosomal subunits are underaccumulated, and relatively little stable 17S rRNA is produced at low temperatures. Not a conditional lethal mutation (896, 927). Conditionally defective in expression of *S*-adenosylmethionine synthetase activity (900).

***crib(PJ31562)*: cold-sensitive ribosome biosynthesis**

IVR. Near *crib-1* (3%) (*895*).

Defective in biosynthesis of cytosolic ribosomes at 10°C, but normal at 25°C. Grows at 16% of the wild-type rate at 10°C and 90% at 25°C. Underaccumulates 17S rRNA and, hence, 37S ribosomal subunits. Partial complementation in forced heterokaryons with *crib-1* (895). Called PJ31562.

***csh*: cushion**

IR. Between *thi-1* (12 to 20%) and *ad-9* (5%) (816). (*P. St. Lawrence, cited in references 47, 789 or 812*)

Restricted colonial growth (812).

***csp-1*: conidial separation-1**

IL. Between *arg-3* (1%) and the *T(39311)* right breakpoint (*972*, PB).

Conidia fail to separate and become airborne. Photograph (972). Recessive. Cultures on agar readily scored by the "tap test." In water, conidia are freed at 1/10 the wild-type concentration (972). Used in connection with *bd* for study of circadian rhythms (e.g., reference 114). Useful in student laboratories to avoid contamination (966). Carotenoids tend to be yellowish in young cultures (PB).

***csp-2*: conidial separation-2**

VIIL. Linked to *thi-3* (<1%), probably to the right. Left of *T(T54M40)* (*972*, PB).

Conidia fail to separate and become airborne. Cultures on agar readily scored by the tap test. Resembles *csp-1*. Conidia are freed in water suspension long after induction of aerial growth and at only 1/100 the concentration of the wild type. A *csp-1; csp-2* double mutant releases no detectable free conidia under the same conditions (972). Most *csp-2* alleles complement *csp-1* in forced heterokaryons to form the wild-type number of free conidia (972), but *csp-2* (UCLA102) does not (969). Conidiating colonies of the *csp-2; sn cr-1* strain on replica plates can be overlayered without the conidia being spread (744); photograph (747).

***cum*: cumulus**

IIIL. Left of *r(Sk-2)* (4%), *acr-7* (5 to 18%), and *acr-2* (18%). No recombination with $Sk\text{-}2^{K}$. (*PB*; B.C. Turner, personal communication)

Initially colonial; then spreads and sends up blooms of aerial hyphae which conidiate profusely at the shallow ends of agar slants. Good female fertility. Similar in morphology to the mutants *sn* and *sp* and to the mutant *cot-4* at 25°C (PB).

***cut*: cut**

IVL. Between *cys-10* (28 to 37%) and *fi* (4 to 10%) (*802*; D. D. Perkins, unpublished data). Linkage to IR, shown in original *cut* strain HK53, was due to an unrecognized I;IV translocation (808). When an allelic *cut* point mutation (LLMI) became available, it was mapped in IV rather than in I and segregated independently of I markers (802).

Sensitive to high osmotic pressure. Phenotype similar to that of *os* mutants. Scorable either by morphology or by failure to grow on agar containing 4% NaCl. Morphology approaches normal at high humidity (573). Allele HK53 is inseparable from *T(I;IVL)HK53* (808).

***cwl*: cross wall**

II. Linked to *arg-5* (3%) (*PB*).

Hyphal septa are largely absent. Hyphae tend to bleed, forming an exudate on the agar surface and in lens-shaped pockets beneath. Slow growing, aconidial. Subject to alteration by modifiers that restore septa and increase growth rate, but original mutant gene can be extracted by crossing. Recessive. Stocks conveniently kept as heterokaryons (382; A. Hammill, via FGSC; PB). Called *mo(R2441)*.

***cy*: curly**

IL. Linked to *arg-1* (0/34) and *ad-5* (1/54), probably to the left (PB). (*689*)

Curly hyphae grow at wild-type rate (689). Gross morphology is indistinguishable from that of the wild type. Scorable by examining young hyphae on agar or glass walls of culture tubes before conidiation. Symbol changed from *c* to avoid confusion with *het-c*.

***cya-1*: cytochrome *a*-1**

IL. Linked mating type (6%), indicated to the left (*87*).

Deficient in cytochrome aa_3. Cannot reduce tetrazolium. Very slow growth. Female sterile (87). Possibly not really a *cya* mutant (H. Bertrand, personal communication).

***cya-2*: cytochrome *a*-2**

VR. Linked to *al-3* (3%), indicated to the right (*87*).

Deficient in cytochrome aa_3. Cannot reduce tetrazolium. Slow growth. Female sterile (87).

***cya-3*: cytochrome *a*-3**

VII. Between *chol-2* (10%) and *cyt-2* (10%) (*87*).

Deficient in cytochrome aa_3. Cannot reduce tetrazolium. Slow growth (87). aa_3 deficiency suppressed by antimycin A (84). Spectrum (84).

***cya-4*: cytochrome *a*-4**

IIL. Linked near *thr-3* (*87*).

Deficient in cytochrome aa_3. Cannot reduce tetrazolium. Slow growth. Spectrum (84, 87). Cytochrome oxidase subunits 5 and 6 are deficient or lacking (90).

***cya-5*: cytochrome *a*-5**

IVR. Right of *pan-1* (2%) (*739*).

Deficient in cytochrome aa_3. Slow growth. Subunit 1 polypeptide of cytochrome *c* oxidase absent by immunological criteria. Poor recovery (10%) from crosses. Selected as tetrazolium nonreducer. (90, 739, 740) Called *cya-U-34*.

***cya-6*: cytochrome *a*-6**

IVR. Right of *pan-1* (2%) (*739*).

Deficient in cytochrome aa_3. Alleles 2 and 35 are heat sensitive. Selected at 41°C by slow growth on salicylhydroxamic acid and resistance to tetrazolium. At least five subunits of cytochrome *c* oxidase are present at 41°C by immunological criteria, but are not associated. Complements *cya-5*. (739) Alleles called *cya-6-2* and *cya-6-35*.

***cya-7*: cytochrome *a*-7**

III. Linked to *ad-4* (25%) (739).

Deficient in cytochrome aa_3. Allele *cya-7-13* is heat sensitive. Selected at 41°C by slow growth on salicylhydroxamic acid and resistance to tetrazolium. At least five subunits of cytochrome *c* oxidase are present at 41°C by immunological criteria, but are not associated. (739) Called *cya-7-13*.

***cyb-1*: cytochrome *b*-1**

VR. Between *al-3* (24%) and *his-6* (10%) (*87*).

Deficient in cytochrome *b*. Cannot reduce tetrazolium. Slow growth. Spectrum (84, 87). Suppresses the aa_3 deficiency of the mutant *cyt-2* and of mitochondrial mutant [*mi-3*] (84).

***cyb-2*: cytochrome *b*-2**

Unmapped. Report of VI linkage (87) may be incorrect (H. Bertrand, personal communication).

Deficient in cytochrome *b*; erratic deficiency of aa_3 (H. Bertrand, personal communication). Cannot reduce tetrazolium. Very slow growth. Reduced female fertility. Spectrum. (84, 87)

***cyb-3*: cytochrome *b*-3**

IIL. Left of *ro-3* (9%) (*PB*).

Deficient in cytochrome *b*. Cannot reduce tetrazolium. Slow growth. Heat sensitive: mutant phenotype at 38 to 39°C; nearly normal at 25 and 34°C (1133). Grows slowly from ascospores at 34°C.

cyh-1: cycloheximide resistant-1

IR. Right of *nit-1* (6%). Left of *T(STL76)* and *al-2* (8 to 13%) (*496*, 797, 808).

Resistant to cycloheximide (496, 748). Resistance is recessive in duplications (1090). Dominance reported in forced heterokaryons (496, 748) may have been due to skewed nuclear ratios (1090). Protein synthesis on ribosomes of the mutant *cyh-1* proceeds in the presence of cycloheximide in a cell-free system (834). Readily scored on slants with 10 μg of cycloheximide per ml autoclaved in the medium. Excellent as a marker and valuable for selecting somatic recombinants or deletions in heterozygous duplications (748, 1091). Used to show that the cycloheximide-induced phase shift of the circadian clock involves protein synthesis (738). Called *act-1*: actidione resistant-1.

cyh-2: cycloheximide resistant-2

VR. Right of *lys-2* (<1%). Left of *leu-5* (<1 to 2%) and *sp* (2 to 9%) (*496*, 818, PB).

Resistant to cycloheximide (496, 748). Protein synthesis on mutant ribosomes proceeds in the presence of cycloheximide in a cell-free system (834). Excellent marker. Readily scored on slants with 10 μg of cycloheximide per ml autoclaved in the medium or with 1 μg added after autoclaving. Resistance in heterokaryons has been reported to be dominant (496, 626) or recessive (939); it may depend on nuclear ratios or media. Used in mutagenicity test systems (626). Used to show that the cycloheximide-induced phase shift of the circadian clock involves protein synthesis (738). Double mutant *cyh-1; cyh-2* grows slowly and is much more insensitive to cycloheximide than either single mutant (496).

cyh-3: cycloheximide resistant-3

Unmapped. Unlinked to *cyh-2*. Stated to be distinct from *cyh-1* (1108).

Resistant to 100 μg of cycloheximide per ml. Double mutant *cyh-2; cyh-3* is morphologically abnormal, resistant to >2,400 μg/ml (1108). The one known allele, CH96, was first called *act-5* (1107) and then *act-3* (1108).

cys: cysteine

Cysteine auxotrophs are characterized as being unable to use inorganic sulfate but able to grow on either cysteine or methionine (721). Some *cys* mutants can use sulfite or thiosulfate. The sulfur permease mutants *cys-13* and *cys-14* are exceptions, having no demonstrable requirement. Cysteine mutant strains tend to accumulate secondary mutations in the pathway, suggesting that some double-mutant combinations have a selective advantage over single-mutant strains (721). Unambiguous definition of loci based on map location is thus important, and care must be taken that derived stocks carry the original *cys* mutation. Cysteine and methionine loci provide several examples of closely linked pairs of genes: *cys-1 cys-2*, *cys-5 cys-11*, and *met-7 met-9*. With at least two *cys* mutants (*cys-3*, *cys-5*), and possibly others, ascospore maturation and recovery of *cys* progeny requires that crossing medium be supplemented, even when the protoperithecial parent is cys^+. Cysteine mutants grown on limiting supplement show a shortened period of circadian conidiation rhythm (329, 333). Partial suppressors of *leu-4* which originate as double-mutant microcolonies on minimal medium are leaky *cys* mutants of various types (425); see *leu*.

cys-1: cysteine-1

VII. Between *cys-2* (1 to 3%) and *ylo-1* (8%) (721, 1012). (*980*)

Uses sulfite, thiosulfate, cysteine, or methionine. Original isolate (allele 84605) also had a partial requirement for tyrosine and showed high tyrosinase activity at 25°C but not 35°C (479, 721, 980). These properties reverted, however, whereas the cysteine requirement is stable (479). Used in studies of intra- and interlocus recombination (721, 1015, 1016, 1024–1026).

cys-2: cysteine-2

VII. Between *un-4* (4%) and *cys-1* (1 to 3%). Very close to or contiguous with *cys-1*, but is probably a separate locus (721, 1012). (*M. Fling, cited by T. H. Pittenger, Genetics 39:326–342, 1954*)

Uses cysteine or methionine. Strains carrying these alleles are heterogeneous in response to thiosulfate, but do not use sulfite (721). Lacks sulfite reductase, as do the *cys-4* and *cys-10* mutants (596). No interallelic complementation. Used in studies of intra- and interlocus recombination (see *cys-1*).

***cys-3*: cysteine-3**

IIL. Right of *pi* (4%). Left of *T(AR18)* and *pyr-4* (18 to 21%) (*721*, 808, 816).

Uses sulfite, cysteine, or methionine; little or no response to thiosulfate (640, 721). Regulator of genes of sulfur uptake and metabolism (e.g., sulfate permease, aryl sulfatase, choline sulfatase) (284, 640, 667). Grows well on methionine. Resistant to chromate (640). Used extensively for studying regulation; for a review, see reference 642. *cys-3* ascospores darken slowly or not at all, even when a *cys-3* strain is the fertilizing parent and when a strain carrying heat-sensitive allele NM27t is crossed at 25°C, the permissive temperature for growth. Adding methionine to crossing medium promotes darkening but fails to give good recovery of *cys-3* progeny. Recovery of a few percent *cys-3* progeny is possible in well-aged crosses. (721, PB). *cys-3* can be used effectively as an autonomous ascospore color mutant for demonstrating segregation patterns in asci (811; see reference 858 for photograph; see Ascospore color mutants).

***cys-4*: cysteine-4**

IVR. Right of *mat* (10%) and *T(NM152)*. Left of *uvs-2* (5%) (721, 808, 1023). (*815*)

Uses cysteine; slight response to thiosulfate (721). Poor growth on methionine. Lacks sulfite reductase, as do *cys-2* and *cys-10* mutants (596).

***cys-5*: cysteine-5**

IL. Between *leu-4* and *ser-3* (0.1%) (816, 1125). (*N. H. Horowitz, cited in references 721 and 815*) Probably a locus distinct from *cys-11*, with the order *leu-3 cys-5* (<1%) *cys-11 mt* in a cross showing no negative interference (723).

Uses sulfite, thiosulfate, cysteine, or methionine (721). Lacks 3'-phosphoadenosine-5'-phosphosulfate reductase (F.-J. Leinweber, cited in references 721 and 723). Enzymatically distinct from *cys-11* (adenosine 5'-triphosphate sulfurylase), which it complements in heterokaryons (721, 723; F.-J. Leinweber, cited in references 721 and 723). Leaky, but not so as to interfere with scoring. Ascospores may be oozed from perithecial beaks rather than shot. For good recovery of *cys-5* progeny, crossing media should be supplemented even when the protoperithecial parent is cys^+; otherwise *cys-5* ascospores may fail to blacken. *cys* (NM86) and *cys* (85518), initially listed as *cys-5* alleles, are now designated *cys-11*.

***cys-6*, *-7*, *-8*: cysteine-6,-7, -8**

Lost. Identity or nonidentity with other loci was never established (721, 823).

***cys-9*: cysteine-9**

IR. Between *cr-1* (3%) and *thi-1* (13%) (*721*).

Uses sulfite, thiosulfate, cysteine, or methionine. Somewhat leaky. (721)

***cys-10*: cysteine-10**

IVL. Left of *acon-3* (1 to 6%), *ace-4* (19 to 33%), and *cut* (28 to 37%) (578, PB). (*721*)

Uses cysteine, cystathionine, homocysteine, or methionine, with a slight response to thiosulfate (469, 596, 721); however, E. Käfer (personal communication) found good growth on thiosulfate. Growth is better on casein hydrolysate than on methionine (D. D. Perkins, unpublished data). *cys-10 chol-1* double mutants grow better on methionine alone than on methionine plus choline (721). Lacks sulfite reductase, as do *cys-2* and *cys-4* mutants (596). Formerly called *met-4*; see reference 721.

***cys-11*: cysteine-11**

IL. Linked to *cys-5* (<1%), between *leu-3* (8%) and mating type (5%). Probably a locus distinct from *cys-5*, with the order *leu-3 cys-5* (<1%) *cys-11 mt* in crosses showing no negative interference (721, 723). (*N. H. Horowitz, cited in references 721 and 815*)

Uses sulfite, thiosulfate, cysteine, or methionine (721). Affects adenosine 5'-triphosphate sulfurylase (639; F.-J. Leinweber, cited in references 721 and 723). Enzymatically distinct from *cys-5* (3'-phosphoadenosine-5'-phosphosulfate reductase), which it complements in heterokaryons (721, 723; F.-J. Leinweber, cited in references 721 and 723). Called *cys(NM86)* (721). The *cys*(85518) mutant also lacks adenosine 5'-triphosphate sulfurylase (639), and thus *cys*(85518) is evidently an allele of *cys-11* rather than of *cys-5*; this is in harmony with the existing recombination data (723).

***cys-12*: cysteine-12**

IR. Right of *ad-9* (12%); linked to *al* (0/76) (*723*).

Uses cysteine or methionine (723). No information on precursors used.

***cys-13*: cysteine-13**

IR. Right of *his-3* (2%) (*640*).

Resistant to chromate; no demonstrable requirement. Deficient in sulfur permease I (conidial type) (639, 640). Score on minimal agar with 25 mM chromate and 0.25 mM methionine after 3 days or longer, 34°C. (Strains carrying regulatory gene *cys-3* are also chromate resistant.)

***cys-14*: cysteine-14**

IV. Linked to *cot-1* (21%) (*640*).

Deficient sulfate transport in the mycelial stage, but sensitive to chromate. Lacks sulfur permease II (mycelial type) (639, 640). Double mutant *cys-14; cys-13* cannot transport inorganic sulfate, grows on methionine; both single mutants are prototrophic (640).

***cys-15*: cysteine-15**

IVR. Between the *T(S1229)* breakpoints; hence, right of *pdx-1* (0/55 asci). Left of *met-1* (3%) (55, *768*, 808).

Unable to use sulfate. Uses sulfite, thiosulfate, cysteine, or methionine (721, 768). Only one allele is known, with requirement not separated from a deficiency of D-amino acid oxidase (768); this is thought to be due to a closely linked coincident lesion. (See *oxD* for other D-amino acid oxidase mutants having no cysteine requirement.) Formerly called *cys(oxD^1)*.

cys(oxD^1)

Changed to *cys-15*.

***cyt*: cytochrome**

Cytochrome-deficient Mendelian mutations have been subdivided into three main classes as follows (87): *cyt*, deficiency of more than one cytochrome; *cya*, deficiency of cytochrome aa_3; *cyb*, deficiency of cytochrome *b*. Apart from diagnostic spectra, the mutants are characterized by slow growth (4 to 7 days for conidiation of mutants versus 3 days for the wild type) and by the inability to reduce tetrazolium (87). These properties have been used in screening new mutants. *cni* also affects cytochrome spectra (309). The relation of *tet* (tetrazolium-resistant nonreducer) to cytochrome mutations is not known. Cytochrome defects that result from mutations of the mitochondrial genome (e.g., [*mi-1*] and [*mi-3*]) are not considered here except as they interact with chromosomal genes such as *su([mi-1])*; see reference 394.

***cyt-1*: cytochrome-1**

IL. Between *leu-3* (5 to 8%) (583) and *T(OY321)* (D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation). (*694*)

Deficient in cytochromes aa_3 and *b*. Very slow growth. Female sterile (87, 694). Scoring aided by slower growth on complete medium relative to that on minimal, presumably owing to inhibition by yeast extract (694, 816). *cyt-U-9* is linked and may be allelic with the original *cyt-1* mutation C115 (87; H. Bertrand, personal communication).

***cyt-2*: cytochrome-2**

VIL. Between *cya-3* (10%) and *lys-5* (6%) (87, 1012). (*694*)

Completely deficient in cytochromes aa_3 and *c*. Very slow growth. Female sterile. Complements *cyb-2*. (87, 694) Spectrum (87). Cytochrome oxidase subunit 1 polypeptide abnormal (90). aa_3 deficiency suppressed by *cyb-1* and by antimycin A (84).

***cyt-3*: cytochrome-3**

The original mutation *cyt-3-5* (87) is evidently an allele of *cyt-4* (H. Bertrand, personal communication).

***cyt-4*: cytochrome-4**

IR. Between the breakpoints of *T(AR173)*; hence, right of the centromere, *sn*, and *arg-3* (2%); left of *nuc-1* and *lys-4* (808; D. D. Perkins, unpublished data). (*87*)

Deficient in cytochromes aa_3 and *b*. Slow growth. *cyt-4-7* and strains carrying the three alleles listed below are all defective in splicing of the large mitochondrial rRNA (83; H. Bertrand, personal communication). Phenotype partially suppressed by the electron transport inhibitor antimycin (83). Complements *cyt-18* and *cyt-19*, which are also required for the splicing of large mitochondrial rRNA (83). *cyt-3-5*, *cyt-U-10*, and *cyt-U-14* of reference 87 are all alleles of *cyt-4* and have been designated *cyt-4-5*, *cyt-4-10*, and *cyt-4-14*, respectively, by H. Bertrand (personal communication).

***cyt-5*: cytochrome-5**

IVR. Left of *trp-4* (9%) (*87*).

Deficient in cytochromes aa_3 and *b*. Slow growth. Not defective in the splicing of mitochondrial rRNA, unlike *cyt-19* mutants (*cyt-19* is closely linked but complements *cyt-5*) (83, 87). Allele *cyt-5-4* was previously called *cyt-4*.

***cyt-6*: cytochrome-6**

VII. Near *wc-1* (2%). Indicated to the left (*87*).

Deficient in cytochromes aa_3 and *b*. Slow growth. *cyt-U-18* may be an allele (0/193 cyt^+ from a cross with *cyt-6*; positive complementation test). Not tested for allelism with *slo-2*. (87)

***cyt-7*: cytochrome-7**

VIIL. Linked to *nic-3* (18%). Indicated to left (*87*).

Deficient in cytochromes aa_3 and *b*. Slow growth. Possible allele of *su([mi-1])-5*. (87)

***cyt-8*: cytochrome-8**

IIIR. Linked near *ad-4* (*87*).

Deficient in cytochromes aa_3 and *b*. Possible allele of *su([mi-1])-1*. (87)

***cyt-9*: cytochrome-9**

V. Between *lys-1* (5%) and *at* (5%) (*87*, PB).

Deficient in cytochromes aa_3 and *b*. Slow growth. (87)

***cyt-12*: cytochrome-12**

IIR. Between *thr-3* (38%) and *trp-3* (18%). (*87*)

Incompletely deficient for cytochromes aa_3 and *c*. Slow growth. Female sterile. Complements *cya-4*. Spectrum. (87)

***cyt-18*: cytochrome-18**

IR. Linked to *al-2* (10%) and *nic-1* (1 to 5%) (*203*, 635).

Heat sensitive; grows slowly. Deficient in cytochromes aa_3 and *b* at 37°C (832). Mitochondrial protein synthesis and assembly of small mitochondrial subunits are also abnormal (205). A novel large RNA precursor (35S) is found at restrictive temperatures (205); the intervening sequence of large (25S) mitochondrial rRNA is apparently not excised. Two alleles (289-67, 299-9) differ in speed of turn-off of RNA processing when the temperature is shifted (635). Complements *cyt-4* and *cyt-19*, which are also required for splicing of large mitochondrial rRNA (83).

***cyt-19*: cytochrome-19**

IVR. Linked to *cyt-5* (1/201) and *trp-4* (9%) (83, *87*).

Deficient in cytochromes aa_3 and *b*. Slow growth. Required for splicing of mitochondrial large rRNA. Complements *cyt-5*. Complements *cyt-4* and *cyt-18*, which are also required for splicing (83). Called *cyt-U-19* (87).

***cyt(289-56)*: cytochrome**

IL. Linked to *mt* (0/151) (*203*).

Deficient in the small subunit of mitochondrial ribosomes, but contains normal ratios of 19S to 25S rRNA in whole mitochondria (205).

***cyt(297-24)*: cytochrome**

II. Linked to *thr-3* (33%), probably to the left (*203*).

Deficient in the small subunit of mitochondrial ribosomes. 19S rRNA is rapidly degraded. The phenotype resembles that of the extranuclear [*poky*] mutant (205), but the deficiency is not suppressed by *su([mi-1])-4* or *su([mi-1])-5* (203).

d

Used as symbol for *het-d*, q.v.

***da*: dapple**

IIL. Linked to *thr-3* (3%) and *arg-5* (*812*, 818). Right of *T(NM149)* (D. D. Perkins, unpublished data).

Produces flecks of conidia on the agar surface (812).

***del*: delicate**

VIR. Right of *rib-1* (12%) and *pan-2* (6%). Left of *trp-2* (0 to 13%) (818). (*789*)

Growth less profuse than that of the wild type; flat, with fine aerial growth at the tops of slants. Prolific hyphal branching. Scorability good. (789)

***dgr*: deoxyglucose resistant**

V. Linked to *caf-1* (8%) (*B. M. Eberhart, personal communication*).

Resistant to inhibition by 2-deoxy-D-glucose. Grows more slowly than the wild type on standard media, but growth is initially faster than that of the wild type on media with mono- or disaccharides plus deoxyglucose. The greatest differential growth in 0.05% deoxyglucose is obtained with 0.1% cellobiose, trehalose, lactose, fructose, or galactose. (298)

***dir*: dirty**

IR. Right of *pa* (37%) (*610*).

Conidia few and misshapen; yellowish exudate (611). Photograph (610). (Stock lost. Possibly *os-1*?)

***dn*: dingy**

IVR. Right of *pyr-2* (4%). Linked to *mat* (1%) (*692*, 812).

Abnormal morphology, slower than normal growth, producing grey patches of microconidia in addition to macroconidia (692). When in combination with *fl*, microconidia are produced exclusively and in abundance, as in the double mutant *pe fl*. The double mutant *fl*; *dn* is fully fertile in homozygous crosses (811) and for this reason may be preferred to *pe fl* as a microconidating strain. Microconidia from *fl; dn* are less viable, however (454).

***do*: doily**

VIIL. Left of *nic-3* (1 to 3%) (304, 812). Linked to *spco-4* (1/>400) (816). (*D. R. Stadler, cited in reference 812*)

Restricted colonial growth (D. R. Stadler, cited in reference 812); growth rate is 4% of the wild-type rate (304). The cell wall galactosamine is 0.5% the wild-type level, the uridine 5′-diphosphate *N*-acetylgalactosamine content is 3%, and the specific activity of uridine 5′-diphosphate *N*-acetylglucosamine-4-epimerase in cell extracts is 20% that of the wild type. Partial back-mutations can differentially affect cell wall and alcohol-soluble galactosamines, indicating pleiotropy (304). Cell walls have a reduced amount of peptides, and the peptides have altered diethylaminoethyl cellulose elution profiles (1165).

***dot*: dot**

IR. Linked to *ad-9* (0/44), right of *thi-1* (2%) (816). (*812*)

Colonial growth (812). More restricted on glycerol complete medium than on minimal medium. A possible "maternal effect" is seen in dot^+ progeny from heterozygous crosses.

***dow*: downy**

IIIR. Right of *ty-1* (21%) and *un-17* (23%); linked to *erg-3* (10%) and *acu-7* (0/72) (*816*).

Soft, matty growth, conidiating and covering slants (816). An excellent marker: good viability, fertility, and scorability.

***dr*: drift**

VIIR. Right of *for* (3%), left of *T(5936)* and *arg-10* (12%) (808, *819*, PB).

Forms conidia in dense masses at the tops of slants; growth elsewhere is flat on the surface. Good scorability. (819) Called *mo(P1163)*.

e

Used as symbol for *het-e*, q.v.

***eas*: easily wettable**

IIR. Linked to *rip-1* (1/151), *trp-3* (0/71), and *fl* (1/52) (PB). (*967*)

Conidia and aerial hyphae are readily wetted by water; in contrast, those of wild type are hydrophobic. Resembles *csp* mutants in that conidia do not readily become airborne, but differs from *csp* mutants in that conidia do not remain joined in the proconidial chains (967). Rodlets are lacking from the surface of conidia (75). Conidiating cultures can be scored by adding a drop of water to the culture, by tapping an inverted slant (967), or by transferring conidia to liquid (PB). Not scorable in combination with *fl*. Somewhat sensitive to high osmotic pressure (PB). A class of slow-growing progeny is produced from crosses heterozygous or homozygous for *eas* (811).

***edr-1*: edeine resistant-1**

VI. Linked to *ad-1* and *pan-2* (0/125) (*1064*).

Resistant to edeine. Selected and tested on 200 μg of edeine per ml (only a fraction of *edr* conidia grow). Recessive. Called *ed*^{r}*-1* (1064).

***edr-2*: edeine resistant-2**

VII. Left of *ad-1* (19%) (*1064*).

Resistant to edeine. Selected and tested on 200 μg of edeine per ml (only a fraction of *edr* conidia grow). Recessive. (1064) In intact cells, edeine inhibits the syntheses of protein, deoxyribonucleic acid (DNA), and RNA in the wild type but not in the mutant; in vitro, edeine inhibits protein synthesis equally in both the mutant and wild type. Hence, the mutant is thought to have a block in edeine uptake. (1110) Called *ed*^{r}*-2*, *ed*^{r}*-29*.

en(am)-1*: enhancer-1 of *am

VR. Between *am* (8%) and *inl* (1%). Linked to *gln* (1%). (122, 339, *345*)

In *en(am)-1 am* double mutants, *en(am)-1* blocks the adaptation of *am* on minimal medium without a source of amino nitrogen (345). The double mutants are inhibited by ammonium and grow adequately only when glutamate is the sole nitrogen source. The *en(am)-1* single mutant grows well on minimal medium, but is unable to use, as the sole nitrogen source, proline (122), methionine, alanine, isoleucine, valine, urocanate, hypoxanthine, uridine, urea, or bovine serum albumin (184). It is relatively resistant to *p*-fluorophenylalanine and ethionine and completely resistant to 0.02 M glycine. These properties cosegregated with *en(am)-1* in all isolates tested (339). Glutamate synthase (GOGAT) is normal (293). The single mutant is scored by using minimal medium with proline as the sole nitrogen source (122) or (better) by using 0.2 mM *p*-fluorophenylalanine (339). Name changed from *i* (inhibitor) (293).

en(am)-2*: enhancer-2 of *am

IIR. Linked near *pe* (*983*).

In *en(am)-2; am* double mutants, *en(am)-2* counteracts leakiness of *am* on minimal medium. *en(am)-2; am* strains grow well on L-alanine (25 mM) or 0.5% casein hydrolysate (983) or on glutamate (5 mM) (293). The single mutant *en(am)-2* without *am* grows normally on minimal medium. Mutant *en(am)-2* lacks glutamate synthase (GOGAT) (see Fig. 19). The double mutant *en(am)-2; am* lacks NADP-glutamate dehydrogenase and GOGAT activities (293). Frequent revertants of the double mutant *en(am)-2; am* on suboptimal medium are attributed to back-mutation at *am* (983). Formerly called *en-am*.

***En(pdx)*: Enhancer of *pdx-1* pigment**

IL. Linked to *mt* (5%), probably to the left (*637*).

En(pdx); pdx-1 double mutants, grown on Vogel medium (1103) or on Westergaard and Mitchell medium (1134) supplemented with ammonium sulfate at 5 g/liter but not at 1 g/liter, excrete yellow pigment into the medium. This property is not shown by either single mutant (637). The addition of a nonlimiting concentration of pyridoxine inhibits production of the pigment on both media. Production of the pigment is also inhibited in heterokaryons between complementing *pdx* alleles. *En* is dominant over *En*$^+$ in heterokaryons between noncomplementing *pdx* alleles (847).

***er*: erupt**

Allelic to *rg-1*, q.v. (382). The symbol *er-1* has been abandoned.

***erg*: ergosterol**

Ergosterol mutants have been detected by resistance to nystatin and other polyene antibiotics. The known mutants are female sterile. Intercrosses for allelism tests can be made, however, by using a heterokaryon as the female parent (419, 699). Ergosterol biosynthesis is illustrated in Fig. 13.

***erg-1*: ergosterol-1**

VR. Between *pk* (2%) and *asn* (9%) (*419*).

The cell membrane is deficient in ergosterol, conferring strong resistance to nystatin and other polyene antibiotics (418, 419). Defective in conversion of fecosterol to episterol (Fig. 13). Lacks fecosterol Δ^8,Δ^7-isomerase (419). Infertile as female. Slow growth, reduced conidiation. The *nys*^{r} mutants of reference 699 are blocked in the same reaction, but *nys*^{r} has not been tested for allelism with *erg-1*.

***erg-2*: ergosterol-2**

VR. Left of *inl* (6%) (*419*).

The cell membrane is deficient in ergosterol, thereby conferring slight resistance to nystatin and other polyene antibiotics (417, 418). Lacks 24(28)dehydroergosterol hydrogenase (terminal step of ergosterol synthesis) (419) (Fig. 13). Poorly fertile as female. Good growth and conidiation.

***erg-3*: ergosterol-3**

IIIR. Linked to *dow* (10 to 14%), probably to the left (*PB*).

The cell membrane is deficient in ergosterol; slightly (2×) increased resistance to nystatin and other polyene antibiotics (417, 418). Biosynthetic lesion not identified (419). Female sterile, but forms tiny protoperithecia. Slow growth, reduced conidiation, uneven production of aerial hyphae.

***erg-4*: ergosterol-4**

IR. Linked to *al-1* (10%) (*PB*).

The cell membrane is deficient in ergosterol, conferring slight resistance to nystatin. Lacks C24 (zymosterol)-methyl transferase. Accumulates zymosterol (Fig. 13). Infertile as female (419). Slow growth, colonial at 34°C, spreading at 25°C (PB).

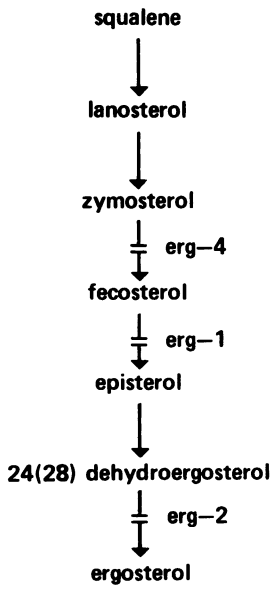


FIG. 13. Probable pathway of sterol biosynthesis, showing sites of gene action (419, 699, and references therein).

***eth-1*: ethionine resistant**

IL. Between *arg-1* and *arg-3* (1%) (816). (*672*)

Resistant to ethionine at 24°C (672). Labile *S*-adenosylmethionine synthetase (515, 547) (see Fig. 17). Resistance attributed to overproduction of methionine (541, 542). Heat sensitive; no growth at 37°C (672). Levels of several enzymes that are normally repressible by methionine are not repressed by methionine in *eth-1*^{R} strains even at the growth-permissive temperature (124, 664). See also reference 965. Both heat sensitivity and ethionine resistance are reparable by high osmotic pressure (664). Called *r-eth-1*.

***exo-1*: exoamylase-1**

I. Linked to *mt* (7%) and *ad-3A* (22%). Stated left of *mt*, but data not given (*325*).

Hyperproduction of β-amylase, α-amylase, glycoamylase, invertase (β-fructofuranosidase), and (to a lesser extent) trehalase (404, 405, 1027). Enzymes secreted abundantly on depletion of exogenous carbon source (404). A polysaccharide is also released (325). Sevenfold increase in conidial enzyme levels. Altered aminosugar content of cell wall (404, 405). Initial allele called SF26, *exo*$_{a}$*-1* (325, 404). Probable second allele found in a strain of *inl* (89601) *a* (194, 325, 1027); allelism evidence in reference 325. With SF26, high amylase and high invertase levels cosegregated in 91 isolates (405). With allele from the 89601 strain, in a mixed-background cross, high amylase and high invertase each act as if due to a single major gene with many modifiers; high amylase and high invertase usually cosegregate and are not correlated with alkaline phosphatase levels (1027). The relation of *exo-1* to gene VI-178, which reverses repression of invertase and trehalase production by mannose, is not known (663). For a linked gene defective in glycoamylase, see *sor(T9)* (called *gla* in reference 50 and called *amy* in reference 325). *exo-1* has not been tested for allelism with *sor(T9)*, but has been stated to be on the opposite side of *mt*. Because *inl* (89601) *a* has been used to obtain mutants by inositol-less death enrichment, *exo-1* may be present but unrecognized in many laboratory stocks.

***f*: fast**

See *su([mi-1])-f*.

***fas*: fatty acid synthesis**

See *cel*.

***fdu-1*: fluorodeoxyuridine resistant-1**

Allelic with *ud-1*, q.v. (126).

***fdu-2*: fluorodeoxyuridine resistant-2**

IV. Right of *cys-4* (2%) (126, *463*).

Resistant to 5-fluorodeoxyuridine, 5-fluorouracil, and 5-fluorouridine. Resistance is partially dominant in heterokaryons. Involved in regulation of *pyr-3*, *udk*, and *ud-1*. (127) Scored by spotting a conidial suspension on medium containing 4×10^{-5} M filter-sterilized 5-fluorodeoxyuridine (463).

***ff*: female fertility**

Infertile as the female, but fully fertile as the male (fertilizing), parent. Besides those listed, 32 more *ff* mutations have been obtained (530), but they are not listed separately because they are unmapped and lack locus numbers. These comprise 28 complementation groups and have been characterized with respect to position of the block in perithecial development, dominance, effects on vegetative growth, supplementability, and independence of mating type (530). These mutants are available from the FGSC. Another symbol, *fs* (female sterile), has been used by other workers for mutant genes causing the same phenotype. Additional mutations, including some that specify impaired female fertility as part of a pleiotropic syndrome and have other names, are listed under *fs*. For additional characteristics, see *fs*.

***ff-1*: female fertility-1**

IIR. Between *aro-1* (5%) and *un-20* (4%) (1052). (*1053*)

Female sterile with no protoperithecia (1052, 1053). Enhanced glycerol utilization; aconidial on liquid glycerol medium. Good conidiation, but reduced aerial hyphae and heavy surface growth on sucrose minimal slants. Growth on glycerol reduces levels of both pyruvate dehydrogenase and dihydrolipoyl transacetylase (211). Effect of carbon source studied (1078). Conveniently scored by failure to produce protoperithecia on small slants of synthetic cross medium (7 days, 25°C). Allele T30 was originally recognized as specifying female sterility and was called *ff-1* (1052, 1053). Allele JC744 was first characterized by glycerol utilization and called *glp*-3 (211).

***ff-2*: female fertility-2**

Unmapped.

No protoperithecia. Normal vegetative morphology (459).

***ff-3*: female fertility-3**

IR. Right of *os-1* (3%) (*193*).

Defective in protoperithecial production. Abnormal morphology. Found in strain T22, which also contains *ty-3* and *ty-4*. Not allelic to *ty-3* or *T*. (193, 460) (Possible allele of *so*?)

***ff-5*: female fertility-5**

IIIR. Between *pro-1* (2%) and *met-8* (1%) (*1052*).

Produces sterile brown protoperithecia without trichogynes and darkens medium (photograph). Vegetatively normal. Closely resembles *ff-6*. Not allelic to *ty-1* or *ty-2*. (1052)

***ff-6*: female fertility-6**

IIIR. Linked near *ty-1* (*459*).

Produces many large black protoperithecia, but no perithecia are formed when used as the female. Black pigment excreted into medium (459).

***fi*: fissure**

IV. Between *cut* (4 to 10%) and *pyr-1* (12 to 19%) (PB). Right of *ace-4* (10 to 17%) (578). (*812*)

Produces exudate in fissures formed under the agar and on the surface. Variable expression; may be difficult to score for some isolates (578, 812). Best scored on minimal synthetic cross medium, 34°C, pH 6 (578).

***fl*: fluffy**

IIR. Between *ace-1* (5 to 11%) and *trp-3* (3%) (816, PB). (*613*)

No macroconidia (609). Highly fertile (612). Used routinely as the female parent in tests for

chromosome rearrangements and for mating type (e.g., reference 801). The *fl* single mutant produces few microconidia when dry; when wetted, sufficient microconidia are produced to have been used in early irradiation and mutation studies (614, 915); large numbers can be obtained under certain conditions; see reference 893. *pe fl* (46, 700) and *fl; dn* (806) double mutants produce abundant microconidia; the latter combination is highly fertile when homozygous. Photograph of microconidial formation (774); see also reference 893. Nuclear numbers in microconidia (46, 64, 478). Wall analysis (207). Immunoelectrophoretic pattern (784). Paradoxical high alcoholic glycolysis on nitrate medium (80). Deficiency of isocitrate lyase on acetate medium; see citations in reference 1088. When *fl A* and *fl a* strains are inoculated separately on crossing medium in plates, a double line of perithecia forms where they meet, similar to that accompanying barrage in *Podospora* (410, 414). *fl* ascospores from certain *fl* × *fl*$^+$ crosses often germinate spontaneously (1127; N. B. Raju, personal communication). Allele C-1835 was called *acon* (717, 812).

***fld*: fluffyoid**

IVR. Left of *his-5* (2%) (991). (*812*)

Resembles fluffy mutants, producing no macroconidia (812).

***flm-1*: flame-1**

Allelic with *os-1*, q.v.

***flm-2*: flame-2**

Allelic with *os-4*, q.v.

***fls*: fluffyish**

IR. Between *nit-1* (5 to 19%) and *al-1* (6 to 19%) (*P. St. Lawrence, cited in references 47, 789, or 812*).

Morphology of young cultures resembles that of fluffy; later conidiates. Suboptimal growth response to methionine (789). Called *un(STL6)*.

***fmf-1*: female and male fertility-1**

I. Between *mt* (2 to 15%) and *cr-1* (2%). Linked to *arg-1* (*531*).

Perithecial development is blocked 15 h after fertilization, before meiosis, when *fmf-1* is present either in the female or male parent. Perithecia attain only 40% normal diameter. Recessive in heterokaryons and can be crossed as one component of a heterokaryon, either as female or as male. Female fertility is also restored in mixed mating type (*fmf-1 A* + *fmf*$^+$ *a*) heterokaryons that are homokaryotic for *tol* (531). Called PBJ6 (527).

***for*: formate**

VIIR. Right of *wc-1* (5%) and *frq* (3%). Left of *dr* (3%) (*812*, 819; J. F. Feldman, personal communication).

Requires formate or formaldehyde. Growth aided slightly if glycine, histidine, or choline is added to formate (446). Will also grow on a mixture of methionine and adenine and suboptimally on adenine alone (446). Lacks cytosolic (but not mitochondrial) serine hydroxymethyltransferase (Fig. 17) (124, 210). Has increased formyl tetrahydrofolate synthetase, methylene tetrahydrofolate dehydrogenase, isocitrate lyase, and glyoxalate aminotransferase activities (209, 210). Use 0.3 mg of formate per ml. Can be autoclaved.

***fpr*: fluorophenylalanine resistant**

Many mutants isolated by resistance to *p*-fluorophenylalanine are actually bradytrophs (very leaky auxotrophs); most require one of many amino acids. The resistance disappears when the required growth factor is added. It is suggested that the resistance is due to transinhibition of amino acid transport caused by increased amino acid pools resulting from deprivation of the required amino acid. (552 and references therein) It is not clear whether any of the mapped *fpr* mutations are of this type.

***fpr-1*: fluorophenylalanine resistant-1**

VR. Linked to *cyh-2* (<1%) (*555*, 1149).

Resistant to *p*-fluorophenylalanine and 4-methyltryptophan. Isolated as resistant to *p*-fluorophenylalanine in the presence of *su(mtr)-1*. Resistance is recessive in heterokaryons; used in mutagenicity test system (626). Suppressed by several *lys* and *arg* auxotrophic genes, which apparently give the double mutant greater sensitivity to *p*-fluorophenylalanine with no increase in uptake (555). Scored on solid medium containing 10 μg of *p*-fluorophenylalanine or 60 μg of 4-methyltryptophan per ml; see reference 550.

***fpr-3*: fluorophenylalanine resistant-3**

IIIR. Linked to *trp-1* (0.35%) and *thi-2* (5%) (*550*).

Resistant to *p*-fluorophenylalanine but not to 4-methyltryptophan. Not resistant in the presence of indole. Amino acid uptake is normal through transport systems I and II, as defined in reference 550. Isolated in a *su(mtr)* strain. Scored on solid medium containing 10 μg of *p*-fluorophenylalanine per ml (550), added before autoclaving.

***fpr-4*: fluorophenylalanine resistant-4**

VR. Right of *inl* (11%) (*550*).

Resistant to *p*-fluorophenylalanine and 4-methyltryptophan. Isolated in a *su(mtr)* strain. Not tested for amino acid uptake. Scored on solid medium containing 10 μg of *p*-fluorophenylalanine or 60 μg of 4-methyltryptophan per ml. (550)

***fpr-5*: fluorophenylalanine resistant-5**

I. Left of *al-2* (25%) (*550*).

Resistant to *p*-fluorophenylalanine, but not to 4-methyltryptophan. Isolated in the wild type. Not tested for amino acid uptake. Scored on solid medium containing 10 μg of *p*-fluorophenylalanine or 60 μg of 4-methyltryptophan per ml. (550)

***fpr-6*: fluorophenylalanine resistant-6**

VIR. Between *pan-2* and *trp-2* (*247*).

Resistant to *p*-fluorophenylalanine. The only allele, UM300, was found in a variant unable to take up arginine to satisfy requirement of *arg* mutations. This blockage is manifest mainly when ammonium is in the medium. Uptake of many other metabolites (amino acids, uridine, sugars) is also affected. Primary defect unknown (247; R. H. Davis, personal communication). Called UM300 or *fpr(UM300)*. Not tested for allelism with *mts* or *mod-5*, which map in the same area and cause increased rather than decreased uptake.

***fr*: frost**

IL. Between *ro-10* (18%) and *un-5* (6%) (798, PB). (*789*)

Delicate branching on agar surface and delicate aerial growth with no conidia (789). Multiple hyphal branching (382). Deficient in glucose-6-phosphate dehydrogenase (as are *col-2* and *bal* mutants) (949, 952). Partially deficient in linolenic acid (115); morphology partially corrected by exogenous linolenic acid (892, 943). Low adenylate cyclase activity and low adenosine 3′,5′-phosphate (943, 950). Used to determine what functions are controlled by adenosine 3′,5′-phosphate (779). Unlike *cr-1*, *fr* is not corrected morphologically by exogenous cyclic nucleotides (892, 951). Scott (943) reported that morphology is corrected by theophylline; Rosenberg and Pall (892) reported no correction by phosphodiesterase inhibitors. Cell wall analysis; photograph (112, 278, 946). Reduced amount of cell wall peptides (1165). Recessive in duplications (808). Female sterile. Both known alleles (B110 and R2499) revert to fr^+.

***frq*: frequency**

VIIR. Between *un-10* (9%) and *for* (3%) (J. F. Feldman, personal communication). Right of *met-9* (9 to 13%). Linked to *oli* (<2%); possibly allelic (282). (*330*)

A series of clustered genes or of multiple alleles resulting in altered periods in the circadian rhythm cycle of conidiation. Identified mutations and their periods (at 25°C without *csp*) are: *frq-1*, 16.5 h; *frq-2*, 19.3 h; *frq-3*, 24.0 h; and *frq-4*, 19.3 h (331); *frq-6*, 19.2 h; *frq-7*, 29.0 h; and *frq-8*, 29.0 h (wild type, 21.5 h) (329, 377). Strains carrying allele UV-III-9 show erratic periodicity. Temperature compensation and interactions with other loci have been described; dominance is incomplete; growth rates are normal (326, 375–377). Scoring is accomplished by zonation in growth tubes or plates, using strains that carry *bd* and preferably *csp-1* or *csp-2*. Presence of *csp* shortened period length about 1 h in the one strain tested (279). Period-altering mutations elsewhere in the genome are given different names (e.g., *prd* and *chr*). The symbol *frq* is reserved for this locus or region (329). For review of circadian mutants, see references 326 and 328.

***frq-5*: frequency-5**

Changed to *prd-1*, q.v.

***fs*: female sterile**

Infertile as the female but fully fertile as the male (fertilizing) parent. No or few functional perithecia are produced. Another symbol, *ff* (female fertility, q.v.), has been used by other

workers for mutants having the same phenotype. Female fertility is also impaired or absent in some mutants that were named for other traits, e.g., *cyt-1, cyt-2, erg, fr, glp-3, gul-3, gul-4, leu-1, R, ro, sk, so, ssu, ty-1, ty-2, var-1*. Numerous additional female-sterile mutants have been isolated (91, 253, 491, 530, and references cited therein), but the genes have not been mapped and/or tested for allelism with the mutations listed here. Many of these also affect vegetative morphology or growth rate. Tests on *ff* mutants, q.v., show that different mutants are blocked at different points in perithecial development (530). Female sterility has no genetic or functional relationship to mating type (530). Crosses homozygous for any *fs* gene listed can be made, and progeny can be obtained, by using a heterokaryon of marked *fs* and fs^+ strains as the female parent (732; O. M. Mylyk, personal communication); the same is true for most *ff* genes (530).

The term "sterile" has been used in different ways: for situations in which no protoperithecia are formed, or in which perithecium development is blocked before ascospore formation, or even in which ascospores are produced that are inviable. The term "barren" has been proposed specifically for crosses in which perithecia develop but few or no ascospores are produced (860). See also *ff* and *pp*.

fs-1: female sterile-1

I or II. Linked to *T(I;II)4637*. Unlinked to *mt* (*732*).

Perithecia are absent or infrequent when used as the female (protoperithecial) parent. Fertile as the male. Some strains produce occasional perithecia and ascospores. Vegetative growth is somewhat stringy, slightly slower, and paler than that of the wild type. Recessive in heterokaryons. Complements *fs-2, -3, -4, -5, -6*, and *-n*. (732; O. M. Mylyk, personal communication). Shown nonallelic with *fs-2, -3, -5*, and *-n* in crosses (732).

fs-2: female sterile-2

Tentatively II; probable loose linkage to *fs-1* (*732*) and *cot-5* (14/48) (PB).

No perithecia when used as the female. Fertile as the male. Abnormal morphology, somewhat colonial. Grows at 25°C, but not at 34°C. Recessive in heterokaryons. Complements *fs-1, -3, -4, -5, -6*, and *-n*. (O. M. Mylyk, personal communication)

fs-3: female sterile-3

IL. Left of *mt* (16%) (*732*).

No perithecia when used as the female. Fertile as the male. Vegetative growth is slightly slower and paler than that of the wild type. Recessive in heterokaryons. Complements *fs-1, -2, -4, -5, -6*, and *-n* (732; O. M. Mylyk, personal communication).

fs-4: female sterile-4

I? Linked mating type (22%) (*732*). May be inseparable from a chromosome rearrangement (O. M. Mylyk, personal communication).

No perithecia when used as the female. Fertile as the male. Complements *fs-1, -2, -3, -5, -6*, and *-n*. Vegetative growth is slightly slower and paler than that of the wild type. Recessive in heterokaryons. (732; O. M. Mylyk, personal communication)

fs-5: female sterile-5

I or II. Probable loose linkage with *fs-1* (*732*).

Perithecia absent or rare when used as the female; some strains produce occasional perithecia and ascospores. Fertile as the male. Slow growth, mostly aerial near the surface of the agar. Cultures turn brown with age (732). Complements *fs-1, -2, -3, -4, -6*, and *-n*. (O. M. Mylyk, personal communication)

fs-6: female sterile-6

I or II. Linked to *T(I;II)4637*. Unlinked to *mt* (*732*).

No perithecia when used as the female. Fertile as the male. Vegetative growth is slightly slower and paler than that of the wild type. Recessive in heterokaryons. Complements *fs-1, -2, -3, -4, -5*, and *-n* (732; O. M. Mylyk, personal communication).

fs-n: female sterile-n

I. Linked to *mt* (35 to 45%) and to *T(I;II)4637* (*732*).

No perithecia when used as the female. Fertile as the male. Vegetative growth is slightly slower and paler than that of the wild type. Recessive in heterokaryons. Three complex ascus segregations in 51 asci suggest two closely linked genes.

If so, they must not complement each other, although they complement *fs-1, -2, -3, -4, -5*, and *-6*. (732; O. M. Mylyk, personal communication)

***Fsp-1*: Four-spore-1**

IIR. Right of *pe* (4%) (*D. D. Perkins, unpublished data*).

Some of the asci contain four large ascospores rather than the normal eight. In these asci, ascospores are formed at the four-nucleate stage after meiosis II. Dominant, with variable penetrance depending on genetic background. Ascospores from four-spored asci produce homokaryotic cultures. Rarely, three-spored or two-spored asci are formed, and these include some heterokaryotic ascospores. One postmeiotic mitosis is omitted in the four- and three-spored asci, and two divisions are omitted in the two-spored asci. Vegetative morphology is normal. (856) Used in the study of *Sk* (Spore killer) (857). The cytological basis of *Fsp-1* is distinct from that in *N. tetrasperma*. For a description of a dominant eight-spored mutant of *N. tetrasperma* see reference 129.

***Fsp-2*: Four-spore-2**

IR. Right of *nic-2* (6%) (N. B. Raju, personal communication). (*253*)

In crosses heterozygous for *Fsp-2*, nearly all asci are four-spored at 16°C and eight-spored at 25°C (253). Crosses homozygous for *Fsp-2* make four-spored asci at both temperatures. The cytological basis is similar to that for *Fsp-1* (N. B. Raju, personal communication).

***fz*: fuzzy**

Unmapped.

Abnormal morphology; one component of the combination of mutant genes that results in the cell-wall-less "slime" phenotype (321).

***G*: gulliver**

See *gul-1*.

***gap*: gap**

IL. Between *mt* (6%) and the centromere (4%) (*610*).

Conidia in a few scattered clusters on long nonconidial hyphae. Photograph (610). (Stock lost.)

***gla*: glucoamylase**

See *sor(T9)*.

***glm*: glutamine**

Changed to *gln*.

***gln-1*: glutamine-1**

VR. Linked to *inl* (2%, probably to the right) (*869*).

Requires glutamine (869). Probably the glutamine synthetase structural gene (229, 912) (see Fig. 19). Mutants have altered enzyme (912). Sensitive to chlorate on both ammonium and glutamate; resistant to chlorate on glutamine (292). NADPH-nitrate reductase, NAD(P)H-nitrite reductase, and uricase are freed from repression by ammonium or glutamate but not glutamine in the *gln-1a* mutant (291, 294, 836, 1118). Allele *gln-1b* is more derepressed than allele R1015 (called *gln-1a*) (292, 837). For interaction with *am*, see reference 503. Formerly called *glm* (869).

***glp*: glycerol phosphate**

Symbol replaces *gly* for mutants with altered ability to use glycerol as a carbon source. Scored on slants of minimal synthetic cross medium (1134) with 2% glycerol versus 2% sucrose as the carbon source (1102). Poor growth of the wild type on glycerol medium is markedly improved by the addition of 0.5% L-asparagine and 100 μg of ascorbic acid per ml (189); this might facilitate testing. For diagram of pathways of glycerol utilization in various organisms, see reference 1078 or 1102.

***glp-1*: glycerol phosphate-1**

IR. Linked to *ad-9* (2%) and *nit-1* (11%); probably between them (466, *763*).

Unable to use glycerol as the sole carbon source (763). Can use dihydroxyacetone or glyceraldehyde (261). Probably regulatory. Deficient in inducible glycerol kinase under normal conditions (466, 764); wild-type levels of normal enzyme are induced by cold or by deoxyribose in strains carrying some, but not all, alleles (261, 466); glycerol transport is normal (261). Fine-structure map (262). Called *gly* and *gly-u*.

***glp-2*: glycerol phosphate-2**

IIR. Right of *T(ALS176)*; hence, of *arg-5* (8%).

Left of *T(NM177)*; hence, of *pe* (7%). Linked to *aro-3* (3%) and *ff-1* (*glp-3*) (15%) (*263*).

Unable to use glycerol, dihydroxyacetone, or glyceraldehyde as the sole carbon source (261, 263). Lacks both mitochondrial and cytosolic flavin-linked glycerol-3-phosphate dehydrogenase (263). Three independent isolates all have altered ropy-like vegetative morphology, but are female fertile, unlike most *ro* mutants (212, 263). The report of complementation groups at this locus (214) is in error (J. B. Courtright, personal communication). Fine-structure map (262). Called *gly-2*.

glp-3: glycerol phosphate-3

Allelic with *ff-1*, q.v.

glp-4: glycerol phosphate-4

VI. Right of *ad-1* (0 to 2%) and *ylo-1* (1 to 6%). Left of *rib-1* (3 to 4%) and *pan-2* (4 to 6%) (*1102*).

Unable to use glycerol as the sole carbon source (1102). Uses dihydroxyacetone or glyceraldehyde (261). Lacks both inducible and constitutive glycerol kinase (1102), but there is some doubt that these are two different enzymes (J. B. Courtright, personal communication, based on reference 262). Revertant with altered kinetic properties (262). Allele G660 originated in *N. tetrasperma* and was introgressed into *N. crassa* (77, 1102). Fine-structure map (262).

glp-5: glycerol phosphate-5

I. Left of *cr-1* (15%) (*1102*).

Unable to use glycerol as the carbon source. Lacks glyceraldehyde kinase (1102), but the significance of this is uncertain because of findings reported in reference 1078. Allele M1051 originated in *N. tetrasperma* and was introgressed into *N. crassa* (77, 1102).

glp-6: glycerol phosphate-6

V. Left of *inl* (30%) (*840*).

Deficient in NAD-linked glycerol-3-phosphate dehydrogenase. Called 42-94 (840; H. B. Howe, Jr., personal communication).

glt: glycyl-leucyl-tyrosine resistant

Unmapped.

Unable to transport oligopeptides necessary to support growth of specified amino acid auxotrophs (1155). Has only 10% of the wild-type uptake rate (1154). (Oligopeptide uptake system transports tri-, tetra-, and pentapeptides, but not di- or higher than pentapeptides.) Obtained, using *tys*, by selecting mutants resistant to glycyl-L-leucyl-L-tyrosine but still sensitive to tyrosine (1155). See reference 1151 for a review of peptide uptake.

gluc-1: β-glucosidase-1

IIIR. Linked to *dow* (10%) (*B. M. Eberhart, personal communication*).

Activity of the thermostable aryl-β-glucosidase reduced to 10% of the wild type (300) by one allele, and to <1% in a second-step mutant then called *gluc-2*, which showed 0/200 recombination with the original mutation and is probably allelic (299). Low activity is dominant in heterokaryons (630). Selected by the *p*-nitrophenyl glucoside staining reaction (297). Scored by breakdown of the β-glucoside esculin (0.01%) as measured by fluorescence at pH 5.5 (300), or by precipitation of ferric ammonium citrate (0.1%) by esculetin (2 days, 25°C) (B. M. Eberhart, personal communication).

gluc-2

See gluc-1.

gly or _gly-u_: glycerol utilization

Changed to *glp*.

gpi-1: glucosephosphate isomerase

IVR. Linked to *ad-6* (10%) (*711*).

Lacks glucosephosphate isomerase (phosphohexoisomerase). Grows slowly and colonially on glucose or sucrose. Unable to use fructose, but growth on glucose is stimulated by added fructose. Growth is enhanced in double mutants with either *sor(T9)* or *pp*. Allele *T*66M37 was originally called *gpi-2* (711).

gpi-2

See *gpi-1*.

gran: granular

VR. Linked to *pl* (0/75); between *pab-2* (1 to 8%) and *his-6* (8 to 27%) (816, PB). (*812*)

Delicate granular conidiation, with conidia adherent rather than powdery (812). Sparsely branched hyphae (382). Morphologically distinct from *pl* mutants. Reduced amount of cell wall peptides (1165).

***grey*: grey**

IVR. Linked to *cot-1* (4%) (*499*).

Produces grey conidia (microconidia?) in the presence of *cr-1* (499). Attempts to obtain grey progeny have been unsuccessful (PB).

***gs(3)*: gamma sensitive**

Not mapped.

Sensitive to ionizing radiation, but not to UV. Normal UV-induced mutation. Evidence not given for nonallelism with *gs(6)* and *gs(20)* (662).

***gs(6)*: gamma sensitive**

Not mapped. (Perhaps VI.)

Sensitive to ionizing radiation and UV. Decreased UV-induced mutation. Evidence not given for nonallelism with *gs(3)* and *gs(20)* (662).

***gs(20)*: gamma sensitive**

Not mapped.

Sensitive to ionizing radiation but not to UV. Decreased UV-induced mutation (662).

***gsp*: giant spore**

IL. Left of mating type (10%) (1008). (*589*)

Some asci contain a single giant ascospore; others have two very large ascospores or four double-size ascospores; some contain eight ascospores. Proportions of these types vary on different crossing media. Vegetative growth is weak with normal morphology. The giant ascospores have multiple germination pores. The mutant ascus phenotype is recessive (589, 1008).

***gua-1*: guanine-1**

I. Linked to *arg-3* (8%) (*1171*); probably between *his-2* (3%) and *cr-1* (3%) (PB).

Requires guanine. Inhibited (competitively) by adenine and by complex complete medium. Adapts phenotypically after several days and grows up on minimal or complete medium, but retains the requirement on subculture. Adenine prevents or decreases adaptation. Guanosine is preferred to guanine as a supplement because of greater solubility. Best scored at 2 and 3 days on slants of minimal medium plus 1 mg of adenine versus minimal medium plus 0.2 mg of guanosine per ml. (1171) Deficient in inosine 5'-monophosphate dehydrogenase (10% of the wild-type level with allele OY301) (405a) (Fig. 8).

***gua-2*: guanine-2**

IVR. Linked to *cot-1* (5%) (*405a*).

Requires guanine. No inosine monophosphate dehydrogenase activity. Does not adapt to grow on minimal medium (405a) (Fig. 8).

***gul*: gulliver**

Name given (875) to suppressors of *cot-1*, which produce large colonies at restrictive temperatures at which unsuppressed *cot-1* mutants make tiny colonies. Scorable in the presence of *cot-1* at 34°C, 2 days after transfer of small inocula to solid medium.

***gul-1*: gulliver-1**

VR. Between *am-1* (<0.01%; <1%) and *ace-5* (<1%) (577, 998). (*875*)

Modifier of the colony size of the mutant *cot-1* at restrictive temperatures (875, 1068). *cot-1; gul-1* strains take 60 h to reach the stationary phase (32 to 34°C) compared with 12 h for *cot-1 gul-1*$^+$ strains (1068). Female fertile with viable ascospores. Able to make heterokaryons. Of 36 independent *gul* mutations, 25 were *gul-1* alleles (1068). Recombination within *gul-1* is unaffected by *rec-3*, which acts on the nearby *am-1* locus (998). Formerly called *G* (875).

***gul-2*: gulliver-2**

Not mapped.

Phenotype similar to that of *gul-1* strains (1068).

***gul-3*: gulliver-3**

IVR. Linked to *cot-1* (10%) and *pyr-2* (7%) (*1068*).

Modifier of the colony size of the mutant *cot-1*

at restrictive temperatures. Female sterile. *gul*$^-$ ascospores are black but inviable. Occasional *gul*$^-$ progeny arise from *gul*$^+$/*gul*$^-$ pseudowild disomic ascospores. Unable to make heterokaryons (1068).

***gul-4*: gulliver-4**

VII. Linked to *nic-3* (17%) (*1068*).

Resembles *gul-3* (1068).

***gul-5*: gulliver-5**

VI. Linked to *trp-2* (10%) (*1068*).

Modifier of the colony size of the mutant *cot-1* at restrictive temperatures. Female fertile. *gul*$^-$ ascospores are black but inviable. (1068)

***gul-6*: gulliver-6**

Not mapped. Unlinked to *cot-1* (IVR), *inl* (VR), *nic-3* (VIIL), *gul-5* (VI), or *gul-2* (1068).

Said to resemble *gul-3* (1068), but ascospore ripening and recovery from ascospores have been found to be good (PB).

***has*: hydroxamic acid sensitive**

Not mapped. Unlinked to *azs* (311).

Lacks salicyl hydroxamic acid-sensitive respiratory pathway; cannot produce the hydroxamate-sensitive respiratory pathway when grown in the presence of chloramphenicol. Grows slowly in the presence of antimycin A (311). Double mutant *has; azs* is unable to grow in the presence of antimycin A; the wild type and the *has*$^+$; *azs* single mutant are able to grow well on antimycin A. The double mutant *has; azs* (called ANT-1: antimycin sensitive) was used to obtain oligomycin-resistant (312) and succinate dehydrogenase-deficient (307) mutants. From strain ANT-1, called alx-1 (308).

***het*: heterokaryon incompatibility**

If two strains carry different alleles at one or more *het* loci, they are unable to form stable heterokaryons (378, 379). Protoplasmic killing occurs after fusion of unlike hyphae (384) or after microinjection of cytoplasm or extracts into unlike strains (1145). Photographs (384). When duplications (partial diploids) are heterozygous for *het* alleles, growth is inhibited and highly abnormal (761, 803). The incompatibility due to *het* genes is strictly vegetative; it does not reduce fertility. Ten *het* loci have been identified, and various others are inferred to exist (729). *het-c*, *-d*, *-e*, and *-i* were first defined by heterokaryon tests. The remainder (*het-5* through *-10*) were detected by using duplications. The mating type alleles *A* and *a* also act as *het* genes in *N. crassa* (66, 384, 761, 830), although some slow heterokaryotic growth may occur (422). Microinjection experiments implicate proteins in the killing reaction (1138, 1145). Review and literature citations: 232, 803. Lindegren and Rockefeller wild types are *het-C*, *het-D*, and *het-E* (1144; J. F. Wilson, personal communication). St. Lawrence 74A and Oak Ridge wild types are *het-C*, *het-d*, and *het-e* (1144). St. Lawrence 74A and Oak Ridge OR8-1a are *het-i* (831). Differences at *het* loci are very common in natural populations (730).

***het-c*: heterokaryon incompatibility-c**

IIL. Left of *pyr-4* (1%). Not included in duplications from *T(AR18)* or *T(P2869)*; hence, right of *cys-3* and *het-6* (729, 808, PB). (*378*)

Stable heterokaryons are not formed by strains that are *het-C* + *het-c* (378, 379); strains carrying *het-C*/*het-c* duplications show inhibited "brown flat" morphology, spreading to cover the slant but not conidiating (794, 803). Putative multiple alleles, suggested by abnormal duplication phenotypes when chromosomes from various natural sources were tested, may be due instead to additional *het* loci in the segment tested (729, 795). Photograph: see Fig. 3 of reference 729.

***het-d*: heterokaryon incompatibility-d**

IIR. Right of *fl* (25%) (*378*). Included in duplications from *T(ALS176)* (805).

Stable heterokaryons are not formed by *het-D* + *het-d* strains (378, 379); strains carrying *het-D*/*het-d* duplications show inhibited spreading growth on slants, with fine subsurface hyphae and no conidia. These are distinguishable from strains carrying *het-C*/*het-c* duplications, which have a coarser texture (805).

***het-e*: heterokaryon incompatibility-e**

VIIL. Left of *nic-3* (28%) (*1144*). Included in duplications from *T(T54M50)* (803).

Killing reaction after fusion of *het-E* and *het-e* is more rapid and severe, and growth inhibition of strains carrying *het-E*/*het-e* duplications is

more severe than for strains carrying incompatible combinations of *het-c* or of mating type alleles (803, 1144). Photograph of heterozygous duplication colony (729).

***het-i*: heterokaryon incompatibility-i**

I or II. Linked to *T(IR;IIR)4637 al-1* (*831*).

Recognized by cessation of growth of forced heterokaryons under certain conditions. *het-I* nuclei eliminate *het-i* if initial frequency exceeds 30% *het-I*. When more than 80% are *het-i*, growth of a forced heterokaryon can continue without a change of ratio. Called *I* and *i*. (831)

***het-5*: heterokaryon incompatibility-5**

IR. Between *T(NM103)* and the tip; hence, right of *thi-1* (*729*).

Vegetative incompatibility recognized by inhibited duplications and subsequently confirmed by heterokaryon tests (729, 730).

***het-6*: heterokaryon incompatibility-6**

IIL. Included in duplications from *T(AR18)*; hence, right of *cys-3* and left of *het-c* and *pyr-4* (*729*).

Vegetative incompatibility recognized by inhibited duplications from translocations *AR18*, *P2869*, and *NM149*. No heterokaryon tests (729).

***het-7*: heterokaryon incompatibility-7**

IIR. Between *T(D305)* and the tip; hence, right of *ro-2* (*729*).

Vegetative incompatibility recognized by inhibited duplications from *T(D305)*. No heterokaryon tests. (729)

***het-8*: heterokaryon incompatibility-8**

VIL. Between *chol-2* (19%) and *ad-8* (12%) (*729*, 730).

Vegetative incompatibility recognized by inhibited duplications from *T(T39M777)* and subsequently confirmed by heterokaryon tests (729, 730). Photograph of heterozygous duplication colony (729).

***het-9*: heterokaryon incompatibility-9**

VIR. Between *T(AR209)* and the right tip (*729*).

Vegetative incompatibility recognized by inhibited duplications from *T(AR209)*. Photograph of heterozygous duplication colony. (729)

***het-10*: heterokaryon incompatibility-10**

VIIR. Between *T(5936)* and the tip; hence, right of *dr* (*729*).

Recognized on the basis of inhibited duplications from *T(5936)*. Photograph of heterozygous duplication colony (729).

***hgu-4*: histidylglycine uptake-4**

VR. Between *cyh-2* (7%) and *ure-2* (10%) (1149). (*1153*)

Cannot use L-histidylglycine to support growth of *his-6* mutants (1153). Reduced approximately 50% in transport of most amino acids tested. Resistant to many amino acid analogs (1149).

***his*: histidine**

Most histidine auxotrophs are inhibited by complex media or by certain combinations of amino acids with which histidine does not compete effectively for permeases of the basic, neutral, and general amino acid transport systems. A histidine mutant can grow on minimal medium plus histidine in the presence of either a basic amino acid or a competing neutral amino acid, but not in the presence of both (434, 628, 646). Histidine mutants were not obtained in early mutant hunts in which complex media were used, but were recovered on histidine-supplemented minimal medium (434, 595). For general studies, see references 162, 434, and 1123. For details of histidine biosynthesis, see Fig. 14. Enzymes of histidine biosynthesis are derepressed coordinately with those of tryptophan, arginine, and lysine (137, 1131); reviewed in reference 642. See *cpc-1*. Called *hist*.

***his-1*: histidine-1**

VR. Right of *ure-1* (1%). Left of *pho-2* (3%), *al-3*, and *inl* (1 to 10%) (397, 570, 578, 1036). (*434*)

Requires histidine (434). Accumulates imidazole glycerol phosphate. Lacks imidazole glycer-

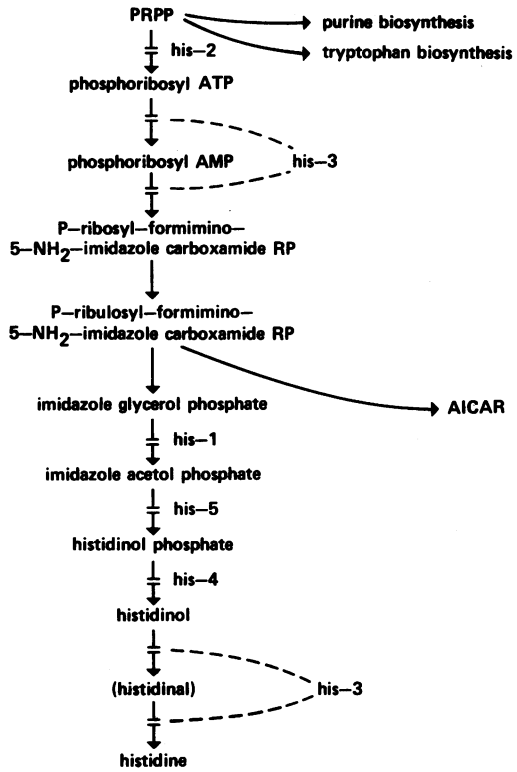


FIG. 14. Biosynthetic pathway of histidine, showing the sites of gene action (16, 24, 25, 162, 673, 1123). ATP, Adenosine 5'-triphosphate; AMP, adenosine 5'-monophosphate; PRPP, 5-phosphoribosyl pyrophosphate. AICAR (5'-phosphoribosyl-5-aminoimidazole-4-carboxamide ribosylphosphate) is an intermediate in purine synthesis. See Fig. 8. For relations between histidine and purine synthesis, see reference 786.

ol phosphate dehydrase (24, 25) (Fig. 14). Intralocus complementation (162). Recombination between *his-1* alleles is controlled by *rec-1* (172, 520, 1070). Initial *his-1* allele called C84.

his-2: histidine-2

IR. Right of *T(AR190)* and *un-2* (<1%). Left of the *T(AR173)* right breakpoint and of *nuc-1* (<1%) (172, 670, 808). (*434*)

Requires histidine (434). Affects adenosine 5'-triphosphate phosphoribosylpyrophosphate pyrophosphorylase (16) (Fig. 14). Intralocus complementation (162). Recombination between *his-2* alleles is controlled by *rec-3* (173); it is not affected by *rec-1* (172). Initial *his-2* allele called C94.

his-3: histidine-3

IR. Right of *met-10* (R. L. Metzenberg, personal communication). Left of *cog* (1 to 3%) (172, 174), *ure-4* (1%) (78), and *ad-3A* (1%) (271). (*434*)

Requires histidine (434). Complex gene coding for histidinol dehydrogenase, phosphoribosyl-adenosine 5'-triphosphate-pyrophosphohydrolase, and phosphoribosyl-adenosine 5'-monophosphate-cyclohydrolase (16, 673) (Fig. 14). All three activities appear to be catalyzed by a single protein (673). Strains carrying different individual alleles may lack only the early reaction(s) or only histidinol dehydrogenase, or both. Those that lack only histidinol dehydrogenase accumulate histidinol (16, 162, 1123). Mutants produce cross-reacting material (220). Used to study intralocus complementation and recombination (15, 16, 27, 162, 164, 171, 172, 1121, 1122, 1124). Intralocus recombination is regulated by *cog* and by *rec-2* (27, 171); it is not affected by *rec-1* (172). Translocation *T(IR;VII)TM429*, with one breakpoint in *his-3*, has been used to show that *cog* is *cis*-acting (171). Initial alleles: C140 and T1710 (= C1710).

his-4: histidine-4

IVR. Between *cot-1* (1 to 4%) and *met-5* (4%) (812). (*434*)

Requires histidine (434). Accumulates L-histidinol phosphate. Lacks histidinol phosphate phosphatase (24, 25) (Fig. 14). Allele P143h is heat sensitive (his^+ at 25°C); C141 is not (815). Both are leaky (726).

his-5: histidine-5

IVR. Between *pyr-3* (1%) and *trp-4* (3 to 7%) (991). (*162*)

Requires histidine. Accumulates imidazole acetol phosphate and some imidazole glycerol phosphate. Evidently lacks imidazole acetol phosphate transaminase (16, 162, 1123) (Fig. 14). Intralocus complementation (162) and recombination (172).

his-6: histidine-6

VR. Right of *un-9* (6%) and *pyr-6* (6 to 18%). No recombination with terminal translocation *T(NM149)* (793, 808, *816*, 818, PB). Report of IV linkage (646) not confirmed.

Requires histidine. Blocked before imidazole glycerol phosphate (162, 1123) (Fig. 14). No intralocus complementation (95 alleles) (162). Intralocus recombination (172).

***his-7*: histidine-7**

IIIR. Between *leu-1* (8 to 20%) and *thi-2* (1 to 2%) (219, 1052, PB). (*162, 1123*)

Requires histidine. Blocked before imidazole glycerol phosphate (162, 1123) (Fig. 14). Intralocus recombination (172).

***hist*: histidine**

Changed to *his*.

Histidine sensitive

Many mutagen-sensitive mutants are histidine sensitive (see *mus, uvs*, and *mei*). Several other histidine sensitives which are unmapped are not sensitive to the few mutagens tested (see, e.g., reference 255); these are not listed pending evidence for nonallelism with known loci.

***hlp-1*: histidinol permeability-1**

VIIR. Between *sfo* (1 to 9%) and *nt* (28 to 37%). Left of *hlp-2* (8 to 25%) (*458*).

Enables strains carrying a *his-3* allele to use L-histidinol. This is proposed to be due to increased uptake through basic L-amino acid transport (system III as defined in reference 778). The *hlp-1* mutation confers increased sensitivity of *lys* and *arg* mutants to inhibition by arginine and lysine, respectively. (458)

***hlp-2*: histidinol permeability-2**

VIIR. Between *sfo* (3 to 7%) and *nt* (29%). Right of *hlp-1* (8 to 25%) (*458*).

Enables a *his-3* mutant to grow on L-histidinol. Growth of the double mutant *his-3; hlp-2* on histidine is inhibited by methionine, isoleucine, valine, and asparagine (458).

***hom*: homoserine**

IR. Right of *arg-6* (1%), *Tp(T54M94)*, and *al-2* (2 to 7%). Between the breakpoints of *T(STL76)* and *T(4637)*; hence, left of *al-1* (<1%). Linked to *cnr* (1%), probably to the left (789, 812). (*1061, 1063*)

Uses homoserine, or methionine plus threonine (1063). Affects aspartate β-semialdehyde dehydrogenase (518) (see Fig. 17). Inhibited on complex complete medium and by methionine and other amino acids (1063); supplemented minimal medium should therefore be used. Symbol changed from *hs*.

***hs*: homoserine**

Changed to *hom*.

***i*: (inhibitor)**

Changed to *en(am)-1*.

***i*: (heterokaryon incompatibility-*i*)**

See *het-i*.

***i*: (intensifier)**

Used for unmapped intensifier of carotenoid pigment (982).

***Iasc*: Indurated ascus**

VR. (*A. M. Srb, personal communication*)

Ascus wall hardens and darkens, so that the entire ascus resembles a giant ascospore. Pores are formed and striations appear. Asci are germinable. Mutant ascus phenotype is dominant with variable expression. Some asci are normal. Vegetative growth is weak (1008). Resembles the indurated ascus phenotype described in *N. tetrasperma* (285).

***ile-1*: isoleucine-1**

VII. Between *ars* (1%) and *wc-1* (3%). Probably right of *met-7* (<1 to 2%) (666, PB). (*812*)

Uses isoleucine, α-amino-*n*-butyric acid, threonine (1061), or canavanine (63). Affects threonine dehydratase (549, 552) (synonym: threonine deaminase [549]) (see Fig. 15). Leaky on minimal medium: treacherous to score with large inocula. Tests should be read early (24 h, 34°C). Moderate inhibition by methionine (1061). Selectable as tiny germlings from ascospores germinated on minimal medium (666). Name changed from *thr-1* (549).

***ilv*: isoleucine plus valine**

Three loci specify enzymes that catalyze corresponding steps in the parallel biosynthetic pathways of isoleucine and valine (Fig. 15). These enzymes are located in the mitochondria (79, 597 and references therein) and may indirectly affect electron transport (79). The en-

zymes may be in an aggregate; for a review, see reference 237. The requirements are for both amino acids. A ratio of 20–30% isoleucine to 80–70% valine is optimal (99). At least some *ilv* mutants are inhibited by norleucine, norvaline, phenylalanine (99), or tryptophan (J. F. Leslie, personal communication).

Enzyme production in response to end-product-derived signals depends on the *leu-3*$^+$ product and α-isopropylmalate. In *leu-3*$^+$ strains, threonine deaminase production is repressed as a function of available isoleucine, acetohydroxyacid synthetase is repressed as a function of valine, and isomeroreductase and dihydroxyacid dehydratase are repressed as a function of isoleucine and leucine. In the absence of effective *leu-3* product, α-IPM, or both, enzyme production is repressed even under severe end product limitation. (771) Formerly called *iv*.

ilv-1: isoleucine plus valine-1

VR. Between *per-1* (4%) and *lys-2* (4 to 7%) (3, 14, 489). Left of *ilv-2* (<1 to 9% prototrophs; some crosses give anomalous high frequencies [557]). (*482*)

Requires both isoleucine and valine, or corresponding keto acids (557, 1097, 1112). Affects dihydroxy acid dehydratase (22, 727) (Fig. 15). Most alleles are leaky (557). Leucine has a sparing effect on valine requirement (99). Called *iv-1*; groups 2 and 3.

ilv-2: isoleucine plus valine-2

VR. Closely linked to the right of *ilv-1* (557). (*482*)

Requires both isoleucine and valine. Affects α-keto-β-hydroxylacyl reductoisomerase (1112) (Fig. 15). Known alleles are not leaky (557). Allele T313 is heat sensitive (557). Called *iv-2*; group 1.

ilv-3: isoleucine plus valine-3

IVR. Linked to *met-2* (0/129) (PB). Between *leu-2* (4%) and *ad-6* (9%) (*579*).

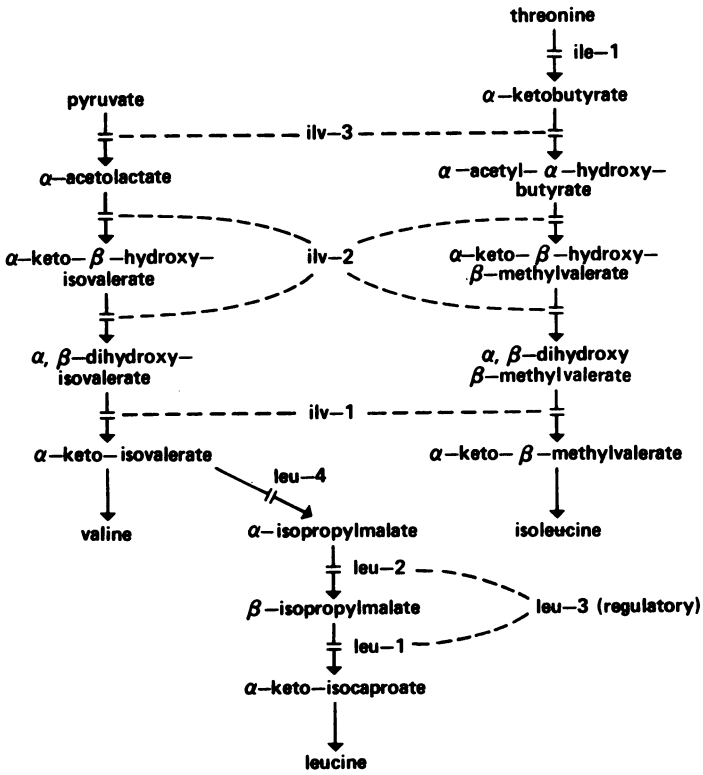


FIG. 15. Biosynthetic pathways of isoleucine, valine, and leucine, showing sites of gene action (22, 136, 426, 549, 727, 854, 1112). Isoleucine and valine are synthesized along parallel pathways catalyzed by common enzymes. The leucine precursors α- and β-isopropylmalate were formerly called β-OH-β-carboxyisocaproate and α-OH-β-carboxyisocaproate, respectively.

Requires both isoleucine and valine. Accumulates pyruvate. Very low acetohydroxy acid synthetase activity (136) (Fig. 15). Alleles recombine and complement (579, 1112). Markedly inhibited by methionine (PB). Called *iv-3*; group 4.

***In( ):* inversion**

Inversions can be used for mapping genes by duplication coverage (analogous to deletion mapping). They are listed here only if they have contributed critical information on gene sequences or locations of tips or centromeres. An inversion that includes the centromere (i.e., that is pericentric) and has one breakpoint at a tip is equivalent to a translocation in which a distal segment of one arm is transferred to the tip of the other arm. Such an inversion of linkage group I is symbolized *In(IL → IR)* or *In(IR → IL)*. When such an inversion is crossed by normal sequence, recombination produces meiotic products that are duplicated for the transferred segment. Crosses between two overlapping inversions produce recombinant meiotic products that are duplicated for segments between the displaced breakpoints. For theory, diagrams, and methods, see reference 808.

In(NM176)*: terminal pericentric inversion *In(IL → IR)NM176

A distal segment of IL is interchanged with the IR tip. Viable duplication progeny from *In* × normal sequence contain two copies of the segment, which includes *cys-5*, *ser-3*, and markers distal to them, but does not include *un-3* or *mt*. When *In(NM176)* is crossed with overlapping inversion *In(OY323)*, an additional class of duplication progeny are produced that contain two copies of the intervals between the breakpoints of the two inversions; see *In(OY323)* (57, 808, 1093).

In(OY323)*: pericentric inversion *In(IL;IR)OY323

A long segment of I is inverted, which includes the centromere. When *In(OY323)* is crossed with overlapping inversion *In(NM176)*, viable duplication progeny are produced that contain two copies of the intervals between the breakpoints of the two inversions. This duplicated segment includes *ace-3* and *nic-1* but not *lys-3* in IR, and *leu-3* but not *nit-2* in IL. (57)

In(H4250)*: terminal pericentric inversion *In(IL → IR)H4250

A distal segment of IL is interchanged with the IR tip. Viable duplication progeny from *In* × normal sequence contain two copies of the segment, which includes *suc* and markers distal to it but does not include *phe-1* (761, 808).

***inl*: inositol**

VR. Between *pho-3* (3 to 4%) and *pab-1* (1 to 10%). Right of *al-3* (362, 397, 1036). (*482*)

Requires inositol (65). Lacks D-myoinositol-1-phosphatase (1142). Lack of glucocycloaldolase found by Piña and Tatum (826) is attributed by Williams (1142) to drastic repression of glucocycloaldolase by the concentration of inositol used for growth. Growth is colonial on low levels of inositol (367). Tends to extrude dark pigment into the medium when grown on suboptimal inositol. Composition of phospholipids and cell walls is abnormal on limiting inositol (367, 439, 440, 501). Inhibited by hexachlorocyclohexane (366, 457, 931). Conidia are subject to death by unbalanced growth on minimal medium (1028, 1033), a property exploited for mutant enrichment ("inositol-less death") (606, 647) because double mutants are at a selective advantage. Heat-sensitive allele 83201 is especially useful for mutant enrichment (832, 1043). Used in the first experiments reporting transformation of *Neurospora* by *N. crassa* DNA (677, 679) and reported to be efficient as a recipient in absence of inositol (1162). Used to study glucose (917) and sulfate (641) transport systems. Used extensively for studying induced reversion (392). Used for studying the mechanism of inositol-less death (647, 702), mutagenicity of ferrous ions, and regulation of mitochondrial membrane fluidity; for a review, see reference 702. Spontaneous reversion rates (386). Allele-specific partial suppressor (390). Allele 46802 is nonrevertable and inseparable from translocation *46802* (386, 808). Strains carrying heat-sensitive allele 83201 show slow semicolonial growth in liquid minimal medium at 25°C (641), but look normal on slants (D. D. Perkins, unpublished data). Strains carrying allele 89601 contain cross-reacting material (1183). Mutant gene *exo-1* is present in the *inl* (89601) *a* stock FGSC 498 and may, therefore, be present in stocks of mutants derived by inositol-less death. (See references 194, 325, and 1027). Called *inos*.

***inos*: inositol**

Changed to *inl*.

***int*: intense**

IVR. Linked to *pan-1* (0/50) (*816*).

Brighter orange than the wild type, perhaps

because of morphology rather than carotenoid content (816).

***inv*: invertase**

VR. Right of *pab-2* (3%) and *ro-4* (5 to 8%). Left of *asn* (4 to 9%) (*918*, PB).

Unable to use sucrose as a carbon source. Grows well on glucose or fructose and fairly well on Casamino Acids or yeast extract. Invertase structural gene; invertase deficient and uninducible by normal inducers. Makes cross-reacting material (919). Invertase is also affected by *cot-2*, q.v.

***ipa*: "it pokes along"**

IL. Between *mt* (20%) and *arg-1* (1%) (*994*; E. G. Barry, personal communication).

Hyphae from germinating ascospores or conidia grow for long distances without branching. Cultures thus are 1 day late growing up (E. G. Barry, personal communication). Modifies *pro-3*. Double mutant *pro-3; ipa* does not respond to arginine and grows less well than the single mutant *pro-3; ipa*$^+$ on proline, citrulline, or ornithine. The single mutant *ipa* grows on minimal medium at half the wild-type rate. Arginine uptake is normal: *arg-2; ipa* or *arg-5; ipa* double mutants can grow on arginine. Inhibition studies suggest that *ipa* may be unable to shunt exogenous arginine into the proline pathway (994).

***ipm-1*: isopropylmalate permeation-1**

Unmapped. Unlinked to *ipm-2* or *leu-4*.

Able to use α-isopropylmalate to support growth of *leu-4* mutants and for induction of α-isopropylmalate isomerase and β-isopropylmalate dehydrogenase, in contrast to *ipm*$^+$ strains, which are unable to take up this intermediate (870, 871).

***ipm-2*: isopropylmalate permeation-2**

Unmapped. Unlinked to *ipm-1* or *leu-4*.

Improves α-isopropylmalate uptake by the mutant *ipm-1* in supporting growth of the mutant *leu-4* (870, 871). By itself *ipm-2* is not very effective in promoting permeability of α-isopropylmalate.

***iv*: isoleucine plus valine**

Changed to *ilv*.

***kyn-1*: kynureninase**

VII. Linked to *nic-3* (30%) and *wc-1* (20%) (*G. Lester and P. J. Russell, personal communication*).

Partially defective in induction of the kynurenase I (inducible) isozyme by kynurenine, indole, or tryptophan, but has normal levels of the constitutive kynureninase II isozyme. Possibly regulatory. Scored and selected by a low level of anthranilate accumulation on medium supplemented with a high level of tryptophan, which results in a low level of UV fluorescence compared with that of the wild type (926).

***lac*: lactose nonutilization**

Multigenic basis. No major gene identified. Strains designated *lac* showed poor growth on lactose. Lactase (β-D-galactosidase) properties were unaltered; levels were normal when grown on sucrose and depressed on lactose (584, 585). These strains differ from wild type at several loci, each with a small and additive effect on lactose utilization; e.g., three component genes from "*lac*$^-$" strain 31389 × wild type were shown to be unlinked and were designated *n-lac-1, pow(n-lac-2)* (powdery conidia), and *floc(n-lac-3)* (flocculent morphology). These genes are not specific for lactose utilization but also result in an altered adaptation response to other carbon sources and in other pleiotropic effects. Thus, probably no identified locus qualifies to be designated *lac*. (357) The failure to find a single-gene mutant unable to use lactose is ascribed to the fact that *Neurospora* has two β-galactosidases (605 and references therein).

***le-1*: lethal ascospore-1**

IVR. Linked to *pan-1* (1 to 2%) (382). Right of *cot-1* (14%) (*713, 716*).

Autonomous ascospore lethal with colonial growth. Ascospores are black but mostly fail to germinate. A few do so after special treatment (382). Aconidial colonial growth with dense granular aerial mycelium, turning brown with age (713, 716). Photographs (382, 716). Reduced amount of cell wall peptides (1165). Alleles B55 and S4355 of reference 382 were presumed to be allelic with similar mutations called *col-le-1* (CM3) and (*col-le-2*) by (713, 716), but direct tests were not made.

***le-2*: lethal ascospore-2**

VIIL. Linked to *met-7* (7%). Indicated left (*382*).

Autonomous ascospore lethal with colonial growth. Ascospores are black but mostly fail to germinate, although a few are recovered after aging. Compact colonial growth (382).

leu: leucine

For biosynthetic pathway, see Fig. 15. Leucine mutants have been used extensively for studies of regulation (see references 427 and 833). Leucine mutants acquire suppressors when grown on Difco agar-sorbose medium. The suppressors are leaky auxotrophs blocked at various steps in sulfur metabolism; apparently these blocks allow more efficient use of the traces of leucine in the agar (425; S. R. Gross, personal communication). Most aliphatic and aromatic amino acids can inhibit growth of leucine mutants at appropriate concentrations, probably because of competition for a common uptake system (S. R. Gross, personal communication). For regulation, see individual loci; reviewed in references 427 and 642.

leu-1: leucine-1

IIIR. Between *ad-4* (1 to 5%) and *his-7* (8%) (219, 578, 815, 1052). (*868*)

Requires leucine (867, 868). Lacks β-isopropylmalate dehydrogenase (426) (Fig. 15). Accumulates α-isopropylmalate and β-isopropylmalate (427). Synthesis of the enzyme also requires the function of regulatory gene *leu-3*$^+$ and the presence of α-isopropylmalate, which acts as inducer (427). Resistant to aminotriazole (D. D. Perkins, unpublished data). Female sterile (O. M. Mylyk, personal communication). Used to study reversion and competition in heterokaryons (901).

leu-2: leucine-2

IVR. Between *trp-4* (2%) and *ilv-3* (4%) (579, 991). (*D. C. Regnery, cited in reference 633*)

Requires leucine (867, 868). Structural gene for isopropylmalate isomerase (432, 871) (Fig. 15). Altered heat inactivation of hybrid enzymes (432). Structural differences of hybrid enzymes (871). Accumulates α-isopropylmalate (427). Synthesis of the enzyme also requires the function of regulatory gene *leu-3*$^+$ and the presence of α-isopropylmalate, which acts as an inducer (427). Resistant to aminotriazole (D. D. Perkins, unpublished data). Alleles show intralocus complementation (424). Allele 37501 is heat sensitive (30°C versus 20°C) and is leaky at 25°C (D. C. Regnery, personal communication).

leu-3: leucine-3

IL. Right of the *In(OY323)* left breakpoint and *nit-2* (12 to 18%). Left of *cyt-1* (5 to 8%) and *T(OY321)* (57, 816, PB; D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation). (*868*)

Requires leucine (867, 868). Regulatory mutation; prevents synthesis of α-isopropylmalate isomerase and β-isopropylmalate dehydrogenase and prevents full derepression of α-isopropylmalate synthetase; also involved in regulation of isoleucine and valine synthesis, q.v. (427, 771, 833) (Fig. 15). The original allele, 47313, is leaky, but some other alleles, e.g., R156, are not.

leu-4: leucine-4

IL. Right of *T(OY321)*; hence, of *cyt-1*. Left of *cys-5* (≪1%) (1125; D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation). (*429*)

Requires leucine. Structural gene for α-isopropylmalate synthetase (426, 427, 432) (Fig. 15). Feedback-negative mutants (426, 427). Hybrid synthetases with altered properties (432). Complementation between alleles (432, 1125).

leu-5: leucine-5

VR. Between *cyh-2* (1%) and *sp* (3 to 9%) (818, 839, PB). (*812*)

Strains carrying the only auxotrophic allele, 45208t, have a partial leucine requirement at low temperatures and a tighter leucine requirement at 34°C and stop growth at 37 to 39°C, regardless of leucine supplementation (290, 839). Altered leucyl-tRNA synthetase (839). Apparently a gene complex consisting of structural genes for cytoplasmic leucyl-tRNA synthetase and a separate mitochondrial leucyl-tRNA synthetase. Mutations mapping in the *leu-5* region can affect either enzyme separately or both simultaneously (69, 431). In the [*cni-3*] mitochondrial mutant, mitochondrial tRNA synthetase is greatly increased, whereas the cytoplasmic enzyme is unchanged (430). Allele 45208t causes alterations in unrelated enzymes, apparently via mistranslation (607, 839). Used to study the hypothesis that senescence is due to faulty protein synthesis (607). Assembly of glycerolkinase and glycerol-3-phosphate dehydrogenase into inner mitochondrial membrane is not impaired in *leu-5* strains (213). Poor recovery from ascospores at 34°C (complex complete medium); best germinated at 25°C. Allele 45208t is somewhat unstable (431).

***lis-1*: light insensitive-1**

IR. Between *ad-3* (6%) and *al-1* (16%) (775a; J. Paietta, personal communication).

Circadian conidiation is not suppressed in constant light. Photoinduced carotenogenesis and phase shifting of periodic conidiation are not altered. Recessive in heterokaryons. (755a)

***lis-2*: light insensitive-2**

VI. Between *chol-2* (11%) and *trp-2* (25%) (775a; J. Paietta, personal communication).

Resembles *lis-1*.

***lis-3*: light insensitive-3**

VR. Right of *inl* (4%) (775a; J. Paietta, personal communication).

Resembles *lis-1*.

***lp*: lump**

II. Right of *thr-3* (10%). Linked to *bal* (25%) (818, *812*).

Restricted colonial growth. Differs from *bal*: faster growth, aerial hyphae (812).

***lys*: lysine**

All lysine auxotrophs are inhibited competitively by arginine (287, 288). Resistance to arginine is conferred on *lys-1* mutants by a presumed transport mutation arg^R, q.v. See the *arg* entry for medium that provides both lysine and arginine requirements. Lysine biosynthesis is by the α-aminoadipate pathway in *Neurospora* and other higher fungi (see reference 1103) (Fig. 16). Complex interactions between *lys*, *pyr*, and *arg* mutations have been described (485). Enzymes of lysine biosynthesis are derepressed coordinately with those of arginine, histidine, and tryptophan (1131). See *cpc-1*.

***lys-1*: lysine-1**

V. Right of *caf-1* (4 to 14%). Left of *cyt-9* (5%) and *at* (1 to 20%) (817; K. S. Hsu, personal communication; PB). (*403*)

Uses lysine, α-aminoadipic acid, or ε-hydroxynorleucine (α-amino-ε-hydroxycaproic acid) (399, 400, 684, 1087). Accumulates homocitrate on limiting lysine concentrations (464) (Fig. 16). Fine structure and complementation between alleles (8). Initial allele: 33933.

***lys-2*: lysine-2**

VR. Right of *ilv-1* (4 to 7%). Left of *cyh-2* (<1%) and *leu-5* (9%) (3, 818, 839). (*399*)

Requires lysine. Will not use ε-hydroxynorleucine (400). Probably blocked in conversion of α-aminoadipate semialdehyde to saccharopine (1087) (Fig. 16). Initial allele: 37101.

***lys-3*: lysine-3**

IR. Right of *al-1* (9%) and *al-2* (12 to 15%). Left of *In(OY323)* and *nic-1* (<1%) (2, 907). Not included in duplications from *In(OY323)* × *In(NM176)*; hence, left of *ace-3* (57). (*288*)

Uses lysine or ε-hydroxynorleucine. Probably blocked in conversion of α-aminoadipate to α-

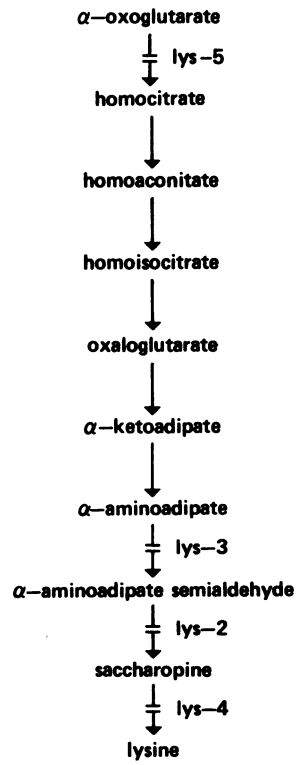


FIG. 16. Biosynthetic pathway of lysine, showing sites of gene action of *lys-4* and *lys-5* and probable sites of *lys-2* and *lys-3* gene action (119, 400, 464, 762, 1087). α-Amino-ε-hydroxycaproic acid can be converted to α-aminoadipate semialdehyde (1178), but apparently is not an intermediate.

aminoadipate semialdehyde, based on precursor utilization (400, 1087) (Fig. 16). Complementation between alleles (13). Ascospores are white and inviable in homozygous *lys-3* × *lys-3* crosses, but some heteroallelic crosses are fertile (13). Inhibited by methionine. Initial allele: 4545.

***lys-4*: lysine-4**

IR. Between *nuc-1* (1%) and *his-3* (1%) (271, 670). (*288*)

Requires lysine (400). Lacks saccharopine-cleaving enzyme activity (1087) (Fig. 16). Complementation between alleles (13). Use 0.5 mg of lysine per ml. Initial allele: 15069.

***lys-5*: lysine-5**

VIL. Right of *cyt-2* (6%), *aro-6* (3%), and *cpl-1* (5 to 7%). Left of *un-4* (2%) (*1012*, PB).

Requires lysine. Partial response to glutaric acid (762). Lacks homocitrate synthase activity (464, 762) (Fig. 16). Some alleles such as 37402, called *asco*, are autonomous ascospore lethals or semilethals, resulting in mostly immature white spores (see reference 1012 for a photograph). Viability of the lys^- spores is improved by long incubation (7). With other alleles such as DS6-85, ascospores blacken and germinate normally. Accumulates malate plus citrate on media with limiting lysine concentrations (464). Four complementation groups (12). Allele 37402 called *asco*.

***lys*^{R}: lysine resistant**

IR. Between *his-3* and *nic-2* (C. C. Ho, personal communication). (*566*)

Growth of the double mutant *arg-1* lys^R is resistant to the normal inhibition by L-lysine. Proposed to be due to basic amino acid transport system (566). Possibly allelic with *su(mtr)-1* (565).

***m*: microconidial**

Allelic with *pe*, q.v.

***ma-1*: malate utilization-1**

Unmapped. Probably in a left arm, any of III to VII.

Unable to use malate as a carbon source when the tricarboxylic acid cycle is blocked by a *suc* (pyruvate carboxylase) mutation. Altered mitochondrial malate dehydrogenase. Scorable only in *suc* mutants (703, 706, 709).

***ma-2*: malate utilization-2**

Unmapped. Unlinked to *ma-1*. Probably in a left arm, any of III to VII.

Unable to use malate as a carbon source when the tricarboxylic acid cycle is blocked by a *suc* (pyruvate carboxylase) mutation. Altered mitochondrial malate dehydrogenase. Scorable only in *suc* mutants (703, 706, 709).

***mac*: methionine-adenine-cystine**

Probably allelic with *met-6*, q.v., but there are some differences between them. Requires methionine. Growth on methionine is stimulated by adenine with possible additional stimulation by cystine. Inhibited by guanine (290). One occurrence: allele 65108.

***mat*: mat**

IVR. Right of *pyr-2* (3%). Left of the *T(NM152)* right breakpoint and *cys-4* (10%) (*633*, 721, 812).

Spreading colonial morphology, with conidiating tufts. Grows better on sucrose minimal medium than on glycerol complete medium (789).

Mating type

See *A/a*.

***mb-1*: male barren-1**

VII. Linked to *nic-3* and *wc-1* (23%) (*1100*, PB).

Perithecial development is blocked when the *mb-1* mutant is used as the male parent to fertilize an mb^+ female: many perithecia are produced, mostly small, brown, without beaks or ascospores; a few perithecia mature and produce ascospores (1100). Perithecia are reported to be normal and fertile when the *mb-1* mutant is the female parent fertilized by an mb^+ strain (1128); however, some abnormality as the female has been observed cytologically after pachytene (N. B. Raju, personal communication). Homozygous barren (PB). Recessive in heterokaryons, complementing *mb-2* and *mb-3* (1101). One occurrence: allele 8455.

***mb-2*: male barren-2**

IR. Between *cyh-1* (5%) and *al-1* (7%) (PB). (*1100*)

Perithecial development is blocked when the *mb-2* mutant is used as the male parent to fertilize an mb^+ female; many perithecia are produced, mostly small, brown, without beaks or ascospores; a few perithecia mature and produce ascospores (1100). Perithecia are normal and fully fertile when the *mb-2* mutant is used as the female parent and fertilized by an mb^+ strain (1128). Homozygous barren (PB). Recessive in heterokaryons, complementing *mb-1* and *mb-3* (1101). One occurrence: allele 8553.

***mb-3*: male barren-3**

IR. Linked to *cyh-1* (18%), *al-1* (2%), and *mb-2* (6%) (*1100*, PB; J. F. Leslie, personal communication).

Perithecial development is blocked when an *mb-2* mutant is used as the male parent to fertilize an mb^+ female; many perithecia are produced, mostly small, brown, without beaks or ascospores; a few perithecia mature and produce ascospores (1100). Perithecia are normal and fully fertile when an *mb-2* mutant is used as the female parent and fertilized by an mb^+ strain (1128). Development of perithecia may be slower than normal, however, when an *mb-2* mutant is used as the female (N. B. Raju, personal communication). Homozygous barren (PB). Recessive in heterokaryons, complementing *mb-1* and *mb-3* (1101). Six occurrences.

mbic

See *Bml*.

***md*: mad**

VR. Between *sh* (3%) and *sp* (9%) (*296*).

Spreading morphological which modifies the banding phenotype of *cl*. Characteristic branching pattern. Photographs. (296)

***me*: methionine**

Changed to *met*.

***mea-1*: methylammonium resistant-1**

Unmapped.

Presumed defective in transport of ammonium (293). *nit-2; mea-1* double mutants show nitrogen-starved growth on ammonium (R. H. Garrett, personal communication).

***med*: medusa**

IVR. Linked to *met-5* (5%) and *pan-1* (8%) (*382*).

Slow growing, spreading, morphological mutant, with distinctive grooves on the agar surface (382). For a photograph, see Fig. 17 of reference 382. Reduced amount of cell wall peptides (1165).

***mei*: meiotic**

Meiosis impaired. In addition to partial or complete blocks of meiosis and ascus development, some mutations designated *mei* have effects that may include sensitivity to radiation, to radiomimetic drugs, and to histidine and increased duplication instability. Both recessive and dominant meiotic mutations are known. Mutations that affect meiotic or premeiotic events may have other names, depending on the phenotype first recognized. See: *asc; fmf-1; mb; mus-7, -8, -9,* and *-11; uvs-3, -5,* and *-6*. Meiotic mutants are very common in natural populations of *N. crassa* (601).

***mei-1*: meiotic-1**

IVR. Linked to *arg-2* (<1%), probably to the right (*995*).

Meiosis is impaired in homozygous crosses. Recessive. Meiotic divisions occur and many ascospores are produced, but 70 to 90% are inviable and white. The viable ascospores are usually disomic for one or more linkage groups, indicating high nondisjunction at the first division (254, 995). Chromosome pairing is defective: axial elements of synaptonemal complex are present, but a completed complex is rarely seen. Separation at anaphases I and II is defective, leading to four-poled second and third division spindles (625). Not sensitive to UV, methyl methane sulfonate, ionizing radiation (939), or histidine (939; D. Newmeyer, unpublished data).

mei-1 is present in wild-collected strain Abbott 4A (995), which is an ancestor of many Beadle and Tatum mutants (68). Possible allele *asc(DL243)* complements *mei-1* but did not recombine with it (0/3,000) (253). *DL243* and *mei-1* strains differ phenotypically. In *DL243* mutants, the major block is before karyogamy; the few

asci produced have normal meiosis I but high nondisjunction at meiosis II, with most chromosomes usually attached to only one spindle-pole body (254). Possible allele *asc(DL95)* complements *mei-1* and *asc(DL243)*, but did not recombine with *DL243* (0/96) (253). *DL95* is phenotypically like *mei-1* but is less extreme (254).

***Mei-2*: Meiotic-2**

VR. Linked near *inl* (*995*). Between *al-3* (20%) and *his-6* (A. L. Schroeder, personal communication).

Meiotic divisions occur, and many ascospores are produced, but many are inviable and white. Crosses heterozygous or homozygous for *Mei-2* give extensive nondisjunction of all linkage groups (995). Chromosome pairing much reduced (B. C. Lu, cited in reference 995). Sensitive to methyl methane sulfonate, histidine, and gamma rays (939). Dominant in the original strain (995), but progeny show incomplete penetrance (939).

***mei-3*: meiotic-3**

IL. Right of *arg-1* (3%). Probably right of *eth-1* (1%) and *arg-3* (1%). Left of the *T(39311)* right breakpoint; hence, left of the centromere and *sn* (*757*, 808).

Homozygous barren (757). Recessive. Blocks meiosis in zygotene (860). Sensitive to UV, histidine (757, 759), mitomycin C (195), ionizing radiation, and methyl methane sulfonate (939). Sensitivity to UV and histidine is temperature sensitive; best scored at 39°C, at least for strains carrying allele N289 (757). Causes increased duplication instability (mitotic recombination, deletion, or both) (757).

***mei-4*: meiotic-4**

IIIR. Linked to *leu-1* (12%), probably to left (*757*).

Homozygous barren. Recessive. Highly variable expression, depending on genetic background (757). Crosses with more extreme genotypes are blocked at crozier formation or karyogamy (860). Crosses with less extreme genotypes complete normal first division but become irregular at later divisions, producing abnormal spores (B. C. Lu and D. R. Galeazzi, cited in reference 860). Not sensitive to UV, methyl methane sulfonate, or gamma rays in spot tests, or to histidine (757, 939).

***mel-1*: melon-1**

VIIL. Left of *thi-3* (27%) (*819*).

Growth as hemispherical colonies, similar to those of the mutant *bal* (717). Growth stimulated rather than depressed by sorbose (715). Cell wall analysis (278). Photographs (278, 717). Called *col(C-L2b)*. Not tested by crossing to possible allele *do*.

***mel-2*: melon-2**

Allelic with *bal*, q.v. (812).

***mel-3*: melon-3**

III. Linked to *leu-1* (17%) (*717*).

Growth as hemispherical colonies similar to those of the mutant *bal*. Photographs. A separate modifier gene is also located in linkage group III. (717)

***Mep*^{r}: methylpurine resistant**

See *mep*.

***mep(3)*: methylpurine resistant**

Not mapped. Segregates 1:1.

Resistant to 6-methylpurine. Adenine phosphoribosyltransferase activity near normal in vitro. Uptake is normal. 6-Methylpurine prevents purple pigment production by *ad-3* single mutants on low adenine concentrations, but it does not prevent pigment production by the *ad-3A; mep(3)* double mutant, suggesting that *mep(3)* results in an alteration in the regulation of de novo purine synthesis. Has low hypoxanthine phosphoribosyltransferase activity, as do *mep(10)* and *ad-8* mutants, q.v. Selected on 1 mM 6-methylpurine–sorbose medium (785). Phenotype consistent with lowered affinity of glutamine amidotransferase for 6-methylpurine as a feedback inhibitor (788). Not tested for allelism with *mep(10)* or with *ad* mutations. Called *Mep*^{r}*-3*, but not indicated to be dominant (785). Called *mep-1* by Barratt and Ogata (51).

***mep(10)*: methylpurine resistant**

Not mapped. Segregates 1:1.

Resistant to 6-methylpurine. Adenine phosphoribosyltransferase activity is negligible in vitro. Adenine uptake is normal. Resistance may

result from inability to convert the analog to nucleotide form. Has low hypoxanthine phosphoribosyltransferase activity, as do the mutants *mep(3)* and *ad-8*, q.v. Selected on 1 mM 6-methylpurine–sorbose medium. Not tested for allelism with *mep(3)*. Called *Mep*^{r}*-10*, but not indicated to be dominant. (785) Called *mep-2* by Barratt and Ogata (51).

***met*: methionine**

Auxotrophs designated *met* require methionine, and some can use its immediate precursors, homocysteine and cystathionine; they cannot use cysteine. (Mutants able to use cysteine as well as methionine are designated *cys*.) For the methionine biosynthetic pathway, see Fig. 17. For a review, see reference 351. For regulation, see individual loci and reference 965. Formerly called *me*.

***met-1*: methionine-1**

IVR. Right of *oxD* (3%) and the *T(S1229)* left breakpoint. Left of *col-4* (4%) (55, 158, *718*, 768, 808).

Uses methionine but not homocysteine (469) (Fig. 17). Lacks methylene tetrahydrofolate reductase and, thus, lacks the coenzyme needed for transmethylating homocysteine (124, 963, 964). A report that the mutant *met-1* also lacks cystathionine-γ-synthase (547) proved incorrect; the error resulted because methyl tetrahydrofolate is an essential activator of cystathionine-γ-synthase (965). Methylene tetrahydrofolate reductase is feedback-inhibited by *S*-adenosylmethionine (124). Used in heteroallelic duplications from *T(S1229)* to assay mitotic recombination (56).

***met-2*: methionine-2**

IVR. Between *trp-4* (6%) and *pan-1* (4%) (719). Linked to *ilv-3* (0/129) (*354*, 579).

Uses methionine or homocysteine; accumulates cystathionine (469). Lacks cystathionase II (353) (Fig. 17). Fine-structure map (720, 724). Complementation map (719). Used in major studies of intralocus recombination and its polarity (720, 724).

***met-3*: methionine-3**

VR. Right of *trp-5* (4%) and *pab-1* (1%). Left of *pk* (1%) (6, 296, 362, 1036). (*125*)

Uses methionine, homocysteine, or cystathionine (469). Lacks cystathionine-γ-synthase (547) (Fig. 17). This enzyme is also lacking in the mutant *met-7* (547). The enzyme is activated by methyl tetrahydrofolate and feedback inhibited by *S*-adenosylmethionine (547, 965).

***met-4*: methionine-4**

Changed to *cys-10*, q.v. (721).

***met-5*: methionine-5**

IVR. Between *his-4* (4%) and *nit-3* (15%) (812, PB). (*125*)

Uses cystathionine, homocysteine, or methionine (354, 718). Defective homoserine transacetylase (547, *733*) (Fig. 17).

***met-6*: methionine-6**

IR. Right of *T(NM103)*, *T(ALS182)*, and *thi-1* (7 to 14%) (808, 1091, PB). Left of *ad-9* (2 to 16%) (466, 723, 789). Adjoining or allelic with *mac* (722, 724). (*125*)

Requires methionine; does not use precursors (718; N. H. Horowitz, cited in reference 1180). Strain(s) carrying allele 35809 lacks polyglutamate forms of folate and, thus, apparently lacks the coenzyme needed for transmethylating homocysteine (208, 886, 963, 964) (Fig. 17). An incorrect report that the mutant *met-6* also lacks cystathionine-γ-synthase (547) proved to be due to the methyl tetrahydrofolate being removed during preparation of extracts; methyl tetrahydrofolate is an activator of cystathionine-γ-synthase (965). The relationships between *met-6* (35809) and its probable alleles *met* (S2706) and *mac* (65108) are not clear. *met* (S2706) and *mac* both complement *met-6* (35809), but do not complement each other, indicating that at least *met* (S2706) and *mac* are alleles. In a high-resolution recombination study with flanking markers, *met-6* (35809) and *met* (S2706) behaved like alleles, but *mac* behaved atypically, although almost equally closely linked (724). The *mac* mutant is reported to differ from the others in causing an accessory requirement for adenine and possibly cystine (290), whereas *met-6* (35809) and *met* (S2706) strains are stimulated by adenine only in a CO_2-enriched atmosphere (G. Roberts, cited in reference 724). *mac* and *met-6* (35809) strains evidently lack different folylpolyglutamate synthetase activities (208, 886). Used to study polarity in intralocus recombination (722, 724). Polarity with respect to flanking markers is not reversed when the *met-6* region is inverted relative to the centromere (722).

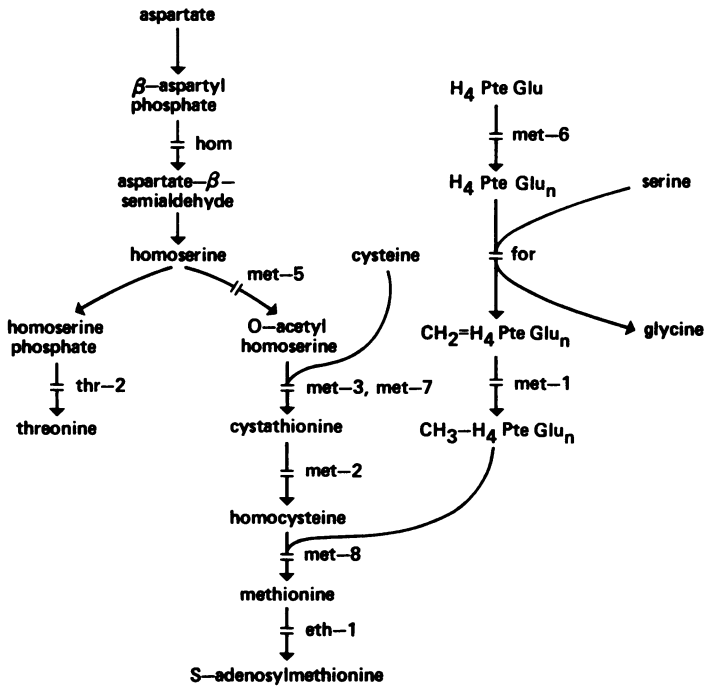


FIG. 17. Biosynthetic pathways of homoserine, threonine, and methionine, showing sites of gene action (124, 208, 209, 351, 352, 518, 547, 965). For conversion of threonine to isoleucine, see Fig. 15. H_4PteGlu, Tetrahydrofolate. It is not clear whether the polyglutamylation step controlled by *met-6* occurs only at the stage shown.

***met-7*: methionine-7**

VIIR. Right of *qa-2* (<1%), *ars* (<1%), and the centromere (one second-division ascus in several hundred). Left of *met-9* (10^{-4}) and *wc-1* (1 to 4%) (146, 725; M. E. Case, personal communication). (*718*; M. K. Allen, cited in references 718 and 789)

Uses cystathionine, homocysteine, or methionine (718; N. H. Horowitz, cited in reference 1180). Lacks cystathionine-γ-synthase (547) (Fig. 17). This enzyme is also lacking in the mutant *met-3* (547). See *met-3* for regulation. Apparently contiguous with *met-9* by coconversion. Flanking markers are recombined in most *met-7*$^+$ *met-9*$^+$ recombinants (725). Functionally distinct from the mutant *met-9*, which has active cystathionine-γ-synthase (547) but cannot use homocysteine. No mutants lacking both functions have been isolated. Allele NM251 is suppressible by supersuppressor RN33 (same as *ssu-1*?) (725). Allele K79 is inseparable from reciprocal translocation *T(I;VII)K79* (808).

***met-8*: methionine-8**

IIIR. Between *ff-5* (1 to 4%) and *ad-4* (4%) (219, 815, 1052). (*718*)

Uses methionine but not precursors (718). Lacks methyl tetrahydrofolate homocysteine transmethylase (124, 964) (Fig. 17).

***met-9*: methionine-9**

VIIR. Between *met-7* (10^{-4}) and *wc-1* (1 to 2%) (725). (*815*)

Requires methionine; cannot use precursors (290, 718, 725). Apparently contiguous with *met-7*, q.v. (725). Functionally distinct from *met-7*. The mutant *met-9* retains the *met-7*$^+$ function, producing cystathionine-γ-synthase (547). Allele NM43 is heat sensitive (725).

***met-10*: methionine-10**

IR. Near *lys-4*. Right of *nuc-1*, *T(AR173)*, and *his-2*. Left of *his-3* and *ad-3* (757, 808; R. L. Metzenberg, personal communication). (*P. Dodd, cited in reference 816*)

Requires methionine. The only known allele is heat sensitive, with the requirement at 34°C, not at 25°C (816; P. Dodd, cited in reference 816); does not grow at 39°C even with methionine (757).

***meth*: methionine**

Changed to *met*.

methionine overproduction

See *eth-1* (542).

[*mi-1*]: maternal inheritance-1 (synonym: [*poky*])

Mitochondrial mutant with slow growth and deficient cyanide-sensitive respiration (see reference 394). See *su([mi-1])*.

Microconidiation genotypes

Microconidia, being uninucleate, are valuable for such applications as somatic analysis and mutagenesis. They are much less abundant in the wild type than are multinucleate macroconidia, except under certain conditions (893). Several genotypes are known that increase the production of microconidia, notably, *pe* and *dn*, but these single-mutant strains also continue to produce macroconidia. A few microconidia, and no macroconidia, are produced by the single-mutant strains *fl* and *cpt*, q.v.; large numbers can be obtained from *fl* strains under certain conditions (893). Large numbers of almost exclusively uninucleate microconidia can be obtained by using the double mutants *pe fl* or *dn; fl*, in which *fl* blocks macroconidiation and the *pe* or *dn* component promotes microconidiation. *dn; fl* strains have the advantage over *pe fl* strains of greater fertility in homozygous crosses (806), but microconidia from *dn; fl* strains are less viable (454). Cultures abundantly producing only microconidia appear grey-brown rather than orange. See *fl*, *dn*, and *pe*. For colonial microconidiating strains, see *col-1*, *col-4*. See references 415 and 416 for other gene interactions.

***mig*: migration of trehalase**

IR. Between *met-6* (7%) and *al-2* (20%). Between *tre* (≪1%) and *ad-9* (*1045*, 1176).

Altered electrophoretic mobility of trehalase (1045). Putative trehalase structural gene (1047). (However, see qualifications in reference 1176.) Polymorphic in laboratory stocks of *N. crassa* and in wild isolates of *N. intermedia* (1176). The adjoining mutant gene *tre* results in production of a protein inhibitor of trehalase (1045).

***mo*: morphological**

Name and symbol used by Garnjobst and Tatum (382) for a miscellaneous group of mutants having spreading growth on agar, sometimes with scanty or fine hyphae and reduced conidiation. The symbol *morph* has also been used. Other categories of morphological mutants were designated *col*, *spco*, or *smco*. Other workers have assigned descriptive names, e.g., *bal*, *fr*, *ro*, and *sc*. See also *moe*. For reviews covering morphological mutants and morphogenesis, see references 112, 197, 642, 675, 942, 946, and 1088. Growth rates and hyphal diameters of 18 morphological mutants are given in reference 197.

***mo-1*: morphological-1**

I. Linked mating type (9%) (*382*).

Altered morphology. Slow growth from ascospores (382).

***mo-2*: morphological-2**

VII. Linked to *nt* (29%) and *for* (16%) (*382*, PB).

Slow growing, poorly pigmented mycelium. No conidia. Poor recovery from ascospores (PB).

***mo-3*: morphological-3**

See *sk*.

***mo-4*: morphological-4**

IIIR. Right of *leu-1* (8%). Linked to *pro-1* (10%) (*382*). (F. J. Doe, personal communication)

Altered morphology. Conidiates throughout slant. Complements *col-14*, *col-16*, and *spg* (382).

***mo-5*: morphological-5**

I. Linked mating type (20%) (*382*).

Few conidia. May make exudate on slant (382).

mo(KH160)

See *shg*: shaggy.

mo(P1163)

See *dr*: drift.

mo(P2402t)

See *un-20*.

***mod-5*: modifier of permeability**

VI. Linked to *trp-2*, near the centromere (3%) (*909*).

Improves growth of *trp-1*, *trp-2*, *trp-3*, *trp-4*, *aro-1*, *tyr-1*, *tyr-3*, *pt*, *met-7*, and *pyr-1* strains on complex media. Increases sensitivity to 4-methyltryptophan and *p*-fluorophenylalanine. Recessive in heterokaryons. Attributed to permeability change that facilitates entry of metabolites (53, 909). Scorable on slants of minimal medium plus 4-methyl-DL-tryptophan (0.9 mg/ml, autoclaved; tests read at 7 days 34°C) (PB). Map location similar to that of *mts*, but not tested for allelism. *mts* differs in not allowing the mutant *pyr-1* (H263) to grow on complex media (160). (Locus symbols *mod-1*, *-2*, *-3*, and *-4* have not been used.)

***mod(sc)*: modifier of scumbo**

IV. Linked to *pan-1* (17%) (*497*).

Restricts the growth of *sc* but not of four other morphological mutants (*cr-1*, *fr*, *bis*, *sp*) or of the wild type (497).

***moe-1*: morphological, environment sensitive-1**

Probably allelic with *sk*, q.v.

VII. Linked to *nt* (12 to 19%) (*382*), probably to the right (PB).

Morphology identical to that of *sk* mutants; linkage similar (PB). Morphology reported influenced by temperature and medium: more spreading on minimal or complete medium at 25°C; zoned growth at 34°C on complete (382). Temperature effect could be due to *scot*, the presence of which in the strain of origin was not recognized. Photographs of strain R2408: Fig. 19 through 22 in reference 382. Reduced amount of cell wall peptides (1165).

***moe-2*: morphological, environment sensitive-2**

VI. Linked to *trp-2* (14%), probably to the left (*382*).

Grows with concentric zones on minimal medium and as restricted colonies on glycerol complete medium (34°C). Photographs of strain R2532: Fig. 23 and 24 in reference 382. The *scot* mutation may have been present in the strain of origin.

***moe-3*: morphological, environment sensitive-3**

IV. Left of *pan-1* (17 to 25%) and *bd* (18%) (*929*).

Blocks conidial germination at high temperature. Colonial at high temperature if on dialysis tubing on agar surface, but fairly normal vegetative growth if submerged. Strong circadian conidiation rhythm at low temperature (929). Effect on conidial germination (but not on vegetative growth) counteracted by high conidial concentration or CO_2 (190, 929). Histidine is stimulatory, but there is disagreement as to whether it affects germination or vegetative growth (929; G. W. Charlang, personal communication). Partially curable by siderophores (ferricrocin). Conidia rapidly lose siderophores on contact with aqueous medium, even at permissive temperatures, suggesting an alteration in the plasma membrane attachment site (190). Called JS134-9.

***morph*: morphological**

Symbol changed to *mo*.

***mt*: mating type**

See *A/a*. Also used as a symbol for *mtr*.

***mtr*: methyltryptophan resistant**

IVR. Between *pdx-1* (2%) and *col-4* (1%) (101, *1017*).

Resistant to 4-methyltryptophan and *p*-fluorophenylalanine. *pmn* (= *Pm-N*, *pm n*), selected by resistance to *p*-fluorophenylalanine, has been shown to be allelic with *mtr* (R. Sadler and S. Ogilvie-Villa, personal communication; see also reference 248). Defective in transport of neutral aliphatic and aromatic amino acids via amino acid transport system I (as defined in reference 777) (248, 602, 1017, 1152). Causes an alteration in surface glycoproteins (1038). Used extensively for transport studies (247a, 1150 [review], 1152), also for studies of the mechanism of intralocus recombination (1021). Resistance is recessive in duplications from *T(S1229)* (PB). Recessive resistance used in a heterokaryon test system for mutation studies (1020). Suppressors obtained and used for selecting other resistance mutants (106, 107, 555, 1018). Allele 26 is a

putative frameshift mutation reverted by ICR-170 (106, 107). *mtr* ascospores are slow to darken and mature; up to 50% of the young ascospores from heterozygous crosses are white (152, PB). With probable allele MN18, ascospore viability is improved by the addition of peptone to the crossing medium when the male parent is added (152). *mtr* has been scored on media containing 10 or 70 μg of filter-sterilized 4-methyltryptophan per ml or on 20 or 60 μg of *p*-fluorophenylalanine per ml (550, 1021, PB). Unlike 4-methyltryptophan, *p*-fluorophenylalanine is heat stable and can be added before autoclaving. Strains with mutations at the *mtr* locus may be obtained by selection for resistance to numerous agents or for defects in uptake ability. Thus, there is confusion in nomenclature. Genes originally designated neu^a, neu^r, neu^t, tr^u (628) may be *mtr* alleles. *mtr* was initially called *mt* (602).

***mts*: methyltryptophan sensitive**

VII. Right of *ylo-1* (<1%) (152, 160).

Sensitive to analogs of all tested aromatic, neutral, and basic amino acids and to analogs of purines and pyrimidines. Ten to 100 times more sensitive than the wild type. Not sensitive to cold, salt, or detergent. Resembles *mod-5* in enabling the mutant *trp-3* (A78) to grow well on complex media, but differs in not doing so for the mutant *pyr-1* (H263). No allelism test with *mod-5*. Obtained by filtration enrichment in the presence of 5-methyltryptophan (152, 160). Used for selection of mutants resistant to analogs: 5-methyltryptophan (152); 8-azaadenine (524). Could be useful where the wild type is not sufficiently sensitive to allow direct selection of resistant mutants. Conveniently scored on *p*-fluorophenylalanine (2 μg/ml, solid medium, autoclaved in medium; *p*-fluorophenylalanine is more heat stable than is 5-methyltryptophan). Best tested with small inocula on slants (10 by 75 mm) and read after 3 days at 34°C (PB). Called *5mt*.

multicent

Linkage tester strain containing *mt, bal, acr-2, pdx-1, at, ylo-1*, and *wc-1*, which are linked to centromeres of linkage groups I through VII, respectively (800). Especially useful to establish linkages of translocations (808). Scoring of test markers is somewhat more laborious than for *alcoy*, which may therefore be preferred for locating point mutations.

***mus*: mutagen sensitive**

Symbol adopted in 1980. Locus numbers begin with *mus-7* to avoid confusion with *uvs-1* through *-6* (537, 539). Previously named mutagen-sensitive genes bearing other symbols retain their original designations in the present compilation. (See *uvs-1* to *-6, upr-1, Mei-2, mei-3, nuh-4*, and *gs*.) Several new unmapped *mus* genes (255) are not listed separately. For properties of double mutants, see reference 539.

***mus-7*: mutagen sensitive-7**

IIR. Between *arg-5* (8 to 12%) and *nuc-2* (11%) (*539*).

Sensitive to X rays, methyl methane sulfonate, and nitrosoguanidine, but not to UV. Extremely sensitive to histidine. Normal spontaneous, UV-induced, and X-ray-induced mutation. Homozygous barren (537, 539). Not tested for allelism with *asc(DL879)*, which maps in same region and causes nondisjunction when homozygous.

***mus-8*: mutagen sensitive-8**

IV. Linked to *pdx-1* (6%) and *mtr* (1%) (537, *539*; E. Käfer, unpublished data).

Sensitive to UV, X rays, methyl methane sulfonate, nitrosoguanidine, and mitomycin C. Decreased spontaneous mutation (537). Homozygous barren (539).

***mus-9*: mutagen sensitive-9**

IR. Between *cyh-1* (18%) and *al-2* (6%) (*537*).

Sensitive to UV, X rays, methyl methane sulfonate, histidine, nitrosoguanidine, and mitomycin C. High spontaneous mutation; little or no mutability by UV or X rays. Homozygous sterile. Reduced conidial viability. (537, 539) Defective in extracellular nuclease, giving reduced halos around colony on DNA agar (537). Initially called *uvs(FK104)* in reference 538.

***mus-10*: mutagen sensitive-10**

VIIR. Right of *met-7* (7%) (*539*).

Moderately sensitive to UV and methyl methane sulfonate. Not sensitive to nitrosoguanidine or mitomycin C. Slight or no sensitivity to X rays or histidine. Homozygous fertile (although less so than the wild type). Normal spontaneous and UV- and X-ray induced mutation. (537, 539)

***mus-11*: mutagen sensitive-11**

VR. Linked to *pab-2* (*539*), near *his-6* (E. Käfer, personal communication).

Extremely sensitive to methyl methane sulfonate and histidine; also sensitive to X rays, nitrosoguanidine, and mitomycin C (<X rays). High spontaneous mutation. Little or no mutability by UV or X rays. Homozygous barren. Reduced conidial viability (537, 539). Not allelic with *Mei-2* (939).

***mus(SC3)*: mutagen sensitive**

Perhaps VI, linked to *lys-5*.

Sensitive to methyl methane sulfonate but not to histidine. Slow growth. Both the mycelium and conidia are sensitive. Very sensitive on methyl methane sulfonate medium, but not after treatment of conidia with methyl methane sulfonate. (255) Allelism with *mus(SC10)* has not been excluded.

***mus(SC10)*: mutagen sensitive**

Sensitivity cosegregates with a translocation involving linkage groups II, III, and VI.

Sensitive to methyl methane sulfonate, UV, and X rays. Sensitive to histidine at 37°C but not at 25°C. High spontaneous mutation. Female sterile. Complements *uvs-4* (in IIIR) (255; A. M. De Lange, personal communication). Allelism with *mus-7* (II) or *uvs-5* (III) is not excluded.

***mus(SC15)*: mutagen sensitive**

V. Left of *inl* (10%) (*255*).

Highly sensitive to methyl methane sulfonate but not to histidine (255). Sensitive to X rays (A. M. De Lange, personal communication). Both the mycelium and conidia are sensitive. Very sensitive on methyl methane sulfonate medium; the effect is slight after treatment of conidia with methyl methane sulfonate (255). No allelism test with *mus(SC17)*.

***mus(SC17)*: mutagen sensitive**

V. Left of *inl* (27%) (*255*).

Sensitive to methyl methane sulfonate but not to histidine. Sensitivity is shown by the mycelium, not by conidia, and only after preincubation at 15°C. Growth is cold sensitive on minimal medium (255).

***mus(SC28)*: mutagen sensitive**

IR. Right of *al-1* (18%) (*255*).

Sensitive to methyl methane sulfonate. Both the mycelium and conidia are sensitive (255).

***nada*: NAD(P)ase**

IV. Left of *ad-6* (18%) (*747*).

NAD(P) glycohydrolase structural gene. Normal morphology. Identified by a plaque test, using *Haemophilus influenzae*. Recessive in heterokaryons. Allele 62ts is temperature sensitive, with altered substrate affinity. (747) Used in a study of glutamic acid decarboxylase during conidial germination (196).

***nap*: neutral and acidic amino acid permeability**

VR. Linked to *inl* (15%) (*516*); right of *ure-2* (32%) (1149).

Selected as resistant to ethionine plus *p*-fluorophenylalanine (516). Causes reduced amino acid uptake by neutral, basic, and general systems. Also causes reduced uptake of uridine and glucose. Defect is not in amino acid-binding glycoproteins. (865) See reference 1149 for aspartate uptake and resistance to inhibitors. Scored by spotting conidial suspension on minimal medium plus 1.5% sucrose, agar, 0.3 mM ethionine, and 0.02 mM *p*-fluorophenylalanine.

***nd*: natural death**

IR. Between the centromere (15%) and *al-2* (20%) (*981*).

Decreasing clonal growth potential under all nutritional conditions, followed by abrupt irreversible cessation of growth (707, 981). Hypersensitive to sorbose. Conidia die rapidly on slants at 4°C (707). Recessive in heterokaryons. An aged strain can be rejuvenated through heterokaryosis or by crossing to nd^+. Extracts nontoxic (981). Used to examine hypotheses of senescence based on faulty protein synthesis (607) and lipid autoxidation with free-radical reactions (702). Stocks maintained in balanced heterokaryons. Initial growth rate of the original strain, 2.5 mm/h; however, *nd* progeny free of modifiers grow initially at 4.5 mm/h (wild-type rate) (707).

***ndc-1*: nuclear division cycle-1**

VR. Left of *arg-4* (2%) (*976*).

Heat-sensitive conditional mutant. Growth at 25°C but not at 34°C. Recessive. Division cycle blocked just before initiation of DNA synthesis while spindle-pole bodies are duplicated but not separated. (976) Scored as an irreparable *un* mutant (see *un*).

neu: neutral amino acid transport

See *mtr*.

nic: nicotinic acid

nic mutants are preferably supplemented with nicotinamide rather than nicotinic acid at most pH values because of permeability (97). *nt* mutants are best treated as *nic* mutants for purposes of growth and scoring. For good recovery of some *nic* mutants from crosses, crossing media should be supplemented with nicotinamide at levels higher (10×) than those required for growth, even when the protoperithecial parent is nic^+ (789; P. St. Lawrence, personal communication). See Fig. 18 for the biosynthetic pathway. For regulation, see references 111, 371, 604, and 926.

nic-1: nicotinic acid-1

IR. Right of *ace-3* (<1%), *lys-1* (1%), and *In(OY323)*. Left of *os-1* (10 to 29%) (2, 57, 131, 578, 789, 816, 907). (*482*)

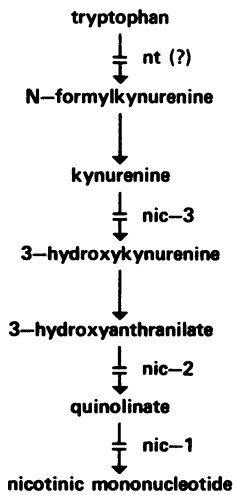


FIG. 18. Pathway from tryptophan to nicotinic mononucleotide, showing sites of gene action (96, 100, 368, 1168). The enzymatic reactions between 3-hydroxyanthranilate and nicotinic mononucleotide have not been demonstrated directly in *Neurospora*.

Uses nicotinic acid or nicotinamide, but not precursors (97, 100) (Fig. 18). Accumulates quinolinic acid (100). Used to study intralocus recombination (907). Called the *q* locus.

nic-2: nicotinic acid-2

IR. Between *ad-3B* (4%) and *ace-7* (4 to 7%) (271, 578). (*482*)

Grows on nicotinic acid, nicotinamide, or high concentrations of quinolinic acid (97, 1168). Cannot use kynurenine, hydroxykynurenine, or hydroxyanthranilic acid (96, 1168). Accumulates 3-hydroxyanthranilic acid (96) (Fig. 18). Aging cultures accumulate red-brown pigment in the medium. Used to study intralocus recombination (908). Translocations *T(4540)* and *T(S1325)* are inseparable from *nic-2* (808, 908, 911).

nic-3: nicotinic acid-3

VIIL. Right of *spco-4* (1%) and *do* (3%). Left of *thi-3* (9 to 27%) and *csp-2* (16 to 22%) (539, 812, 816, PB). (*M. K. Allen, cited in references 718 and 789*)

Uses nicotinic acid, nicotinamide, 3-hydroxyanthranilic acid, 3-hydroxykynurenine, or high concentrations of quinolinic acid (96, 1168). Accumulates α-*N*-acetylkynurenine; blocked in conversion of kynurenine to 3-hydroxykynurenine (1168) (Fig. 18). Pyridine nucleotide levels (111).

nit: nitrate nonutilizer

Conveniently scored on synthetic crossing medium (1134), in which nitrate is the sole nitrogen source. Also scorable on slants by pH change when grown with ammonium nitrate as the nitrogen source and bromcresol purple (4 mg/ml) as an indicator (791). In most crosses, a *nit* mutant can be used as the fertilizing parent; in *nit* × *nit* or other crosses where it is required as the female parent, crossing medium can be altered by substituting ammonium nitrate for potassium nitrate (155). Nitrite is toxic at low pH; test media containing nitrite should be neutralized, and the nitrite should preferably be filter-sterilized (G. S. Sorger, personal communication). For a summary of nutritional requirements, based on the data of various authors, see reference 1080. *nit-1*, *nit-7*, *nit-8*, and *nit-9* involve a molybdenum-containing cofactor common to nitrate reductase and xanthine dehydrogenase (591, 1080, 1081) (Fig. 19 and 24). For a review of nitrate assimilation, see reference 385.

For regulation, see reference 643 (review), references 292, 835, 837, and 1081, and entries for *nit* loci, *gln-1*, and *nmr*.

nit-1: nitrate nonutilizer-1

IR. Right of *Tp(T54M94)* and *ad-9* (3 to 15%). Left of *cyh-1* (6%) (466, 496, 816). (*482*)

Cannot use nitrate or hypoxanthine as a nitrogen source, but uses nitrite, ammonia, or amino acids (1000). Does not prevent formation of nitrate reductase apoprotein (999), but lacks the molybdenum-containing cofactor common to nitrate reductase and xanthine dehydrogenase (591, 741) (Fig. 19 and 24). The nitrate reductase in *nit-1* extracts does not catalyze the complete electron transport sequence from NADPH to NO_3^-, but does catalyze the initial part of this sequence if a suitable electron acceptor (e.g., cytochrome *c*) is provided (999). See reference 198 for a model of interaction of *nit-1* and *nit-3* gene products. See references 226, 999, and 1000 for regulation.

nit-2: nitrate nonutilizer-2

IL. Right of the *T(39311)* left breakpoint and of *un-5* (2%). Left of *In(OY323)* and *leu-3* (12 to 18%) (57, 808, 816, PB). (*335*, *1135*)

Cannot use nitrate, nitrite, purines, or most amino acids as a nitrogen source but will grow on ammonia, glutamine, or glutamate. *nit-2*$^+$ is a major nitrogen control gene and mediates nitrogen catabolite repression. The *nit-2* mutant is missing (or has severely reduced levels of) nitrate reductase, nitrite reductase, uricase, xanthine dehydrogenase, allantoinase, allantoicase, L-amino acid oxidase, general amino acid permease, extracellular protease, and an intracellular neutral phenylmethylsulfonyl fluoride-sensitive protease (227, 324, 441, 872, 1001, and references therein). Also affects levels of glutamate dehydrogenases (226) and uptake of uracil and uridine (128). Prevents leaky growth of the mutant *am* on minimal medium (155). The product of the *nit-2* gene has been tentatively identified as a nuclear DNA-binding protein, whose affinity for DNA is reduced in the presence of glutamine (433). Allele K31 (called pink) originated in *N. sitophila* and was introgressed into *N. crassa* (335); protein product of K31 may show altered mobility (433). Recombination within the *nit-2* locus is subject to regulation by *rec-1* (157). Heterozygosity for closely linked *ss* reduces recombination within *nit-2* (161). Called *amr*: ammonium regulation in reference 872.

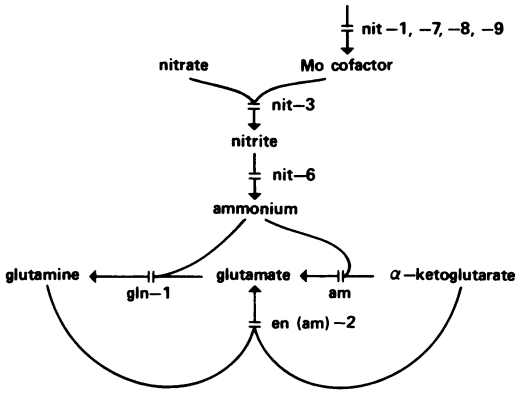


FIG. 19. Nitrate reduction pathway showing sites of gene action. *am* only blocks the NADP-specific glutamate dehydrogenase, not the NAD-specific enzyme. (185, 293, 336, 591, 912, 999, 1081)

nit-3: nitrate nonutilizer-3

IVR. Between *met-5* (15%) and *pyr-2* (2 to 9%) (1000, PB). (*453*)

Cannot use nitrate as a nitrogen source, but uses nitrite, ammonia, hypoxanthine, or amino acids (28, 999). Structural gene for NADPH nitrate reductase (28) (Fig. 19). Allele 14789 apparently codes for an altered enzyme that cannot catalyze the whole electron transport sequence from NADPH to NO_3^-, but can catalyze the terminal portion of this sequence, providing that a suitable electron donor (reduced viologen dye) is provided (999). See reference 198 for a model of interaction of *nit-1* and *nit-3* gene products. For regulation, see references 226, 999, and 1000. The *nit-3*$^+$ gene has been cloned and is expressed in *Escherichia coli* (989).

nit-4: nitrate nonutilizer-4

IVR. Right of *pyr-1* (1 to 6%). Probably right of *col-4* (2%). Left of *pan-1* (6 to 27%) (1000, PB). (*94*)

Cannot use nitrate or nitrite as a nitrogen source, but uses ammonia and amino acids (94). Regulator for induction by nitrate of nitrate reductase and nitrite reductase (1080). Allele nr15, called *nit-5* (1000), is phenotypically identical to other *nit-4* alleles; shown to be allelic by failure to complement or recombine (0 prototrophs per 2,080 progeny) (1080). Original allele was discovered in a wild isolate of *N. intermedia* from Borneo and introgressed into *N. crassa* (94).

nit-5

Allelic *nit-4*, q.v.

***nit-6*: nitrate nonutilizer-6**

VIL. Right of *chol-2* (6%). Left of *ser-7* (10%) and *ad-8* (17%) (*PB*; O. C. Yoder, personal communication).

Unable to use nitrate or nitrite as a nitrogen source (185). Lacks nitrite reductase (185) (Fig. 19), which is subject to positive nitrogen metabolite repression (186). Affected by *nit-2* and MS5 regulator genes (838, 1076a) (see *nmr-1*). Used to study repression of nitrate reductase (26) and nonenzymatic reduction of nitrite (185). Induced by nitrite (198).

***nit-7*: nitrate nonutilizer-7**

IIIR. Between *trp-1* (26 to 32%) and *dow* (45%) (D. D. Perkins, unpublished data).

Cannot use nitrate or hypoxanthine as a nitrogen source. Resembles *nit-1*, *nit-8*, and *nit-9* in affecting the molybdenum-containing cofactor common to nitrate reductase and xanthine dehydrogenase (1080, 1081) (Fig. 19 and 24).

***nit-8*: nitrate nonutilizer-8**

IR. Linked to *nit-1* (32%) (*1080*). Right of *mt* (10 to 15%) (D. D. Perkins, unpublished data).

Cannot use nitrate or hypoxanthine as a nitrogen source. Lacks the molybdenum cofactor for nitrate reductase and xanthine dehydrogenase (1080, 1081) (Fig. 19 and 24).

***nit-9*: nitrate nonutilizer-9**

IVR. Right of *nit-4* (9%). Linked to *nit-3* (35 to 38%) (*1080*).

Cannot use nitrate or hypoxanthine as a nitrogen source. Lacks the molybdenum cofactor for nitrate reductase and xanthine dehydrogenase (Fig. 19 and 24). A complex locus with three complementation groups, comparable to *cnxABC* of *Aspergillus nidulans*. (1080, 1081)

***nmr-1*: nitrogen metabolite regulation**

VR. Between *am* (3 to 7%) and *gln-1* (4 to 10%) (*1079*).

Synthesis of nitrate reductase is derepressed on ammonium, glutamate, or glutamine. Hypostatic to *nit-2* and *nit-4*. Prototrophic. Isolated and scored by sensitivity to chlorate in the presence of glutamine. (295, 1079)

MS5 is a possible allele. MS5 is unmapped and is not allelic with *nit-2* or *nit-3* on the basis of two wild-type ascospores from poorly fertile crosses in each case. MS5 is derepressed on glutamine. Levels of nitrate reductase, nitrite reductase, histidase, and acetamidase are elevated in the presence of glutamine and the respective enzyme inducer. Prototrophic. Scored the same as the mutant *nmr-1* and checked by assaying for nonrepressibility of nitrate reductase by glutamine. (838; G. J. Sorger, personal communication)

***NO*: Nucleolus organizer**

VL. Right of terminal *sat* (60, 817) and of terminal translocations *ALS176*, *ALS182*, and *AR190*. Left of *lys-1* and of translocations *AR30*, *AR33*, *AR45*, *NM130*, *AR177*, and *NM183*; thus, left of *caf-1* (60, 817; D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation). *T(OY321)* divides the NO into two portions, each of which retains ability to form a nucleolus (D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation). (*821*)

Genes specifying 5.8S, 17S, and 26S rRNA (but not 5S) are located in the nucleolus organizer region in a tandemly repeated DNA sequence (215, 361). Wild type 74-OR23-1A has 185 tandem repeats (571). Nucleotide sequence of the 5.8S ribosomal DNA has been determined; comparison with yeast cells shows 145 of 158 rRNA residues conserved (974). Hybridization shows that sequences are shared both with *Xenopus* and *Drosophila* (216). *T(AR33)* produces duplications with two copies of the nucleolus organizer (817), which undergo demagnification (887) in such a way that different nontranscribed spacer sequences from both parental nucleolus organizers are retained (888). Genes specifying 5S rRNA are not included in the ribosomal DNA repeat unit, but are located elsewhere in the genome as dispersed single genes surrounded by heterogeneous flanking sequences (361, 975). For rRNA processing, see *rip-1*.

***nt*: nicotinic acid or tryptophan**

VIIR. Between *arg-10* (2 to 12%) and *sk* (7 to 18%) (789). (*874*)

Uses nicotinic acid. May respond also to tryptophan, phenylalanine, tyrosine, quinic acid, and precursors of nicotinic acid or tryptophan, or both, depending on genetic background

(448, 760). Best supplemented with nicotinamide and scored as a *nic* mutant. Probably deficient in tryptophan pyrrolase (tryptophan 2,3-dioxygenase) (Fig. 18), but direct evidence is lacking because tryptophan oxygenase cannot be assayed in *Neurospora* (368). Kynurenine formamidase levels are normal (368). Pyridine nucleotide levels (111).

***nuc-1*: nuclease-1**

IR. Right of *T(AR173)* and *his-2* (<1%). Left of *lys-4* (1%). (*514*)

nuc-1 mutants (other than *nuc-1*^{c}) are unable to use RNA or DNA as a phosphorus source (450, 514). Defective in production of repressible alkaline and acid phosphatases (671, 1077). Several nucleases are absent or reduced (449). *nuc-1* is epistatic to both *pcon*^{c} and *preg*^{c} (671) and to *pgov*^{c} (665). Scored on low-phosphate medium by a staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077), by failure to grow on minimal medium altered so that 0.1 g of RNA or DNA per liter is substituted for the inorganic phosphate source (514, 538), or by failure to grow on low-phosphate medium at a pH above 7 (R. L. Metzenberg, personal communication).

nuc-1^c is constitutive for alkaline phosphatase synthesis and maps very close to *nuc-1*. *nuc-1*^{c} acts only if it is *cis* to normal *nuc-1*. In duplications, *nuc-1*^{c} is dominant to *nuc-1*$^+$, which is dominant to *nuc-1*. *nuc-1*^{c} is epistatic to *nuc-2* (670). *nuc-1*^{c} is scored on high-phosphate medium by a staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077) or by suppression of the *nuc-2* phenotype on low-phosphate medium at high pH (670). Used to study phosphate transport (624). For regulation model see references 665 and 670.

***nuc-2*: nuclease-2**

IIR. Between the *T(NM177)* breakpoints; hence, right of *aro-3*. Left of *preg* (1 to 2%) and *pe* (4%). Probably allelic with *pcon* (0/854) (593, 671). (*514*)

Unable to use RNA or DNA as a phosphorus source (514). Defective in production of repressible alkaline and acid phosphatases (671, 1077). Several nucleases absent or reduced (449). Interaction with other phosphate regulatory genes (665). Recessive to *nuc*$^+$ in partial diploids and heterokaryons (671). Not defective in *nuh* function (538). Scored on low-phosphate medium by a staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077), by failure to grow on minimal medium altered so that 0.1 g of RNA or DNA per liter is substituted for the inorganic phosphate source (514, 538), or by failure to grow on low-phosphate medium at a pH above 7 (R. L. Metzenberg, personal communication). Used to study phosphate transport (624). For a regulation model, see references 665 and 670. See *pcon*.

***nuh*: nuclease halo**

Deficient in extracellular nuclease, giving reduced halos around colonies on DNA agar (538). The mutant *nuh-4* is also sensitive to UV and nitrosoguanidine; the others are not. However, two mutants isolated by UV sensitivity, *uvs-3* and *uvs-6*, also have the *nuh* phenotype (538).

***nuh-1*: nuclease halo**

IIIR. Right of *leu-1* (4%). Left of *nuh-2* (<1%) and *trp-1* (11%) (*538*).

***nuh-2*: nuclease halo-2**

IIIR. Right of *nuh-1* (<1%) and *leu-1* (4%). Left of *trp-1* (11%) (*538*).

***nuh-3*: nuclease halo-3**

VR. Between *cyh-2* (4%) and *al-3* (17%) (*538*).

Releases only small amounts of deoxyribonuclease A (endonuclease) and deoxyribonuclease C (endo-, exonuclease) (359). Not sensitive to UV or chemical mutagens (E. Käfer, cited in reference 195).

***nuh-4*: nuclease halo-4**

Probably allelic with *uvs-3*, q.v. (537, *538*).

***nuh-5*: nuclease halo-5**

IIR. Linked to *trp-3* (30%), near *T(4637)* (*538*).

***nuh-6*: nuclease halo-6**

IR. Between the centromere (5%) and *nic-2* (4%) (*538*).

***nuh-8*: nuclease halo-8**

IR. Right of *nic-1*. (See note added in proof in reference *538*).

Formerly called *nuh(18)*.

***nuh(23)*: nuclease halo**

VR. Linked to *nuh-3* (6%) (*538*).

Nystatin resistant

See *erg*: ergosterol.

***oli*: oligomycin resistant**

VIIR. Between *met-9* (8 to 24%) and *arg-11* (3 to 16%) (960). Linked to *frq-1* (<2%) and possibly allelic (282, 283). (*959*)

Resistant to oligomycin. Defective in energy transduction (313). Structural gene for dicyclohexylcarbodiimide-binding proteolipid (subunit 9) of F_0 portion of mitochondrial adenosine triphosphate synthetase (958). The amino acid sequence (81 residues) (959) shows extensive homology with the corresponding proteolipid in yeast, in which, in contrast to *Neurospora*, it is the product of a mitochondrial gene (960, 1109). Specific single amino acid substitutions have been identified for three mutants (959). *oli* mutants are selected effectively by using double mutant *azs; has* (called ANT-1), which is deficient in both salicyl hydroxamic acid-sensitive and azide-sensitive alternate oxidase pathways. Scored on 5 μg of oligomycin per ml of liquid medium, 3 days, 30°C (312). Altered period of circadian rhythm cosegregates and reverts with *oli* (282, 283).

***os*: osmotic sensitive**

Unable to grow on media with elevated osmotic pressure. Scorable on solid or liquid media plus 4% NaCl (1.4 M). Most alleles can also be scored by morphology, having sticky, close-cropped aerial hyphae that tend to rupture and bleed. Morphology is influenced by humidity. Intense pigment of aggregated hyphae has suggested the name "flame," which was originally applied to some *os* mutants. *os* strains are useful for obtaining protoplasts (e.g., reference 971) and are reported to be efficient as recipients for DNA-mediated transformation in media of high osmolarity (1162). In addition to the numerous loci designated *os*, *cut* is a typical osmotic mutation. Mutant *sor(T9)* is osmotic sensitive and sorbose resistant and has low glucoamylase activity, but does not show the typical *os* morphology.

***os-1*: osmotic-1**

IR. Between *nic-1* (10 to 29%) and *arg-13* (1%) (789, 812, 816). (*M. R. Emerson, cited in reference 789*)

Sensitive to high osmotic pressure. Readily scored by morphology on nonmoist slants or by failure to grow on media with 4% NaCl. Most *os-1* alleles result in cultures that form no or few conidia on agar slants. Alleles NM233t and NM204t are heat sensitive (25°C versus 34°C). In media of high osmolarity, *os-1* strains form protoplasts (323, 438). *os-1* (B135) is an essential genotypic component of the wall-less strain slime (321). Protoplasts of strains carrying heat-sensitive allele NM233t are stable at 37°C, with a 7.5-h redoubling time, and show good regeneration. The biochemical defect differs from that affected by either polyoxin or sorbose (chitin or glucan synthases) (970, 971). Cell wall pores are four times larger in an *os-1* mutant than in the wild type; *os-1* also has a higher exclusion threshold and a 30-fold-higher galactosamine/glucosamine ratio (1083, 1084). Intralocus complementation (676). Allele Y256M209 called *flm-1*.

***os-2*: osmotic-2**

IVR. Right of *cot-1* (4%) (816; *A. L. Schroeder, personal communication*).

Sensitive to high osmotic pressure. Readily scored by morphology on nonmoist slants.

***os-3*: osmotic-3**

Described as a IR mutation right of *nic-2* (4%) (654). Because of stock loss and ambiguity, validity as a separate locus cannot be confirmed (655, 802).

***os-4*: osmotic-4**

I. Right of *T(39311)* and *arg-1* (1%). Left of *T(AR173)* and *T(AR190)*; hence, of *un-2* and *his-2*. Linked to *sn* (0/33). (Data for allele Y256M233.) (*802*, PB)

Sensitive to high osmotic pressure. Readily scored by morphology on nonmoist slants. Allele Y256M223, originally called *flm-2*, is preferred over NM201o, on which the locus designation was initially based. (802)

***os-5*: osmotic-5**

IR. Right of *cyh-1* (12%). Left of the *Tp(T54M94)* right breakpoint and of *arg-6* (1%). Linked to *al-2* (<1%). (*802*, 808)

Sensitive to high osmotic pressure. Scorable by morphology on nonmoist slants.

***os-6*: osmotic-6; *os-7*: osmotic-7**

Symbols used in reference 676 for *os-1*-linked osmotic mutations obtained among *inl*$^+$ transformants in experiments using wild-type *Neurospora* DNA. Osmotic and osmotic-like mutants have also been reported in other transformation experiments (1162). It seems wise not to define new loci on the basis of variants arising in transformation experiments or to use data from transformed strains or their derivatives in mapping. Accordingly, *os-6* and *os-7* have not been included in the list of established loci.

***ota*: ornithine transaminase**

IIIR. Between *ad-4* (15%) and *tyr-1* (14%) (*241*). Linked to *pro-4* (4%) (D. J. West, cited in Neurospora Newsl. **16**:19–22, 1970).

Ornithine-δ-transaminase deficient (241) (Fig. 10). Conidiates somewhat less than does the wild type (S. Brody, personal communication). Selected by ability to use exogenous ornithine as a precursor for arginine in an *arg-5 arg-12*^{s} double mutant. Catabolism of ornithine (to glutamic-γ-semialdehyde) is blocked, resulting in ornithine concentrations high enough to compensate for the low activity of the ornithine carbamyl transferase in the *arg-12*^{s} mutant. The *ota* single mutant is prototrophic but prevents the efficient use of ornithine or arginine as the sole nitrogen source (241). Used to study flux through the arginine biosynthetic pathway (401). Used to study the utilization of endogenous versus exogenous ornithine (234). Sideramine production is completely blocked in absence of ornithine in the *ota; arg-5; aga* triple mutant, which is used to study iron transport (1146, 1147).

***oxD*: D-amino acid oxidase**

IVR. Between the *T(S1229)* breakpoints; hence, right of *pdx-1* (0/55 asci). Left of *met-1* (3%) (55, *768*, 808).

Lacks D-amino acid oxidase. Unable to use D-methionine to satisfy the growth requirement of the mutant *met-1*. Increased sensitivity to toxic effects of D-phenylalanine and D-tyrosine. Unable to use D-methionine as the sole sulfur source (768). Resistant to D-ethionine (477). Strains carrying allele *oxD*^{1} are cysteine auxotrophs, probably owing to a closely linked coincident lesion (768); see *cys-15*.

Oxidase, terminal

See *aod, azs, has*, and *cni-1*.

***pa*: pale**

IR. Between *cr-1* (10%) and *dir* (37%) (*609*, 610).

Conidia sparse, clumped, and pale. Photograph (610). (Stock lost. Possibly *wc-2*?)

***pab-1*: *p*-aminobenzoic acid-1**

VR. Between *inl* (1 to 10%) and *met-3* (1 to 2%) (362, 1036). (*47*)

Requires *p*-aminobenzoic acid (1057) (Fig. 11). Apparently cannot use folate (mono- or triglutamate) (1179).

***pab-2*: *p*-aminobenzoic acid-2**

VR. Right of *ad-7* (8%). Left of *inv* (3%) and *asn* (1 to 15%). Linked to *ro-4* (0/407) (156, 816, 818, 918, 1036). (*47*)

Requires *p*-aminobenzoic acid (1182) (Fig. 11). Allele 71301 called *pab-3* (1182); shown to be allelic by Drake (289).

pab-3

See *pab-2*.

***pan-1*: pantothenic acid-1**

IVR. Between *ad-6* (1 to 2%) and *cot-1* (2 to 3%) (633, 692, PB). (*482*). *cel, col-1, int, pho-3*, and *thi-5* all appear to be closely linked in this crowded region.

Requires intact pantothenic acid for growth under standard conditions. Able to synthesize both precursors, β-alanine and pantoyl lactone (1058). Ability to synthesize pantothenic acid from β-alanine plus pantoyl lactone is demonstrable in vitro but not in vivo unless cultures are aerated (1111, 1113, 1114). Unlike *pan-2, pan-1* has no effect on ascospore ripening in heterozygous crosses. Called group A. For alleles see reference 138.

***pan-2*: pantothenic acid-2**

VIR. Right of *rib-1* (<1 to 3%). Left of *del* (6%) and *trp-2* (11%) (140, *141*, 143, 818, PB).

Unable to convert ketovaline to ketopantoic acid (138, 140, 141). Used in major studies of intralocus recombination and complementation (140–143). *pan-2* ascospores remain white or

pale if the crossing medium is not supplemented, even when the protoperithecial parent is *pan-2*$^+$. Asci in which gene conversion has occurred at *pan-2* can thus be recognized and isolated (1072, 1073); photographs (1072). For good recovery of *pan-2* progeny, crossing media should be supplemented with pantothenic acid (10 μg/ml) even when the protoperithecial parent is *pan*$^+$. Called group B.

***pat*: patch**

IL. Linked to *mt*, probably to the right (*1014*).

Growth and conidiation occur in patches, in a cyclic pattern under certain conditions (1014). Initially found in a *pro-1* (21863) stock; *pro-1* is not necessary for expression of patch. Original patch isolates were all sorbose resistant (1014), but a sorbose-resistant derivative has been obtained that apparently does not express the patch phenotype; this is called *sor-4*, q.v. (816). It is not clear whether *pat* and *sor-4* are separate genes or whether patch is not scorable in the absence of modifiers present in the parent stock; see p. 267 of reference 816. The original patch strain was used for the first demonstration of a circadian rhythm in fungi (104, 829), but *bd* strains are now preferred.

***pcon*: phosphatase control**

IIR. Right of the *T(NM177)* left breakpoint. Left of *preg* (1 to 2%) and *pe* (4%). Probably allelic with *nuc-2* (0/854) (*593*, 671).

Regulator gene of repressible alkaline phosphatase (671) and other steps in phosphorus uptake and metabolism (665; R. L. Metzenberg, personal communication). Constitutive allele *pcon*^{c} is dominant to, or codominant with, *pcon*$^+$ (671). Scored on high-phosphate medium by staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077). Used to study phosphate transport (624). *pcon*^{c} allele c-6 called *UW-6* in reference 593. See *nuc-2*. For regulation model, see references 665 and 670.

***pdx-1*: pyridoxine-1**

IVR. Right of *pyr-1* (<1 to 10%). Left of *T(S1229)* and *pt* (2%) (40, 55, 692, 808). (*482*)

Uses pyridoxine, pyridoxal, or pyridoxamine (843, 845, 846). Shows intralocus complementation (845, 846) and recombination (848). Provided the first proven example of gene conversion (686). Scoring is sharpened by addition of 100 mg of desoxypyridoxine per liter (845). Several alleles (called *pdxp*: e.g., 44602) are pH sensitive and can grow without pyridoxine on medium containing ammonium ions at a pH above 6 (1029). Conidia are subject to death by unbalanced growth on minimal medium (1033). A yellow pigment is excreted under certain conditions by the *pdx-1; En(pdx)* double mutant; see *En(pdx)*. Allele 44204 originally called *pdx-2* (see reference 848).

pdx-2

See *pdx-1*.

***pe*: peach**

IIR. Between *nuc-2* (4%) and *arg-12* (1 to 5%) (593, 816). (*613*)

Peach-colored conidia and short hyphae formed, more uniformly than by the wild type, as a lawn close to surface of agar. Distinctive morphology (46, 613). Added arginine increases macroconidiation and tends to obscure scoring of *pe* at 25°C, but not at 39°C. *pe* single mutants produce both macro- and microconidia. *pe fl* double mutants produce abundant grey microconidia and no macroconidia (46, 700) (see *fl*). See *col-1*, *col-4*, and references 415 and 416 for interactions with other genes. Called *m* (microconidial) or *pe*^{m} in some contexts.

***pen-1*: perithecial neck-1**

Unmapped.

Perithecia lack beaks (necks) when the *pen-1* mutant is used as the female; perithecia are normal when the *pen-1* mutant is used as the male to fertilize a *pen*$^+$ strain (253).

***per-1*: perithecial-1**

VR. Right of *asp* (26%) and *at* (8 to 14%). Left of *ilv* (4%) (*489*, PB) and *ts* (25%) (*527*).

Perithecial walls are devoid of black pigment when the female parent carries *per-1*, regardless of genotype of the fertilizing parent (489, 490, 527). Alleles are of two types (490). Type I produces young, completely white perithecia that become pale yellowish after several days, and *per-1* ascospores are white (e.g., alleles PBJ1, ABT8, and AR174). Type II produces mature perithecia that are somewhat darker orange with black pigment in the neck, and *per-1* ascospores are normal black (e.g., alleles 29-278, 29-281, and UG1837). Unlike the perithecial

wall trait, the ascospore trait shows no maternal effect. Black pigment develops in a ring around the ostiole of type II perithecia, but is pale or lacking in type I perithecia (490). Mosaic perithecia from heterokaryons have been used for a clonal analysis of perithecial development (527, 528). Expression is completely autonomous in ascospores (photographs in reference 529) and at least partially so in the perithecial walls (527–529). Used to test for variegated-type position effect, with negative results (532). White *per-1* ascospores (type I) germinate without heat shock and are usually killed by hypochlorite or by the 30-min, 60°C treatment used to activate normal ascospores (490, 527). Beaks of perithecia homozygous for allele PBJ1 (type I) are abnormal, and ascospores are not shot properly (N. B. Raju, personal communication). Type I alleles initially called *sw*: snow white (527).

Perithecial development mutants

See *fs, ff, fmf, mb, mei, pen*, and *per*.

Permease

See Transport.

***pf*: puff**

IVR. Right of *pyr-2* (2%). Linked to *mat* (3%) (*812*, 991).

Spreading colonial morphology (812).

***pgov*: phosphorus governance (provisional name)**

IIIR. Linked to *tyr-1* (1 to 4%), probably to the right (*R. L. Metzenberg, personal communication*).

Regulatory gene for phosphorus uptake and metabolism. $pgov^c$ phenotype similar to that of $preg^c$ (665; R. L. Metzenberg, personal communication). Isolated in $preg^+/preg^+$ partial diploids. *pgov* allele c-5 is largely or completely recessive in duplications from *T(D305)* (R. L. Metzenberg, personal communication). Scored on high-phosphate medium by staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077). For regulation model, see references 665 and 670.

***phe-1*: phenylalanine-1**

IL. Right of *In(H4250)* and suc (<1%). Left of *ad-5* (816; H. B. Howe, Jr., personal communication). (*48*) [Duplications from *In(H4250)* × *phe-1* crosses are phe^-, unlike duplications from *T(39311)*. The contrary statement on p. 268 of reference 816 is a misprint.]

Originally reported to grow on phenylalanine, other aromatic amino acids, leucine, or ethyl acetoacetate, with phenylalanine being most effective; several other acids gave smaller responses (48). Utilization of phenylalanine and other compounds varies for different isolates and on different carbon sources; glycerol or ribose is preferable to sucrose (521–523, 753). Strains carrying allele NM160 do not use phenylalanine but grow well on tyrosine or leucine (816; A. G. De Busk, personal communication), at least with the strains and carbon source used. The mutant *phe-1* is inhibited by basic amino acids on low-phenylalanine or leucine medium (48, 521). Growth on β-labeled leucine or β-labeled phenylalanine showed that neither compound is converted to the other (45). Called *phen-1*. Allele NM160 originally called *tyr(NM160)* (316).

***phe-2*: phenylalanine-2**

IIIR. Linked to *vel* (1%), between *T(D305)* and *tyr-1* (2 to 4%) (*40, 316*, 809).

Lacks prephenic dehydratase (40, 316) (Fig. 11). Requirement is very leaky. Grows extensively and is treacherous to score by growth on minimal versus supplemented medium, but can be scored reliably by blue fluorescence under long-wave UV after growth on minimal medium without phenylalanine (816). Appearance of phenylalanine in culture medium (118) is due to spontaneous conversion of accumulated prety-rosine (519). Called *phen-2*. Allele Y16329 formerly called *phen-3* (316).

***phe-3*:**

See *phe-2*.

phen

Changed to *phe*.

***pho-1*: phosphatase-1**

Possibly II. 20% wild-type recombinants with *nuc-2*. Independent of *nuc-1*.

Low activity of the repressible alkaline phosphatase. Recessive. Complements *nuc-1* and *nuc-2* in heterokaryons. Stains pale red on low-phosphate medium with α-naphthyl phosphate plus diazo blue B. (1077)

***pho-2*: phosphatase-2**

VR. Between *his-1* (3%) and *inl* (4%) (*397*; R. L. Metzenberg, personal communication).

Structural gene for repressible alkaline phosphatase (397, 594, 745). Scored on low-phosphate medium by staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077).

***pho-3*: phosphatase-3**

IVR. Linked to *pan-1* (<1%). Right of *leu-2* (8%). Left of *mat* (18%) and the *T(NM152)* right breakpoint (*745*).

Structural gene for repressible acid phosphatase with phosphodiesterase activity (745). Codominant in heterozygous *pho-3*/*pho-3*$^+$ duplications. Apparently a member of both phosphorus and nitrogen regulatory circuits. Not under control of the *nit-2* locus (278b). Scored on low-phosphate medium by staining reaction with bis-nitrophenylphosphate (745).

***pi*: pile**

IIL. Linked to *ro-7* (0/75); left of *cys-3* (4%) (*816*).

Spreading growth (allele B101); no conidia. (816) Growth is better on minimal medium than on complex complete medium. The mutant *pi* (B101) has not been tested for 6-phosphogluconate dehydrogenase, which is deficient in strain(s) carrying putative allele *col-10* (R2438) (947). See *col-10* regarding allelism. As a marker, *pi* (B101) is preferable to *col-10* (R2438) because of growth rate, stability, and ease of handling. *ro-7*, in the same region, is preferable to both (PB).

***pk*: peak (synonym: *bis*, biscuit)**

VR. Between *met-3* (1%) and *cot-2* (8%). Left of *cl* (2%) (296, 818, *1036*, PB).

Initially colonial with flat surface growth; then sending up a mass of aerial hyphae which conidiate profusely (789). Morphology somewhat similar to that of *sn*, *cum*, *sp*, and *cot-4* mutants at 25°C, but distinguishable. Hyphae branch dichotomously (713, 789). Asci are thin-walled, bulbous, nonlinear in homozygous *pk* × *pk* crosses (714, 717, 792). Most alleles are recessive for the ascus effect, but some are dominant. Sorbose resistance mutations at various loci act as dominance modifiers of the ascus effect of dominant alleles (898). Increased activity of L-glutamine:D-fructose-6-phosphate amidotransferase was observed in crude extracts of one *pk* strain but not in nine others; increased activity for this enzyme was also found in *cl* (see below) and in four other nonallelic morphological mutants (899). Hexoseaminoglycan consists of a single component on medium without sorbose, in contrast to two components for the wild type (1003). Antigenic surface mucopolyoside (281). Cell wall analysis and photograph, allele B6 (278), allele C1810-1 (132). Cell wall enzymes (334). Effect of carbon sources (280). One observation suggests functional interaction (possible allelism) with *cl* (1007), but substantial crossing-over frequencies and recovery of the *pk-2 cl* double mutant favor separate loci (296). Several alleles have been previously called *bis* (for nomenclature, see p. 270 of reference 816). Allele C-1610, originally called *pk-1*, is inseparable from a reciprocal translocation, *T(I;V)C-1610 pk* (808). Dominant allele 17-088 is associated with a V;VII translocation (A. M. Srb, personal communication; PB).

***pl*: plug**

VR. Linked to *gran* (0/75); between *asn* (1 to 9%) and *his-6* (16%) (812, 816, *1036*).

Dense hyphae fill diameter of 10-mm tube (789). Morphologically distinct from the mutant *gran*. Complex complete medium stimulates conidiation; scoring of morphology is clearer on minimal medium.

***pmb*: permease basic amino acid**

IVR. Right of *uvs-2* (8%) (*S. Ogilvie-Villa, cited in reference 248*; R. Sadler and S. Ogilvie-Villa, personal communication).

Defective in basic L-amino acid transport (system III as defined in reference 778); reduced uptake of L-arginine, L-lysine, and L-histidine (778, 1152, 1076). Used extensively for transport studies; see reference 1150. Altered surface glycoprotein (1038). *pmb* mutants selected as resistant to L-canavanine (889, 1152). Allelic with *bat* (R. Sadler and S. Ogilvie-Villa, personal communication), which was selected in *arg-12*^{s}; *pyr-3* (CPS$^-$ ACT$^+$) by ability to grow on minimal medium plus arginine, when the parental double mutant was not able to grow because of arginine uptake and feedback onto the arginine-specific carbamyl phosphate synthase (1074). Possibly allelic with *bas*^{a}, which was selected by the inability of the mutant *his-3* to grow on histidine plus methionine (628). Probably allelic with *bm-1* (linked to *pyr-2*, 24%), which was selected by canavanine resistance (913). Probably allelic

with arg^R (565, 566), q.v. Called *Cr-10, Pm-B, pm b, UM-535*, and *can-37*. See Transport.

***pmg*: permease general amino acid**

Not mapped. Centromere linked. Not linked to *mtr* (*pmn*) or to *pmb* in IVR (248). (Previous report of mating type linkage [862] not confirmed).

Greatly reduced in general amino acid transport system II (as defined in reference 777). Reduced uptake of arginine and phenylalanine. Selected by resistance to *p*-fluorophenylalanine in a neutral (system I), basic (system III) double mutant on medium lacking ammonium ions, where system II would be derepressed in the wild type (248, 862, 863). A non-metabolizable substrate specific for this transport system has been found (767). Called *pm g*. See Transport.

***pmn*: permease neutral amino acid**

Allelic with *mtr* (R. Sadler and S. Ogilvie-Villa, personal communication; A. G. De Busk, personal communication), q.v. Called *Pm-N* and *pm n*.

[*poky*]: poky (synonym [*mi-1*])

Mitochondrial mutant with slow growth and deficient cyanide-sensitive respiration (see reference 394). See *su([mi-1])*.

***pp*: protoperithecia**

No data on linkage or allelism with already mapped loci that affect perithecial formation or ascospore viability (e.g., *gul-3, -4, -5; le-1, -2; ff; fs*).

Female sterile. Protoperithecia not formed. Ascospore lethal. Enhances growth of the mutant *gpi* on glucose or sucrose. (711)

***prd-1*: period-1**

III. Linked to *acr-2* (5%), the centromere (0/35 asci), and *pro-1* (20%) (*327*; G. F. Gardner, personal communication).

Altered period of circadian conidiation rhythm. One allele is known, which results in a 25.8-h period (at 25°C without *csp*) (327, 375). Recessive. Grows at 60% of wild-type rate. See reference 327 for period lengths of double mutants carrying *frq-1, -2*, and *-3*. Temperature compensation described (377). Name changed from *frq-5* (375). For reviews of circadian mutants see references 326 and 328.

***prd-2*: period-2**

VR. (*377*)

Altered period of circadian rhythm (25.5 h at 25°C without *csp* for allele IV-2). Slower-than-normal growth. Recessive (329). Temperature compensation described (377). Called IV-2.

***prd-3*: period-3**

I. Near centromere (*377*).

Altered period of circadian rhythm (25.1 h at 25°C without *csp* for allele IV-4) (329). Slower-than-normal growth. Recessive. Temperature compensation described (377). Called IV-4.

***prd-4*: period-4**

Unmapped. Not allelic with *frq* or *prd-1, -2*, or *-3* (329).

Altered period of circadian rhythm (18.0 h at 25°C without *csp* for allele V-7) (329). Normal growth rate. Dominant. Temperature compensation described (377). Called V-7.

***preg*: phosphatase regulation**

IIR. Right of *nuc-2* (*pcon*) (1 to 2%), q.v. (*671*).

Regulator of repressible alkaline phosphatase and other steps in phosphorus uptake and metabolism. Hypostatic to *nuc-1*, epistatic to *nuc-2*. $preg^c$ constitutive mutation is recessive to its wild-type allele. (665, 671; R. L. Metzenberg, personal communication) Scored on high-phosphate medium by staining reaction with α-naphthyl phosphate plus diazo blue B (397, 1077). Used to study phosphate transport (624). For regulation model, see references 665 and 670.

***pro-1*: proline-1**

IIIR. Between *ser-1* (3/38 asci) (504) and *ace-2* (1 to 9%) (578). Right of *sc* (7%) (814). (*482*)

Uses proline but not ornithine, citrulline, or arginine (1009). Structural gene for pyrroline-5-carboxylate reductase (1177) (Fig. 10).

***pro-3*: proline-3 (synonym: *arg-8*)**

VR. Linked to *inl* (0/74). Between *his-1* (4%) and *pk* (2 to 6%). (*812*)

Uses proline, ornithine, citrulline, or arginine (1006). Blocked in the proline pathway, in reduction of glutamic acid to glutamic γ-semialdehyde (1105). Arginine and citrulline are used via the arginine catabolic pathway (arginase and ornithine transaminase), and ornithine is used via ornithine transaminase (151, 234, 1104, 1105) (Fig. 10). Tends to accumulate second mutations, including *arg-2* and *his-1* (58, 994). Ability to grow on arginine is modified by *ipa* (994) and *ota* (234). Suppressed by *su(pro-3)*, which is allelic with or closely linked to *arg-6* (1129). Called *arg-8*.

***pro-4*: proline-4 (synonym: *arg-9*)**

IIIR. Linked to *thi-2* (0/78) and *ota* (4%) (818; *D. J. West, cited in Neurospora Newsl. 16:19–22, 1970).*

Uses proline, ornithine, citrulline, or arginine (1009). Proline pathway blocked in reduction of glutamic acid to glutamic γ-semialdehyde (1105) (Fig. 10). Citrulline and arginine are used via the arginine catabolic pathway (arginase and ornithine transaminase), and ornithine is used via ornithine transaminase (151, 1104, 1105). Leaky. Called *arg-9*.

prol

Changed to *pro*.

***prt*: protease**

See *pts*.

***psi-1*: protein synthesis initiation-1**

IVR. Right of the centromere (*D. R. Stadler, A. M. Towe, and M. Loo, cited in reference 619*). Left of *T(ALS159)* and *pyr-1* (4%) (808, PB).

Conidial germination and hyphal growth inhibited at 35°C but normal (or nearly so) at 20°C. Protein synthesis reduced after shift to restrictive temperature. Recessive in heterokaryons. (619) Scored as an irreparable heat-sensitive *un* mutant (see *un*).

***pt*: phenylalanine plus tyrosine**

IVR. Right of *T(S1229)* and *pdx-1* (2%). Left of *col-4* (2%) (40, 55, 808). (*201*) Original *S4342* strain contained linked but separable insertional translocation *T(S4342)* (808), the presence of which should not change conclusions regarding gene order given in reference 40.

Requires phenylalanine plus tyrosine (201). Lacks chorismate mutase (40, 316) (Fig. 11). Evidently the structural gene; strains carrying allele NS1 have thermolabile chorismate mutase (D. E. A. Catcheside, personal communication). NS1 strains are temperature sensitive, growing on minimal medium at 25°C, where they are readily scorable by blue fluorescence under long-wave UV and by browning of medium of aging cultures (1035). Inhibited by complex complete medium.

***pts-1*: protease-1**

Unmapped. Segregates as single gene not closely linked to *alcoy* markers (40 isolates) (441).

Structural gene for carbon-, nitrogen-, and sulfur-controlled extracellular alkaline protease. Allele found in a single wild-collected strain, Groveland-1c *a*, FGSC no. 1945. Synthesizes fast electrophoretic variant under conditions of limiting carbon, nitrogen, and sulfur. Called *prt* (441). Regulation reviewed in references 642 and 665.

Purine

See *ad* and *gua*.

***put-1*: putrescine-1**

Changed to *spe-1*, q.v.

***pyr*: pyrimidine**

All pyrimidine auxotrophs of *Neurospora* are nonspecific, responding to any pyrimidine nucleoside, nucleotide, or base. The symbol *pyr* is, therefore, used for genes concerned with the biosynthetic pathway. Nucleosides or nucleotides are more effective than corresponding bases as growth supplements for the mutant *pyr-1* (623) and apparently for other *pyr* mutants. However, after a lag, uracil is used nearly as effectively as uridine (683). No cytidine or thymidine-specific requirement exists, because *Neurospora* lacks thymidine kinase (421) and

because any exogenous pyrimidine supplement is cycled back through uridine monophosphate, which provides all the normal end products of pyrimidine biosynthesis (1141). For this reason, DNA cannot be specifically labeled by supplying [^{3}H]thymidine, under normal circumstances. Mutations have been obtained (*uc-2, -3, -4, -5; ud-1*) that block the pathway back through uridine monophosphate and so prevent general labeling from a single precursor (1141). Cytosine in DNA can be labeled specifically by the method of Worthy and Epler (1163). For a general review of pyrimidine metabolism, see reference 766. For systematic gene-enzyme work, see references 133 and 134. For pyrimidine biosynthetic pathway, see Fig. 20. For loci concerned with pyrimidine salvage or pyrimidine uptake, see *uc, ud, udk*, and Fig. 23. Complex interactions between *lys* and *pyr* mutations have been described (485).

Pyrimidine biosynthetic enzymes differ in their modes of regulation. The pyrimidine-specific carbamyl phosphate synthase–aspartate carbamyl transferase complex is derepressed by end product depletion, but is insensitive to repression in the fluoropyrimidine-resistant mutant *fdu-2* (127, 135); dihydroorotase is relatively unresponsive to end-product limitation; and dihydroorotate dehydrogenase is induced by a precursor which is probably, by analogy with *Saccharomyces*, dihydroorotate, the substrate of the enzyme. Regulation of the last two enzymes has not been studied systematically. Pyrimidine regulation of the uptake and salvage pathways of pyrimidine is discussed under individual loci; see *uc-5* and *ud-1*. Many aspects of pyrimidine metabolism are under the control of general nitrogen metabolite regulation (128).

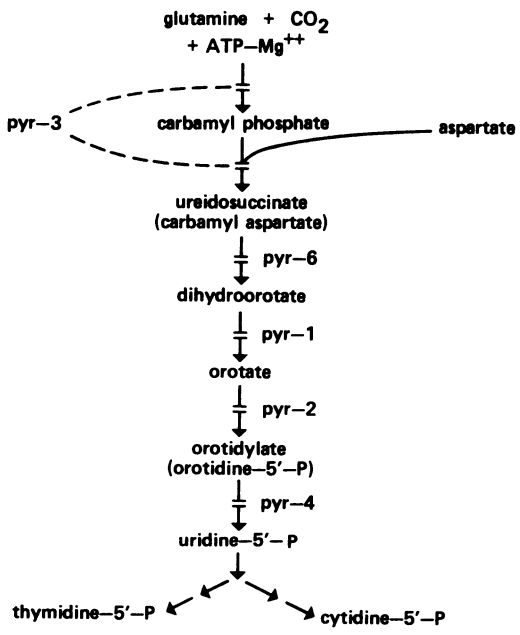


FIG. 20. Pyrimidine biosynthetic pathway, showing sites of gene action (134, 456, 841, 1140). Carbamyl phosphate for arginine synthesis is made as a separate pool by a different enzyme system (see *arg-2, arg-3*, Fig. 10). Interchange between the two pools occurs only in certain mutant combinations. ATP, Adenosine 5′-monophosphate.

***pyr-1*: pyrimidine-1**

IVR. Right of *psi* (4%) and *T(ALS159)* (808, PB). Left of *pdx-1* (<1 to 10%) (692). (*482*)

Requires uracil or other pyrimidine. Lacks dihydroorotate dehydrogenase activity (133, 134) (Fig. 20). All ascospores from *pyr-1* × *pyr-1* crosses are white if cross is on medium containing 0.1 mg of uracil per ml; they are black if the cross is on medium containing 1.0 mg/ml (632).

***pyr-2*: pyrimidine-2**

IVR. Right of *nit-3* (2 to 9%). Left of *mat* (3%) and the *T(NM152)* right breakpoint (633, 812, 1000, PB). (*482*)

Requires uracil or other pyrimidine. Lacks orotidine 5′-monophosphate pyrophosphorylase activity (133, 134) (Fig. 20). Needs medium containing >0.5 mg of uracil per ml for optimal growth. Allele 38502 is leaky.

***pyr-3*: pyrimidine-3**

IVR. Right of the *T(NM152)* left breakpoint and of *T(S1229)*; hence, right of *arg-14*. Left of *his-5* (1%) (238, 808). (*482*)

Requires uracil or other pyrimidine (683). Growth inhibited by purine nucleosides and nucleotides (825). Structural gene for pyrimidine-specific carbamyl phosphate synthase (CPS) and aspartate carbamyl transferase (ACT; also abbreviated ATC) (456, 850) (Fig. 20). Mutants may lack either or both activities, e.g., those carrying alleles KS43 (CPS^+ ACT^-), KS20 (CPS^- ACT^+), and KS11 (CPS^- ACT^-) (1140). Unlike *Saccharomyces*, no feedback-insensitive CPS^+ ACT^+ mutants of *Neurospora* have been discovered (A. Radford, unpublished data). Some mutants have kinetically altered aspartate carbamyl transferase (456, 880). Used extensively for studies of channeling and relation of gene structure to the two enzyme activities (236).

Normally, carbamyl phosphate produced by *pyr-3*$^+$ is used solely for pyrimidine synthesis, and carbamyl phosphate produced by *arg-2*$^+$ and *arg-3*$^+$ is used for arginine synthesis, the enzymes being in different organelles; however, a deficiency of the next enzyme in either pathway permits overflow of carbamyl phosphate into the other pathway (reviewed in reference 236). Hence, CPS$^-$ ACT$^+$ alleles are suppressed by *arg-12*^{s} (246), and CPS$^+$ ACT$^-$ alleles can be selected as suppressors of *arg-2* and *arg-3* (658, 887, and references therein). Some of the CPS$^+$ ACT$^-$ mutations, called *pyr*$^{su\text{-}arg}$, suppress the arginine requirement but retain enough aspartate carbamyl transferase activity that they have no detectable pyrimidine requirement (877, 881). *arg-13*, *arg-4*, *arg-5*, *arg-6*, and *am* partly suppress CPS$^-$ ATC$^+$ alleles (see reference 660). Fine-structure map (851, 1050). Fertility of interallelic crosses is variable and often very poor (658). Complementation between CPS$^-$ ACT$^+$ and CPS$^+$ ACT$^-$ mutants (246) and between some pairs of CPS$^+$ ACT$^-$ mutants is good; otherwise, complementation is poor (849, 1159). Complementation maps (658, 849, 877, 1159). Mutational analysis (852). Direction of translation, based on enzyme types of polar mutants, is from CPS to ACT (850). Allele 37815(t) is heat sensitive (34°C versus 25°C) (68). Allele 1298 is CO_2 remediable (191, 192). Strain KS12, a *pyr-1 pyr-3* double mutant, was originally called *pyr-5* (see reference 346). The different classes of *pyr-3* alleles have been called M (CPS-P-less), N (ACT-less), and MN (lacks both activities).

pyr-4: pyrimidine-4

IIL. Right of *het-c* (1%), *T(P2869)*, and *cys-3* (18 to 21%). Left of *ro-3* (1 to 2%) (721, 816, PB). (*812*).

Requires uracil or other pyrimidine. Lacks orotidine 5'-monophosphate decarboxylase (133, 134, 841) (Fig. 20). Fertile crosses homozygous for *pyr-4* can be made by using very high levels of uridine (15 to 20 mg/ml) (O. M. Mylyk, personal communication).

pyr-5: pyrimidine-5

A *pyr-1 pyr-3* double-mutant strain was originally called *pyr-5* (346).

pyr-6: pyrimidine-6

VR. Between *asn* (6%) and *un-9* (2%) (156, 818). (*133*, *134*)

Requires uracil or other pyrimidine. Lacks dihydroorotase activity (133, 134) (Fig. 20). On a small amount of uridine, a strain(s) carrying the only allele (DFC37) grows, after a pause, well beyond the level normally supported by the supplement; at no time is dihydroorotase detectable (134).

Q, *q*: quinolinic acid

See *nic-1*.

qa: quinate utilization

Gene cluster located in linkage group VII (177, 884), in the order: *qa-1* (~0.02%) *qa-3 qa-4 qa-2* (0.02%) *met-7* (146). No recombination with the centromere has been observed in several hundred asci; *qa* has been tentatively shown right of the centromere, on the basis of close linkage to *met-7* (146; M. E. Case, personal communication). The gene cluster functions in the quinate catabolic pathway. Mutants selected by inability to use quinate as their sole source of carbon (178, 179, 885). The enzymes of the quinate pathway are induced when quinate is present in the medium; but see also reference 423 and references therein. Scored by the ability to use quinic acid (0.3%) as the sole carbon source (146). *qa-1* encodes a regulatory protein; the other *qa* genes encode inducible enzymes (179). The first and second steps of the quinate pathway are paralleled by comparable reactions in the aromatic biosynthetic pathway (Fig. 11). Thus, the enzymes encoded by *aro-9* and *qa-2* can substitute for each other, and under appropriate conditions the *qa-3* enzyme can substitute for the *aro-1* enzyme (147). Separate transcripts are made by components of the *qa* cluster, rather than a single polycistronic messenger RNA (387, 781). For a diagram of the catabolic pathway and its relation to the biosynthetic pathway, see references 146, 387, and 423 and Fig. 21. The entire cluster has been cloned in *E. coli* and returned to *Neurospora* by transformation (941). The cluster consists of adjacent coding sequences totaling about 18 kilobases. A transcriptional map has been constructed, and two new genes of unknown function, *qa-x* and *qa-y*, have been identified from messenger RNAs (781). For regulation, see references 179, 423, 781, and 873.

qa-1: quinate-1

VII. Left of *met-7* (0.2%). Leftmost gene in the *qa* cluster (146). (*884*)

Unable to use quinate or shikimate as the sole carbon source. Quinate catabolic pathway regulatory gene (179). *qa-1* mutants (recessive) are

deficient in all three enzymes of the pathway: quinate dehydrogenase (shikimate dehydrogenase), catabolic dehydroquinase, and dehydroshikimate dehydrase (Fig. 21) (885). *qa-1*^{F} and *qa-1*^{S} are fast- and slow-complementing alleles which appear to define two nonoverlapping segments of the gene. Strains carrying *qa-1*^{C} alleles are constitutive producers of the three enzymes; these are readily found among revertants of *qa-1* (145a, 178, 387). Expression of *qa-1* appears constitutive but also autoregulated (781).

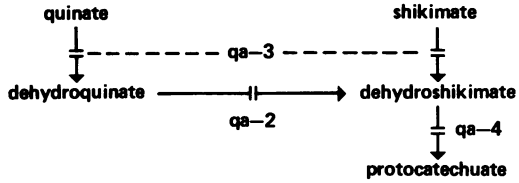


FIG. 21. Quinate (aromatic amino acid) catabolic pathway, showing sites of gene action (146, 148, 387, 423, 517, and references therein). *qa-1* is a regulatory gene affecting all three structural genes. *qa-2*$^+$ activity (catabolic dehydroquinase) can be replaced by the product of *aro-9*$^+$, the equivalent gene in the aromatic biosynthetic pathway. See reference 423 for the catabolic steps subsequent to protocatechuic acid.

_qa-2_: quinate-2

VII. In the *qa* cluster between *qa-4* and *met-7* (0.02%) (146). (*884*)

Lacks catabolic dehydroquinase (148, 517, 884). Unable to use quinate or shikimate as the sole carbon source in the presence of *aro-9*, which results in the absence of biosynthetic dehydroquinase (885) (Fig. 21). *aro-9; qa-2*$^+$ strains grow on minimal medium without supplement. *qa-2* is conveniently scored as an *aro* auxotroph when *aro-9* is present. *qa-2*$^+$ cloned in pBR322 (pVK57) is expressed constitutively from its own promoter in *E. coli* (23, 388, 451); has been returned to *Neurospora* chromosomal sites by transformation (150). Allele M246 is stable (150).

_qa-3_: quinate-3

VII. In the *qa* cluster between *qa-1* and *qa-4* (146). (*177*)

Unable to use quinate or shikimate as the sole carbon source (178). Structural gene for quinate (shikimate) dehydrogenase (178) (Fig. 21). Revertants have altered enzymes. Transcribed in the direction *qa-4* to *qa-1* (149).

_qa-4_: quinate-4

VII. In the *qa* cluster between *qa-3* and *qa-2* (146). (*177*)

Unable to use quinate or shikimate as the sole carbon source (178). Lacks dehydroshikimate dehydrase (178) (Fig. 21).

qa-x, qa-y

See *qa*.

_R_: Round spore

IR. Right of *T(NM169d)* and *aro-8* (4%). Left of *un-18* (11%) (1093). (*H. R. Cameron, personal communication*)

All eight ascospores of heterozygous *R*/+ asci are round rather than ellipsoid. *R* is thus nonautonomous in ascospores and dominant in the ascus (690). Ascospores are round even in nonlinear asci (1008; D. D. Perkins, unpublished data). Usually two germination pores are formed, but sometimes one (1008). Photograph (1008). Vegetative morphology abnormal, somewhat resembling that of *pe* mutants. Initial growth on slants is concentrated around the inoculation point. The vegetative morphology is recessive in heterozygous duplications, as from *T(NM103)* (1091). Female sterile, with no perithecia, but *R* × *R* crosses can be accomplished if *R* is heterokaryotic in the female parent (A. M. Srb, personal communication). Used in studies of duplication instability (1091) and autonomy of ascospore development (529). Allelic recurrences obtained (1008; A. M. Srb, personal communication). For other genetically determined round ascospores, see reference 59. Called *Rsp* (1008), but the original symbol *R* has priority. (*rsp* has been used for cytoplasmically determined respiration-deficient mutants [890].)

_r(Sk-2)-1_: first locus resistant to Spore killer-2

IIIL. Left of *acr-7* (7%) and *sc* (17%) (*1092*; B. C. Turner, personal communication). Allelism of *r(Sk-2)-1* with *Sk-2* is not excluded because *Sk-2*^{K} blocks recombination in this region. *r(Sk-2)-1* itself does not block recombination.

Prevents killing of ascospores by *Sk-2*^{K}. Not resistant to killing by *Sk-3*^{K}. Allele P527 found in *N. crassa* from Louisiana. Only one other known *N. crassa* wild type is resistant. (B. C. Turner, personal communication)

Radiation sensitive

See *mus, uvs*. See also *gs, Mei-2, mei-3, nuh-*

4, and *upr-1*. The symbol *rad* has not been used in *Neurospora*.

rDNA: ribosomal DNA

Used to designate genes specifying 5.8S, 17S, and 26S rRNA, which are located in tandemly repeated units in the nucleolus organizer region. See *NO*.

***rec*: recombination**

A class of genes affecting meiotic recombination in specific loci or regions (175, 520). Initially detected by changed intralocus recombination (up to 25×), but interlocus crossing over may also be affected (up to 40×). Polarity of intralocus recombination may be changed or reduced by the presence of rec^+. Any given target locus or region appears to be affected by alleles at only one specific *rec* locus (Fig. 22). High recombination is recessive. Products of dominant alleles (called rec^+) are thought to repress initiation of recombination at specific recognition (*cog*) sites by binding to an adjacent *con* (control) site. Control of recombination is independent from control of gene expression (153). Allelic differences are present in commonly used laboratory wild types (174). For reviews, see references 167, 169, 170, and 343.

***rec-1*: recombination-1**

VR. Between *ro-4* (7%) and *asn* (5%) (159). (*165*)

Presence of allele $rec\text{-}1^+$ reduces recombination within the loci *his-1* (VR) (520, 1070) and *nit-2* (IL) (155, 157) (Fig. 22). A recessive allele from another lineage was called *rec-z* until probable identity with *rec-1* was established (157). $rec\text{-}1^+$ does not affect recombination within any other *his* locus tested (172).

***rec-2*: recombination-2**

VR. Between *sp* and *am* (174). (*993*)

Presence of dominant allele $rec\text{-}2^+$ reduces recombination within the *his-3* locus (IR); also reduces crossing over in the intervals *pyr-3–his-5* (IVR), *his-3–ad-3* (IR), and *arg-3–sn* (IL) (171, 174, 992) (Fig. 22). Interacts with *cog* in affecting recombination in *his-3* and crossing over between *his-3* and *ad-3* (27, 171). Used in conjunction with translocation TM429 to demonstrate the *cis* action of cog^+ on recombination between sites in *his-3* (171). (See *cog*.) Recessive *rec-3* alleles from other lineages were called *rec-4*, *rec-5*, or *rec-w* until identity was demonstrated (see reference 167).

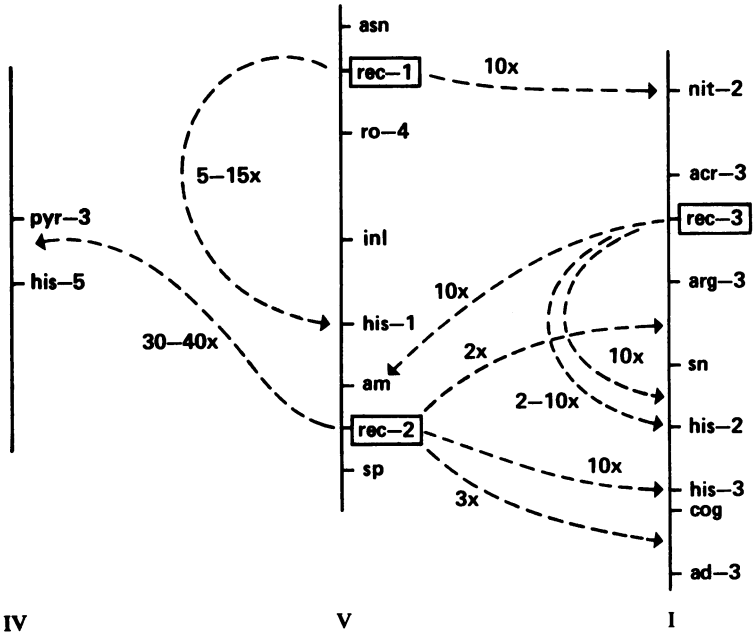


FIG. 22. Map locations of the *rec* genes. Arrows show the sites where they are known to affect meiotic intra- or interlocus recombination frequencies, and the magnitude of the effect (170 and references therein; D. E. A. Catcheside, personal communication).

***rec-3*: recombination-3**

IL. Between *acr-3* (1 to 2%) and *arg-3* (2 to 6%) (168, 173). (*166*)

Presence of allele *rec-3*$^+$ reduces recombination within the loci *his-2* (IR) and *am* (VR) but not in adjoining regions or within *gul-1*, which is less than 0.3% from *am* (173, 997, 998). Crossing over is also reduced in the interval between *sn* and *his-2* (174) (Fig. 22). Three alleles known: *rec-3*, *rec-3*^{L}, and *rec-3*$^+$ (168). A recessive allele from another lineage was earlier called *rec-x* (see reference 167).

rec-4*, *rec-5*, *rec-w

See *rec-2*.

rec-x

See *rec-3*.

rec-z

See *rec-1*.

***rg-1*: ragged**

IR. Right of *T(AR173)*; hence, of *his-2*. Left of *lys-4* (1 to 7%) (271, *789*, 810).

Spreading dense colonial growth with poor conidiation (789). Increased hyphal branching; bumpy mycelial surface. Altered phosphoglucomutase (isozyme I). Accumulates glucose-1-phosphate (117). Cell wall composition (132). Normal levels of NADPH (110) and linolenic acid (115). Photograph (112). The double mutant *rg cr* grows as small discrete conidiating colonies suitable for velvet replication (634). For examples of applications, see references 932 and 1020. Unlike *sn cr*, which it resembles phenotypically, the double mutant *rg cr* is not homozygous fertile. Allele R2357 was formerly called *er*: erupt (see reference 382). Allele S4357 was formerly called *col-7* (see reference 675).

***rg-2*: ragged-2**

I. Linked to *mt* (15%) after introgression into *N. crassa* (D. D. Perkins, unpublished data). Interpreted as unlinked to *rg-1* and *su(rg-2)* in *N. sitophila* (680).

Found in *N. sitophila* crosses involving an introgressed *rg-1*. Morphology similar to that of *rg-1* mutants (680). Altered phosphoglucomutase (isozyme II) (678).

***rib*: riboflavin**

Only two *rib* loci are known in *Neurospora*, compared to six which have been assigned to biosynthetic steps in *Saccharomyces* (see reference 256). Supplemented medium should be shielded from light to avoid destruction of riboflavin.

***rib-1*: riboflavin-1**

VIR. Between *T(AR209)* and *pan-2* (3%). Right of *ad-1* (3 to 6%), the centromere (1%), and *glp-4* (4%) (486, 1012, 1102). (*482*)

Requires riboflavin (681). Used to demonstrate role of flavin as a photoreceptor for carotenogenesis and for phase shifting and suppression of circadian conidiation (775). Allele 51602 is heat sensitive (34°C versus 25°C); allele C106 is not (380).

***rib-2*: riboflavin-2**

IVR. Right of *T(4342)*. Left of *chol-2*. Probably left of *pyr-3* (1/24 asci) (*381*, 808).

Requires riboflavin (381). Used to demonstrate the role of flavin as a photoreceptor for phase shifting of circadian conidiation and carotenogenesis (775).

ribosomal RNA

Genes specifying 5.8S, 17S, and 26S rRNA (but not 5S) are located in the nucleolus organizer region. See *NO*.

***rip-1*: ribosome production-1**

IIR. Linked to *un-15* (1%); right of *fl* and *trp-3* (6 to 9%). (PB).

Conditional defect in production of 60S ribosomal subunits (622). At the restrictive temperature (37°C), RNA synthesis is affected first and then protein synthesis; 60S cytosolic ribosomal subunits are underaccumulated, and relatively little 25S rRNA is produced (618, 620, 622; P. J. Russell, personal communication). Defective ribosome biosynthesis at high temperatures is attributed to a defect in rRNA processing (622). Attains 2.4% of the wild-type growth rate at 35°C and 80% at 25°C (P. J. Russell, personal communication). Scored as an irreparable heat-

sensitive *un* mutant (see *un*). Good fertility, growth, and viability make *rip-1* preferable to *un-15* as a marker for the right end of II. The original strain carrying both *rip-1* and *inl* (89601) was called 4M(t). The *rip-1* allele in this strain was originally called 1(t).

***ro-1*: ropy-1**

IVR. Linked to *pan-1* (0/394) (*633*).

Cable-like aggregates of hyphae grow up the tube from agar slants. Conidia form in dense clumps at the top (789). Hyphae are curled microscopically (382). Cell wall analysis; photograph (278). Reduced amount of cell wall peptides (1165). Growth limited on glycerol medium (212). Fertile as the male; perithecia rare or absent when used as the female.

***ro-2*: ropy-2**

IIIR. Right of *trp-1* (2 to 14%). Left of *T(D305)* and *phe-2* (5%) (11, 812, *814*; D. D. Perkins, unpublished data).

Resembles *ro-1* (789).

***ro-3*: ropy-3**

IIL. Right of *pyr-4* (1 to 2%). Left of *T(NM149)* and *thr-2* (6 to 25%) (808, *812*).

Resembles *ro-1* (812). Growth limited on glycerol medium (212). Called *cfl* on map in reference 812.

***ro-4*: ropy-4**

VR. Linked to *pab-2* (0/407). Between *ad-7* (4%) and *inv* (5%). (156, *382*, 816, PB)

Resembles *ro-1*. Growth limited on glycerol medium (212). Mutations R2428 and R2520, called *ro-5* and *ro-8* in references 382 and 698, are allelic with *ro-4* allele B38 (816). Reduced amount of cell wall peptides (1165).

ro-5

Allelic with *ro-4*, q.v. (see reference 816).

***ro-6*: ropy-6**

IR. Between the *T(4540)* breakpoints; hence, between *nic-2* (0/95) and *thi-1* (*382*, PB).

Resembles *ro-1* (382).

***ro-7*: ropy-7**

IIL. Linked to *pi* (0/75). Left of *cys-3* (11%) (*382*, PB).

Resembles *ro-1* (382). Growth limited on glycerol medium (212). Female sterile, contrary to misprint in reference 811.

ro-8

Allelic with *ro-4*, q.v. (see reference 816).

***ro-9*: ropy-9**

II. Right of *T(NM149)*. Probably left of *arg-5* (8%). Linked to *thr-3* (0/63) (*382*, PB).

Growth limited on glycerol medium (212). Misnamed. Hyphae not curled, unlike those of strains carrying other *ro* genes (D. D. Perkins, unpublished data). Makes barren perithecia in the cross of allele R2526 × *tng*, which is possibly allelic, but R2526 strains are poor female parents. Conidia of R2526 strains are normal sized, unlike those of the mutant *tng*.

***ro-10*: ropy-10**

IL. Left of *fr* (18%) (*PB*).

Resembles *ro-1* (PB).

***rol-1*: ropy-like-1**

IV. Linked to *pdx-1* (0/88) (*382*).

Resembles ropy strains in growth habit on slants, but hyphae are not curled microscopically (382).

***rol-2*: ropy-like-2**

VII. Linked to *met-7* (0/298) (PB). (*382*)

Resembles *rol-1* (382).

***rol-3*: ropy-like-3**

VR. Between *ilv-1* (2%) and *cot-4* (5%) (*698*).

Resembles *rol-1*. Photograph: Fig. 16 in reference 382.

***ros*: rosy**

Allelic with *al-3*, q.v. (*PB*).

Strains carrying *al-3* allele Y234M70, called *ros*, produce pale pink carotenoids more abundantly than strains carrying *al-3* allele RP100 or P7775 (PB). Original from T. Ishikawa. Studied briefly by A. M. Kapular.

***Rsp*: Round spore**

See *R*.

s

Originally used for *arg-12*^{s}. See *arg-12*.

***sar-1*: surfactant resistant-1**

I. Near mating type (*21*).

Resistant to surface-active agents dequalinium chloride, cetyltrimethyl ammonium bromide, and benzalkonium chloride. Resistant growth follows an adaptive lag phase. Em A (FGSC 627) and related *A* laboratory wild types carry a mating-type-linked *sar* gene that may be *sar-1*. Another, phenotypically distinct, mutation close to *mt* is designated *sar-3*, but evidence for nonallelism is not given (21).

***sar-2*: surfactant resistant-2**

Unmapped. Independent of *sar-1* (21).

***sar-3*: surfactant resistant-3**

I. Near mating type (*21*).

Differs from *sar-1* and *sar-2* mutants in growth responses and resistance specificities (21). See *sar-1*.

***sat*: satellite**

VL. Linked to *lys-1* (35%) (*60*); left of *T(OY321)* and *NO* (D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation).

Microscopically visible terminal satellite, distal to the nucleolus organizer in chromosome 2. Seen at pachytene as a tiny dot on the surface of the nucleolus (656). *N. crassa* laboratory strains differ in the presence (*sat*$^{+}$) or absence (*sat*$^{-}$) of the satellite. No satellite has been found in *N. intermedia* or other *Neurospora* species. Best scored during pachytene, using orcein. Photographs (60, 817). Translocations *T(AR190)*, *T(ALS182)*, and *T(ALS176)* involve the terminal satellite region (817).

***sc*: scumbo**

IIIR. Between the centromere and *ser-1* (*504*). Linked to *thi-4* (1/280) and *spg* (0++/179); right of *acr-2* (3 to 6%) (498, 814).

Irregular flat spreading growth with knobby protrusions but no conidia. Mycelium usually appears yellowish rather than orange. Female fertile. Homozygous *sc* × *sc* crosses give nonlinear asci (827, 828, 1011). Cell wall analysis (132). Reduced amount of cell wall peptides (1165). Allele R2503 called *col-14*; allele R2386 called *smco-2* (382). For modifier, see *mod(sc)*.

***scon*: sulfur control**

Probably VR, right of *his-6* (*R. L. Metzenberg, personal communication*). But presence of a translocation (and, therefore, pseudolinkage) cannot be ruled out because white spores are prevalent in *scon*^{c} crosses. Difficult to map because of infertility (R. L. Metzenberg, personal communication).

Regulates a group of enzymes of sulfur metabolism, including arylsulfatase and choline sulfatase. Allele *scon*^{c} is constitutive for these enzymes, but its effect in a heterokaryon is restricted to its own nucleus (123). Hypostatic to regulatory gene *cys-3* (284). Review of regulation (642). Ascospores and conidia germinate poorly (123, 930).

***scot*: spreading colonial-temperature sensitive**

VR. Between *al-3* (7%) and *his-6* (11%) (*810*).

Spreading colonial growth with delayed, reduced conidiation on solid media and pelleted growth in liquid medium, above 34°C. Not distinguishable from *scot*$^{+}$ strains at 25°C. Best scored on glycerol complete medium at 39°C. Present in Beadle-Tatum and Rockefeller-Lindegren wild types and numerous derivatives (810). Probably the same gene was discovered independently by Fincham and by Emerson (322), studied by Pao (780), and called *t*, thermophobic. Strains containing *t* showed start-stop growth in growth tubes, with growth distance depending on carbon source (sucrose versus lactose or galactose) and concentration (J. R. S. Fincham, personal communication). *scot* could be responsible for temperature effects on *moe* mutants of reference 382.

***scr*: scruffy**

IIR. Linked to *arg-12* (2%); probably to the left (PB). (*828*)

Semicolonial growth, few aerial hyphae, dichotomization of hyphal tips, reduced conidiation. Heat sensitive, growing poorly with no conidia at 39°C and more like the wild type at 25°C. Asci abnormal in homozygous *scr* × *scr* crosses, with altered arrangement of ascospores. Occasional asci have fewer than eight ascospores, and these include large spores. Not all asci are nonlinear. Recessive. (827, 828)

***sdh-1*: succinate dehydrogenase-1**

I. Linked to mating type (0/13) (*307*).

Succinate dehydrogenase activity 18% that of the wild type. Succinate oxidase activity low. Selected by failure to reduce nitrotetrazolium blue in the presence of succinate and phenazine methosulfate in overlays after inositol death enrichment on acetate. (307) Deficient in the high potential iron protein iron-sulfur center of the succinate dehydrogenase complex (306).

***ser-1*: serine-1**

IIIR. Between *sc* (4%) and *pro-1* (9%) (*504*).

Uses serine or glycine (504). Slightly deficient in serine hydroxymethyltransferase; raised levels of 10-formyltetrahydrofolate synthetase. Lacks ability to incorporate C_1 units from glycine. Extracts lack detectable methylfolates. Negligibly deficient in 5,10-methylene tetrahydrofolate reductase. (209). Ascospores from *ser-1* × *ser-1* crosses do not blacken or do so only after a long delay (816). Morphologically normal; abnormal morphology at elevated temperatures that was reported in reference 816 was not due to *ser-1* but to *scot* (810).

***ser-2*: serine-2**

VR. Between *met-3* (4%) (438) and *cot-2* (5 to 8%) (156, 158). (*812*)

Uses serine (290) but not glycine (D. D. Perkins, unpublished data). Does not grow on hydrolyzed casein (290). Inhibited by thienylserine (R. W. Barratt, personal communication).

***ser-3*: serine-3**

IL. Right of *cys-5* (<1%). Left of *un-3* (<1%) and *In(NM176)* (*816*, 1093, PB).

Uses serine and grows less well on formate. Also grows fairly well on a combination of adenine, methionine, tryptophan, and lysine. Does not grow on casein hydrolysate (290). No or little response to glycine (D. D. Perkins, unpublished data). Deficient in phosphoserine phosphatase activity (70). Inhibited by leucine. Scorability good, but vigor and leakiness vary markedly in different isolates. Homozygous crosses give mostly white ascospores (816; D. D. Perkins, unpublished data). Alleles, 47903 and JBM5.

***ser-4*: serine-4**

IVR. Right of *arg-2* (<1%) (652). (*651*)

Uses serine or glycine. Incompletely blocked. Not deficient for any of the enzymes involved in serine synthesis from 3-phosphoglyceric acid or glyceric acid. Intracellular pool deficient in serine, glycine, and alanine and accumulates threonine and homoserine (651). Produces abundant L-amino acid oxidase but no tyrosinase while growing slowly on minimal medium (651, 1099). Allele DW110 called P110 in reference 651.

***ser-5*: serine-5**

IIIR. Linked to *trp-1* (1%) and *ser-1* (12%) (*653*).

Uses serine or glycine. Incompletely blocked (653).

***ser-6*: serine-6**

VIL. Between *nit-6* (10%) and *ad-8* (16%) (PB). (*544*)

Responds to serine. Slight response to glycine. Extremely leaky. Scorable by slow, sparse conidiation on minimal slants or auxoanographically (D. D. Perkins, unpublished data). Can be crossed on minimal crossing medium without supplement. Allele DK42 obtained as putative leucine regulatory mutation (544), but a regulatory role is dubious (S. R. Gross, personal communication). Called DK42 (544). Probably allelic with *T(VL;VIL)OY325 ser*, which has a breakpoint left of *lys-5* (11%) in VIL, and shows 0/28 recombination with DK42, which it resembles in leakiness. The mutant OY325 grows more profusely than the mutant DK42 on minimal slants (PB).

***sf*: slow-fine**

I. Linked to *mt* (3%) and *cy* (3% in regular perithecia) (*689*, PB).

Growth from ascospores or conidial inoculum on minimal or complete medium is initially slow. Morphology not distinguishable in mass culture after it is grown up. Originally detected microscopically. Irregular ascus types reported for some perithecia. (689) First hyphae grow clockwise on surface. Scorable by slow initial growth on slants 3 or 4 days after ascospore germination, 25°C (PB).

***sfo*: sulfonamide dependent**

VII. Linked to the centromere (<1%) (*318*), between *thi-3* (6%) (874) and *hlp-1* (1 to 9%) (458).

Requires sulfonamide at 35°C and is stimulated by sulfonamide at lower temperatures (319). Overproduces *p*-aminobenzoic acid; hence, growth is inhibited by exogenous *p*-aminobenzoic acid (1179). For *sfo; pab-1* double mutant on minimal medium plus sulfonamide, *p*-aminobenzoic acid is stimulatory at very low concentrations but inhibitory at higher (319). For *sfo; met-1* double mutant on minimal medium plus sulfonamide, methionine is stimulatory at very low concentrations but inhibitory at higher (1180). Best tested on solid minimal medium growth tubes at 35°C. Suppressor mutations occur frequently (318).

***sg*: spontaneous germination**

Unmapped.

Ascospores germinate without heat shock. Usually associated with a very poor vegetative growth habit. A component of the multiply mutant combination resulting in the cell-wall-less "slime" phenotype. Possibly more complex than a single gene (321). See *slime*.

***sh*: shallow**

VR. Between *ilv* (4 to 7%) and *md* (3%) (296). (*812*)

Spreading morphology. Hyphae do not penetrate deeply into agar (812). Fanlike array of hyphae. Photographs (296, 382).

***shg*: shaggy**

IIIR. Linked to *trp-1* (7%) and *acr-6* (0/368). Recombines with *vel* and *col-13*. (499, PB).

Slow growth over agar surface, forming conidia on irregular aerial hyphae that are most abundant high in the slant. Mutants *acr-4* and *acr-6* originated in *shg* and are resistant to acriflavin only in combination with *shg* (499). Formerly called *mo(KH160)*.

***sit*: siderophore transport**

Unmapped.

Defective uptake of exogenous ferricrocin and coprogen. Several mutations are known that represent at least three loci. Selected in triple mutant *aga; arg-5; ota* (G. W. Charlang and N. P. Williams, personal communication), which is blocked in the known pathways to ornithine (240) and thus depleted of siderophores (1147; G. W. Charlang and N. P. Williams, personal communication).

***sk*: skin**

VIIR. Right of *nt* (7 to 17%) (*789*).

Leathery, nonconidiating rapid surface growth (789). "Mucilaginous substrate hyphae" (1088). *sk* ascospores are slow to mature, but good allele ratios are obtained from crosses held for 3 weeks at 25°C. Female sterile. Allele R2466 called *mo-3*; alleles Y6821, R2408, and R2529 called *moe-1* (382, PB).

***Sk-1*: Spore killer-1**

Unmapped. Less than 1% second-division segregation.

Characteristics similar to those of *Sk-2*, q.v. Found in *N. sitophila*. Sensitive and killer genotypes are about equally frequent among strains from different localities. Not introgressed into *N. crassa* (857, 1092). To avoid confusion, the symbol *Sk-1* should not be used for any other *Sk* occurrence.

***Sk-2*: Spore killer-2**

III. Second-division segregation rare or absent. The killer allele $Sk\text{-}2^K$ recombines with *his-7* (25%) but suppresses crossing over in the interval *r(Sk-2)-1* to *leu-1* (29% in controls). (*1092*). $Sk\text{-}2^K$ does not recombine with *acr-7* (0/1800) or *acr-2* (0/100,000) (B. C. Turner, personal communication).

Kills ascospores of sensitive genotype after meiosis in crosses heterozygous for the killer allele $Sk\text{-}2^K$. In $Sk\text{-}2^K \times Sk\text{-}2^S$ crosses (Killer × Sensitive), each ascus contains four inviable

clear ascospores and four viable black ascospores that are Sk^K. Ascospores are not killed in $Sk^K \times Sk^K$ crosses (1092). Meiosis and postmeiotic mitosis appear normal by light microscopy. Sensitive spores first appear abnormal after one nuclear division in the ascospore. $Sk\text{-}2^S$ nuclei survive if included in the same ascospore with $Sk\text{-}2^K$ (857). Originated in *N. intermedia*; introgressed into *N. crassa*. Most strains from nature are sensitive, but resistant strains of *N. intermedia* are common in some geographic areas, and two resistant strains of *N. crassa* have been found [see *r(Sk-2)-1*]. Strains resistant to $Sk\text{-}2^K$ are not necessarily resistant to $Sk\text{-}3^K$. In $Sk\text{-}2^K \times Sk\text{-}3^K$ crosses, less than 1% of the ascospores are normal and viable (1092; B. C. Turner, personal communication). If $Sk\text{-}2^K$ and $Sk\text{-}3^K$ nuclei are included in the same ascospore, both nuclei survive and the spore is not killed (N. B. Raju, personal communication).

***Sk-3*: Spore killer-3**

III. Near the centromere. The killer allele $Sk\text{-}3^K$ suppresses crossing over in the interval *r(Sk-2)-1–leu-1* (29% in controls). Recombines with *his-7* (11%) but not with *leu-1* (0/72) or *acr-7* (0/60,000) and only 3/19,000 with *acr-2*. (*1092*; B. C. Turner, personal communication)

Origin and characteristics resemble those of *Sk-2*, q.v. However, $Sk\text{-}3^K$ strains are sensitive to killing by $Sk\text{-}2^K$ and vice versa. The killer allele was found in *N. intermedia*, introgressed into *N. crassa*. Most wild-type strains of both species are sensitive, but resistant strains of *N. intermedia* have been found. Strains resistant to $Sk\text{-}3^K$ are not necessarily resistant to $Sk\text{-}2^K$ (857, 1092).

Sk(ad-3A)

See *ad-3A*.

slime

A multiple-mutant strain lacking cell wall and growing as protoplasts or plasmodium. The original strain contained at least two mutations (*fz*, fuzzy; *sg*, slow germination) in addition to the markers already present, *arg-1*, *cr-1*, *aur*, and *os-1*. Of these, *fz*, *sg*, and *os-1* are required for a slime-like phenotype (321). Can be recovered with inserted markers in f_1 of crosses if filtration enrichment is used (746). Cell wall-like material may be produced in small quantities in newly resolved stocks. Loses ability to form heterokaryons or to function as the fertilizing parent after continuous growth (973). Slime protoplasts can be induced to fuse (743). Stocks maintained in same-mating type (321) or mixed-mating type (746) heterokaryons or frozen at −70°C in situ on agar medium (968) or in dimethyl sulfoxide (221) or growth medium (961). Recovered from heterokaryons by filtration; see reference 746. Plasma membrane can be isolated and stored in large quantity (1037). Used for fatty acid analysis of plasma membrane (365), for study of vacuoles (922, 638), and for gentle extraction of enzymes (372). Used to show that polyadenylic acid polymerase and nuclease activities are largely located in the nucleus (962). For general methodology, see references 746 and 1161. For an alternative source of cell wall-free *Neurospora*, see references 970 and 971.

***slo-1*: slow-1**

IR. Between *mt* (14%) and *thi-1* (2 to 5%) (*789*).

Slow growth from conidia or ascospores (789). Conidiation lags significantly behind that of the wild type. Morphology normal. Not tested for cytochrome deficiency or ability to reduce tetrazolium.

***slo-2*: slow-2**

VII. Left of *met-7* (2%) (816). (*W. N. Strickland, cited in reference 816*)

Slow growth from conidia or ascospores. Conidiation lags days behind that of the wild type (816, 1035). Not tested for cytochrome deficiency or ability to reduce tetrazolium.

***smco*: semicolonial**

Symbol and name used by Garnjobst and Tatum (382) for mutants that begin growth on agar as small colonies and sooner or later produce a flare of wild-type-appearing hyphae (with or without conidia). Mutant genes of the series *smco-1* to *smco-8*, described in reference 382, were sometimes named as new *smco* loci without having been tested for allelism with already named morphological mutations having similar map locations.

***smco-1*: semicolonial-1**

I. Linked to *mt* (1%) and *rg-1* (0/72) (*382*).

***smco-2*: semicolonial-2**

Allelic with *sc*, q.v. Linkage in group I sug-

gested in reference 382 was not confirmed (A. M. Srb, personal communication). Independent of *ad-3*. Linked to *his-7* (15%). No recombination (0/19 asci) or complementation with *sc* (A. M. Srb, personal communication).

***smco-3*: semicolonial-3**

I. Linked to mating type (10%) and *al-2* (29%). Recombines with *col-7* and *smco-1* (*382*).

***smco-4*: semicolonial-4**

IVR. Linked to *pan-1* (8%) (*382*)

***smco-5*: semicolonial-5**

I. Linked to mating type (2%). Recombines with *rg* and *smco-1*. Not fertile with *smco-3*. (*382*)

Semicolonial flat growth persists until 4 to 7 days after ascospore germination, when the wild-type mycelium develops. Mycelium is similar to that of the wild-type on transfer, but *smco-5* ascospores repeat the cycle. (382)

***smco-6*: semicolonial-6**

VR. Right of *met-3* (14%). Linked to *asn* (6%), near *pyr-6* (156, *698*).

***smco-7*: semicolonial-7**

VR. Right of *ilv-1* (2%). Linked to *rol-3* (0/154) (*698*).

Conidiates in a crescent at the top of slants. Morphology distinct from that of strains carrying *rol-3*, which complements *smco-7* (382).

***smco-8*: semicolonial-8**

IVR. Linked to *pan-1* (1 to 7%) and *smco-4* (30%). Complements *col-1* and *col-8* (*382*).

Sometimes flares out at the top of slants (382). Unable to grow on galactose or grows as restricted colonies (861). Reduced amount of cell wall peptides (1165).

***smco-9*: semicolonial-9**

IVR. Linked to *pan-1* (5%), *smco-4* (2%), and *smco-8* (13%) (*382*).

Morphology partially normalized by isomaltose or starch. Altered inhibitor of branching enzyme α-1,4-glucan-6-glycosyltransferase (1). Reduced amount of cell wall peptides (1165). Homozygous *smco-9* × *smco-9* cross produces nonlinear asci (similar to those of *pk* strains) (1011). Strain originally used was complex, producing two types of colonial progeny, only one of which behaves as a recessive ascus mutant (1007).

***sn*: snowflake**

I. Right of *T(39311)* and *arg-3* (1 to 6%). Left of *T(AR173)* and *his-2* (<1 to 12%) (174, 808). (*687*)

Spreading colonial growth with good conidiation. Linear growth is less than 1/10 that of the wild type (19). Detectable immediately after ascospore germination by hyphal patterns which suggested the name (688). Abnormal microfilaments (19). Contains actin-like protein (20). Said not to exhibit cytoplasmic streaming (18). Meiosis and ascospore formation are normal in homozygous *sn* × *sn* crosses (N. B. Raju, personal communication). Good female fertility. Morphology similar to that of *sp*, *cum*, and *cot-4* mutants (at 25°C) (PB). Used to study development of crystalline inclusions (17).

The *cr sn* double mutant grows as small, discrete, conidiating colonies suitable for velvet replication. The double mutant *cr sn* resembles the *rg cr* double mutant phenotypically and has the advantage of fertility in homozygous crosses (796); for example of application, see reference 180.

***so*: soft**

IR. Between *arg-13* (2 to 12%) and *aro-8* (7 to 11%) (437, 816). (*789*)

Lawn of fuzzy short aerial hyphae and conidia formed more uniformly than by the wild type, and closer to surface of agar, similar to peach. Delicately pigmented, distinctive morphology (789). Recurrences arise frequently by mutation in strains of Oak Ridge background (538; E. Käfer, personal communication). Best scored early on short, obtuse slants. Phenotype more pronounced on sorbose-sucrose plates. Pleiotropic female sterility and short conidial life span. Maps at same site as *age* 1.3 and indistinguishable from *age* mutants; see *age-1* (K. D. Munkres, personal communication). Aerial phenotype of allele B230 reverts (K. D. Munkres, personal communication).

***sor*: sorbose resistance**

Sorbose is used to induce colonial growth in platings of *Neurospora* (109, 239). Resolution is improved if sorbose is used in conjunction with *cot-1* (165). Numerous *sor* mutants have been obtained which show spreading growth on concentrations of sorbose that restrict the wild type (e.g., references 560, 561, and 898). With the exception of *sor-4*, scoring has been on minimal medium plus 0.025% filter-sterilized sorbose. Certain mutants selected in other ways may also be resistant to sorbose. For example, the mutant *sor(T9)*, selected by ability to hydrolyze starch and defective in extracellular amylase, is simultaneously sorbose resistant and osmotic sensitive (710). Sorbose-resistant mutations at four loci act as dominance modifiers of the ascus effect of dominant *Pk-2* alleles (898). Most *sor* mutants have probably not been examined for possible pleiotropic amylase or osmotic phenotypes.

***sor-1*: sorbose resistant-1**

VIL. Left of *ylo-1* (3%) (*560*).

Defective in sorbose uptake (561) and thereby resistant to growth restriction by sorbose (560). Recessive (562, 564). Symbol changed from *sor-A* (853).

***sor-2*: sorbose resistant-2**

VII. Linked to *nt* (31%) (*560*).

Defective in sorbose uptake (561) and thereby resistant to growth restriction by sorbose (560). Recessive (562, 564). Symbol changed from *sor-B* (853).

***sor-3*: sorbose resistant-3**

IIIR. Linked to *ad-4* (7%) (*560*).

Resistant to growth restriction by sorbose (560). Recessive with respect to colony size. Partially recessive for percent conidial germination on sorbose test media (562, 564). In heterokaryons with *sor-1* or *sor-2*, the phenotype is intermediate between the resistant single mutant phenotype and the sensitive wild type (562). Symbol changed from *sor-C* (853). Called *sor^r-17* (560).

***sor-4*: sorbose resistant-4**

IL. Linked to *phe-1* (<1%). Right of the *In(H4250)* breakpoint and of *suc* (1%). Left of *arg-1* (<1 to 4%) (816). (*1014*)

Resistant to growth restriction by sorbose. Detected in the *pat; pro-1* strain used to demonstrate circadian rhythm (1014). Modifies dominance of *pk* alleles (898). Scoring clear on minimal slants with 2% sucrose plus 3% sorbose after 1 and 2 days, 34°C (816). Called *sor(DS)* (816; see reference 853); called *Pk-mod-D* (898). *sor^r-15* and *sor(T9)* are possible alleles of *sor-4* based on map position (560). It is not clear whether *sor-4* is a locus separate from *pat*, q.v.

***sor-5*: sorbose resistant-5**

V. Linked to *his-1* (*560*).

Resistant to growth restriction by sorbose. Alleles designated *sor^r-14* and *sor^r-19* in reference 560.

***sor-6*: sorbose resistant-6**

Probably III or VI (PB).

Resistant to growth restriction by sorbose (560). Recessive (561). Designated *sor^r-6* in reference 560.

***sor(T9)*: sorbose resistant**

IL. Between *mt* (6%) and the centromere (5%) (*710*).

Resistant to colonializing action of sorbose at 25°C but not at 35°C (712). Low glucoamylase activity. Extracellular amylase activity <0.5 that of the wild type. Slow growth; osmotic sensitivity comparable to that of *os-1* mutants. High extracellular acid phosphatase activity (710). Enhances growth of *gpi* strains on glucose or sucrose; used to obtain mutants defective in glucosephosphate isomerase (711). Obtained by plating mutagenized conidia in medium containing starch and observing cleared zone around colonies. The glucoamylase, osmotic, and sorbose resistance properties cosegregated in 101 isolates (710). Formerly called *T9* (710, 711), *gla* (glucoamylase) (50), and *amy* (325). Possibly allelic with *sor-4* or *sor^r-15* or with both (560). Dissimilar in morphology to *os-4* strains (PB). Linked to regulatory gene *exo-1*, which has not been tested for allelism but is stated to be on the other side of *mt* from *sor(T9)*; see *exo-1*. (*T* in the allele number signifies Tokyo, not translocation.)

***sp*: spray**

VR. Between *leu-5* (3 to 9%) and *am* (1 to 8%). Linked to *cot-4* (11%) (122, 839, *1036*).

Grows initially as colonies that are flat on the surface, and then aerial mycelium fans upward (789). Photographs of allele B132 (278, 296). For ultrastructure and intraconidial conidia, see references cited in reference 1088. Cell wall analysis (278). Reduced amount of cell wall peptides (1165). Morphology similar to that of *cot-4* (at 25°C), *sn*, and *cum* mutants (PB). Excellent female fertility. Used as lawn for mating type tests on plates (990).

***spco*: spreading colonial**

Symbol and name used in reference 382 for mutants that begin growth on agar as a colony but do not remain restricted, spreading to cover the agar surface. Mutant genes of the series *spco-3* to *spco-15*, described in reference 382, were sometimes assigned to new *spco* loci without having been tested for allelism with already named morphological mutations having similar map locations. Growth rates and other characteristics of 11 *spco* mutants are described and analyzed in references 1085 and 1086.

***spco-1*: spreading colonial-1**

See *col-4*.

***spco-2*: spreading colonial-2**

See *wa*.

***spco-3*: spreading colonial-3**

See *spco-7*.

***spco-4*: spreading colonial-4**

VIIL. Linked to *do* (<1%) and *nic-3* (1%, probably to the left) (*816*).

Fine hyphae (382). Initially aconidial. Capable of conidiating on the surface of complete medium (D. D. Perkins, unpublished data). Hyphae extend faster within agar medium than on the surface, resulting in a dense hemispherical colony, most of which is embedded (1085).

***spco-5*: spreading colonial-5**

VII. Linked to *nt* (20%) and *col-17* (6%) (*382*).

Homozygous *spco-5* × *spco-5* crosses make abnormal, nonlinear asci (1007). Reduced amount of cell wall peptides (1165). Complements *col-2* and *spco-4* (382).

***spco-6*: spreading colonial-6**

VII. Linked to *do* (10%), *spco-5* (8%), and *nt* (20%) (*382*).

Complements *col-17*, *col-2*, *spco-4*, and *spco-5* (382).

***spco-7*: spreading colonial-7**

VI. Near *ad-1* (0/65). Right of *ylo-1* (4%); left of *T(AR209)* and *trp-2* (16–21%) (PB). (*382*)

Complements *moe-2*. Allele R2365 preferred, for excellent growth, conidiation, and fertility (PB). Allele R2365, called *spco-3*, was incorrectly assigned to V (42% recombination with *inl*) (382). R2365 × *spco-7* (R2457) crosses result in small perithecia devoid of ascospores (PB).

***spco-8*: spreading colonial-8**

IV. Linked to *pan-1* (23%) (*382*).

***spco-9*: spreading colonial-9**

VR. Linked to *asn* (6%); right of *met-3* (18%) (*698*).

Complements *ro-5*, *cot-2*, and *smco-6*. Morphologically distinct from *col-9*, but allelism tests were inconclusive (*382*, 698).

***spco-10*: spreading colonial-10**

VR. Between *ilv-1* (24%) and *inl* (5%) (21 asci) (*382*, 698).

***spco-11*: spreading colonial-11**

I. Linked to mating type (17%) and *mo-5* (18%) (*382*).

Occasionally conidiates at the top of slants (382).

***spco-12*: spreading colonial-12**

I. Linked to mating type (20 to 35%), *mo-5* (5%), and *spco-11* (43%) (*382*).

Downy center and lacy growing border (382).

Reduced amount of cell wall peptides (1165). Hyphal growth highly branched (1086).

***spco-13*: spreading colonial-13**

VI. Linked to *trp-2* (16%) and the centromere (1/10 asci) (*382*).

May be allelic with *spco-7* or *moe-2* (crosses and heterokaryons unsuccessful) (382).

***spco-14*: spreading colonial-14**

II. Linked to *arg-5* (7%) (*382*).

Complements *da* and *bal*. A few scattered conidia (382).

***spco-15*: spreading colonial-15**

III. Linked to *spg* (10%), *pro-1* (18%), and the centromere (0/10 asci). Recombines with *col-14* and *col-16*. Complements *mo-4*, *col-14*, and *col-16* (*382*).

Stock lost? A morphological mutation in FGSC stock no. 2389, which is designated *spco-15* (R2537), is not linked to III (PB).

***spe-1*: spermidine-1**

VR. Linked to *cyh-2* (2%); left of *inl* (12%) (*657*, PB).

Uses putrescine, spermidine, or spermine. Affects ornithine decarboxylase (L-ornithine carboxy-lyase) (Fig. 10). Does not suppress *pro-4* (657). Excretes yellow pigment into synthetic cross medium (PB). Meiosis normal in homozygous crosses (on 50 μg of spermidine per ml), which produce mostly white and a few viable black ascospores (N. B. Raju, personal communication; R. H. Davis, personal communication). Formerly called *put-1*.

***spg*: sponge**

III. Between *acr-2* (1 to 11%) and *ser-1* (8%). Linked to *sc* (<1%) and *thi-4* (0/103) (*816*, PB).

Conidiating spreading colonies. Morphology distinct from that of *sc* mutants. Good viability. Growth on minimal medium of Vogel (1103) is less spreading than on crossing medium of Westergaard and Mitchell (1134) (PB). Hyphae fuse to form bundles (382). Reduced amount of cell wall peptides (1165).

***ss*: synaptic sequence**

IL. Linked very close to *nit-2* (<0.2%) (*161*).

When there is heterozygosity for alleles ss^E, ss^S, and ss^C (from Emerson, St. Lawrence, and Costa Rica wild types) recombination within *nit-2* is reduced 2- to 20-fold, but crossing over in the flanking interval *un-5* to *nit-2* or *nit-2* to *leu-3* is not affected. *ss* heterozygosity acts multiplicatively with $rec\text{-}1^+$ to reduce recombination within the *nit-2* locus 100-fold. (161)

***ssu*: supersuppressor**

Nonsense suppressors (either proved or putative). In a few cases, putative missense mutations were also suppressed (749). Allele specific but not locus specific. Often infertile or only slightly fertile (*ssu-3*) when used as the female parent, forming no perithecia or empty perithecia (954). Strains of genotype *ssu-1; am* or *ssu-2; am* or *ssu-3; am* or *ssu-4; am* or *ssu-5*, but not *ssu-6*, are cold sensitive, showing less than half the wild-type growth rate at 10°C on minimal medium (894). Nucleus-restricted action has been studied in heterokaryons (409). Interaction of certain supersuppressors with certain *ad-3B* alleles produces erratic stop-start growth (408). For tabular summaries of action on differential test alleles, see references 145, 955, and 957.

***ssu-1*: supersuppressor-1**

VIIR. Right of *met-7* (14%). Left of *nt* (23%) and of missense suppressor $su(trp\text{-}3^{td201})\text{-}1$ (10%) (*954*).

Allele WRN33 selected (953) as suppressor of nonsense mutation *am* (17), which may be either amber or ochre but cannot be UGA (956). Inserts tyrosine in the site where the wild type has glutamate (956). Used to identify suppressible alleles of *aro(p)*, *trp-1*, *trp-2*, *trp-3* (953, 144, 183), and *ad-3B* (749). Although perhaps the most efficient of known *ssu* mutations, *ssu-1* restores only about 20% of the wild-type amount of normal glutamate dehydrogenase in the double mutant with *am* allele (17) (reference 956).

***ssu-2*: supersuppressor-2**

I. Linked to *mt* (22%), probably between the centromere (7%) and *al-2* (26%). Recombines with *ssu-3* (*954*).

Allele WRU35 selected (954) as a coincident suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or

ochre but cannot be UGA (956). Also suppresses certain nonsense alleles of *trp-1*, *trp-2*, and *ad-3B* (see reference 955).

ssu-3: supersuppressor-3

I. Linked to *mt* (22%), probably between the centromere (10%) and *al-2* (33%) (*954*).

Allele WRU118 selected (954) as a coincident suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or ochre but cannot be UGA (956). Fails to suppress most nonsense mutations that are suppressed by *ssu-1* and *ssu-2* (see reference 955). Probably specifies insertion of an amino acid different from that inserted by *ssu-1* or *ssu-4* (Seale and Kinniburgh, cited in reference 955).

ssu-4: supersuppressor-4

VIIL. Between *nic-3* (28%) and *met-7* (20%) (*954*).

Allele WRU18 selected (954) as a suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or ochre but cannot be UGA (956). Also suppresses certain nonsense alleles of *trp-1* and *ad-3B* (see references 749 and 955).

ssu-5: supersuppressor-5

III or IV (145).

Allele Y319-45 selected as a suppressor of a nonsense allele of *aro(p)* (145). Also suppresses the nonsense mutation *trp-3* (td140) and certain *ad-3B* alleles, but not *am* (17) (see references 145, 749, and 957).

ssu-6: supersuppressor-6

VR. Linked to *his-1* (4%) (*145*).

Allele Y319-45 selected as a suppressor of a nonsense allele of *aro(p)* (145). Also suppresses certain nonsense alleles of *trp-3* and *his-3*, but not *am* (17) (references 145, 749, 957).

ssu-7: supersuppressor-7

VIL. Between *ad-8* (8%) and *ylo-1* (14%) (*954*).

Allele WRU7 selected (954) as a coincident suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or ochre but cannot be UGA (956). Also suppresses nonsense alleles of *trp-1*, *trp-2*, and *ad-3B*. *ssu-7* shows the widest spectrum of the known supersuppressors (955). Allele Y319-37 of *ssu-8*, which is in IR, has erroneously been called *ssu-7* in some FGSC lists and by Griffiths (408).

ssu-8: supersuppressor-8

IR. Linked to *al-2* (2 to 8%) (*145*).

Allele Y319-37 selected as suppressor of an *aro-(p)* nonsense allele (145). Also suppresses certain nonsense alleles of *trp-3*, *his-3* (reference 145) and *ad-3B* (reference 749). Allele Y319-37 (FGSC no. 1749), also called 54-su37, was initially thought to be a possible allele of *ssu-2* and thus was listed as *ssu-2* (?); later it was designated *ssu-8* (145). It has been erroneously called *ssu-7* in various FGSC lists and by Griffiths (408) and has been erroneously stated to be in linkage group VI (the location of the real *ssu-7*).

ssu-9: supersuppressor-9

Linkage not known. Locus distinct from other *ssu* genes (955).

Allele WRU98 selected (see 955) as coincident suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or ochre but cannot be UGA (956). Also suppresses nonsense alleles at *trp-1* and *trp-2* (see reference 955).

ssu-10: supersuppressor-10

Linkage not known. Locus distinct from other *ssu* genes (955). Allele RWU121 selected (see reference 955) as a coincident suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or ochre but cannot be UGA (956). Also suppresses nonsense alleles at *trp-1*, *trp-2*, and *ad-3B* (955).

ssu(WRU79): supersuppressor (WRU79)

Linkage not known. Locus distinct from other *ssu* genes (955).

Selected (955) as coincident suppressor of nonsense mutations *trp-3* (td140) and *am* (17), which may be either amber or ochre but cannot be UGA (956). Also suppresses nonsense alleles at *trp-1*, *trp-2*, and *ad-3B*. Spectrum resembles *ssu-1* (955).

_st_: sticky

IR. Between *ad-3B* (5%) and *thi-1* (14%) (*789*).

Mycelia adhere to needle. Subtle morphological difference from wild type. Exudate sometimes present (789). Grows poorly from conidial inocula on to minimal medium, with ballooning of hyphal tips not alleviated by mannose (D. D. Perkins, unpublished data).

***su*: suppressor**

su is used to designate the mutant suppressor allele, and su^+ is used to designate the wild-type nonsuppressor allele. Usage for *Neurospora* thus follows the long-established usage for *Drosophila* and other higher organisms.

su(arg-1)-1*: suppressor-1 of *arg-1

Unmapped. Not linked to *arg-1*.

Restores 23 to 36% of the wild-type L-citrulline:L-aspartate ligase activity to *arg-1* mutant 46004. Called $arg\text{-}1^{R26}$, $arg\text{-}1^{R3}$, *s-26*, and *s-3*. (62).

***su(bal)*: suppressor of balloon**

I. Linked to mating type (13%) (*948*).

Doubles the linear growth rate of the mutant *bal*. Decreases the K_m of glucose-6-phosphate dehydrogenase in the double mutant *bal; su(bal)*. Photographs of *bal* and *bal; su(bal)*. (948). Called *su-B*.

***su(col-2)*: suppressor of colonial-2**

IL. Tightly linked to mating type (0/837) (*948*).

Increases linear growth rate of the mutant *col-2* 10-fold. Morphology is wild type in the absence of *col-2*. Influences electrofocusing patterns of *col-2* glucose-6-phosphate dehydrogenase and, in the absence of *col-2*, of the normal enzyme. Photographs of *col-2* and *col-2; su(col-2)*. (948). Called *su-C*.

su(cot-1)*: suppressor of *cot-1

See *gul*.

***su(ile-1)*: suppressor of isoleucine-1**

VR. Right of *met-3* (4%) (*J. A. Kinsey, personal communication*).

Suppresses requirement of *ile-1* (J. A. Kinsey, personal communication).

***su(inl)*: suppressor of inositol**

Independent of *inl*.

Partial suppressor; allele specific. Restores limited ability to synthesize inositol to strains carrying allele 37401, allowing suboptimal growth on minimal medium. (390).

su(met-2)*: suppressor of *met-2

Unmapped. Not tested for allelism with *su(met-7)-1* or *su(met-7)-2*.

Isolated as a suppressor of *met-2* allele H98 (B. S. Strauss and S. Tokuno, via FGSC). Suppresses leaky mutants blocked between cysteine and homocysteine. It is probably the suppressor used by Wiebers and Garner (1136) to show that the suppressed strain differs from wild type in being able to incorporate sulfur from *S*-methylcysteine into cysteine regardless of the sulfate concentration. The suppressor does not have significantly increased acetylhomoserine sulfhydrylase (547). The FGSC number is 690.

su(met-7)-1*: suppressor-1 of *met-7

IR. Linked to *al-2* (1%) (*386*).

Selected as reversions of *met-7* (4894) by Giles (386). All spontaneous reversions were at this locus. Suppresses *met-7* (4894) and *met-2* (H98). Not tested for suppression of other alleles or loci. Suppressed strains attain wild-type growth on minimal medium after initial retardation, which is alleviated by methionine (386). This is apparently the suppressor used by Fischer (347) to show that the suppressor restores cystathionase I and II activities to 4894 and H98, respectively. Called S-1, FGSC 39.

su(met-7)-2*: suppressor-2 of *met-7

Unmapped.

Suppresses *met-7* (4894). Not tested for suppression of other mutants. Selected as a revertant of *met-7* (4894) by Giles (386). Recovered less frequently than *su(met-7)-1* and only after irradiation. Called S-2. (386).

***su([mi-1])-1*: suppressor-1 of [*mi-1*]**

IIIR. Linked to *ad-4* (14%) (*567*, 568).

Restores normal growth rate; alleviates deficient cyanide-sensitive respiration of [*mi-1*] (=

[*poky*]) and another group I cytoplasmic mutant. No effect on group II cytoplasmic mutant [*mi-3*] (85, 204, 568). Mitochondrial ribosomal subunit ratios and cytochrome spectrum (204). Called *sup-1*.

***su([mi-1])-3*: suppressor-3 of [*mi-1*]**

II. Linked to *fl* (31%). Not allelic with *su([mi-1])-4* or *su([mi-1])-10*: 20 and 25% unsuppressed progeny from intercrosses (*567*, 568).

Restores normal growth rate, alleviates deficient cyanide-sensitive respiration of [*mi-1*] ([*poky*]) and another group I cytoplasmic mutant. No effect on group II cytoplasmic mutant [*mi-3*] (85, 204, 568). Effects on mitochondrial cytochromes and ribosomal subunits (204). Called *sup-3* (568).

***su([mi-1])-4*: suppressor-4 of [*mi-1*]**

II. Linked to *fl* (22%) and *arg-5* (40%). Not allelic with *su([mi-1])-3* or *su([mi-1])-10*: 20 and 25% unsuppressed progeny from intercrosses (567, 568).

Restores normal growth rate, alleviates deficient cyanide-sensitive respiration of [*mi-1*] ([*poky*]) and another group I cytoplasmic mutant. No effect on group II cytoplasmic mutant [*mi-3*] (85, 204, 568). Mitochondrial ribosomal subunit ratios and cytochrome spectrum (204). Called *sup-4* (568).

***su([mi-1])-5*: suppressor-5 of [*mi-1*]**

VIIL. Left of *nic-3* (23%) (*567*, 568). Possibly allelic with *cyt-7* (87).

Restores normal growth rate; alleviates deficient cyanide-sensitive respiration of [*mi-1*] ([*poky*]) and another group I cytoplasmic mutant. No effect on group II cytoplasmic mutant [*mi-3*] (85, 204, 568). Mitochondrial ribosome profile and cytochrome spectra (204). Affects mitochondrial large subunit assembly. With wild-type mitochondria, causes cold sensitivity (203). Called *sup-5* (568) and su^{l}-*5* (203).

***su([mi-1])-10*: suppressor-10 of [*mi-1*]**

II. Linked to *arg-5* (29%) and *fl* (24%) (*567*, 568). Not allelic with *su([mi-1])-3* or *su([mi-1])-4*: 25% unsuppressed progeny from intercrosses (568).

Restores normal growth rate; alleviates deficient cyanide-sensitive respiration of [*mi-1*] ([*poky*]) and all other group I cytoplasmic mutants. No effect on group II cytoplasmic mutant [*mi-3*] (85, 204, 568). Cytochrome spectrum (204). Called *sup-10* (568).

***su([mi-1])-14*: suppressor-14 of [*mi-1*]**

IV. Linked to *arg-2* (14%) (*567*, 568).

Restores normal growth rate; alleviates deficient cyanide-sensitive respiration of [*mi-1*] ([*poky*]) and all other group I cytoplasmic mutants. No effect on group II cytoplasmic mutant [*mi-3*] (85, 204, 568). Cytochrome spectrum (204). Called *sup-14* (568).

***su([mi-1])-f*: suppressor-f of [*mi-1*]**

VR. Left of *inl* (10%) (*986*).

Restores normal growth rate; alleviates deficient cyanide-sensitive respiration of [*mi-1*] ([*poky*]) (693) and all other group I cytoplasmic mutants, but not group II or group III mutants (88, 395). *su([mi-1])-f* differs from other known [*mi-1*] suppressors in not restoring salicyl hydroxamic acid insensitivity (568). Cytochrome spectrum (85). Used for studying oscillations in membrane potential (402). Symbol changed from *f*.

***su([mi-3])-1*: suppressor-1 of [*mi-3*]**

IR. Linked to *al-2* (4%); right of *nit-1* (17%) (*395*).

Restores normal growth rate and cytochrome spectrum to [*mi-3*], a group II cytoplasmic mutant (89, 395). Does not suppress the mutant phenotype of group I or group III cytoplasmic mutants (88).

su(mtr)-1*: suppressor-1 of *mtr

IR. Right of *his-2* (2%) (*1018*).

Selected in the double mutant *trp-1; mtr* by increased uptake of tryptophan (1018) or as his^{+} revertants of *his-2; mtr* strains on histidine plus excess arginine (107). Still resistant to 4-methyltryptophan, but now sensitive to *p*-fluorophenylalanine (1018). The effect of *su(mtr)-1* on *mtr* is locus specific. *su(mtr)-1* appears to have a changed regulation for amino acid transport system II (776). Possibly allelic with lys^{R} (565).

***su*(mtr^{26}): suppressor of *mtr* allele 26**

VIR. Linked to *pan-2* and *trp-2* (*106*).

Allele-specific partial suppressor of the poor growth of the double mutant *mtr* (26); *trp-1* on low levels of tryptophan. [*mtr* (26) is a putative frameshift.] (106).

***su(pan-2^{Y153M66})*: suppressor of *pan-2* allele Y153M66**

Not mapped.

Allele-specific suppressor; not effective on three other *pan-2* alleles or on four super-suppressible alleles at *trp-3* and *am-1* (188).

***su(pe)*: suppressor of *pe* microconidiation**

Perhaps linked to *pe* (18%) in linkage group II (415).

Restores ability of the *col-1; pe* double mutant to produce macroconidia rather than exclusively microconidia (415, 416). Called su^m. See *col-1*.

su([poky])

See *su([mi-1])*.

su(pro-3)*: suppressor of *pro-3

IR. Linked to *al-2* (2%). Possibly allelic with *arg-6* (0/154) (*1129*).

Suppresses the requirement of the mutant *pro-3* (= *arg-8*) for proline, ornithine, citrulline, or arginine. This is due to a feedback-insensitive ornithine synthetic pathway (1129), which allows ornithine to spill over into the proline path.

su(rg-2)*: suppressor of *rg-2

Not linked to *rg-1* or *rg-2*.

Suppresses *rg-2* in *N. sitophila*. Found in *N. sitophila*. Not studied in *N. crassa*. (680).

su(trp-3)*: suppressor of *trp-3

Most suppressors of *trp-3* whose allele numbers are prefaced "td" were assigned numbers corresponding to the allele numbers of the td strains in which they were originally discovered. These have been retained as suppressor locus numbers. Thus, *su(trp-3^{td2})-2* is the original suppressor discovered in strain td2; the number 2 does not imply that there was a previously discovered suppressor of *trp-3* allele td2. This system was not used for the suppressors of td201, which are numbered conventionally. For a review of early work, see reference 1167.

***su(trp-3^{td2})-2*: suppressor-2 of *trp-3* allele td2**

III. Linked to *leu-1* (22%) (*581*).

Allele specific. Suppresses allele td2 but does not suppress td6, td71, or any other allele from td1 through td34 (580, 1169). This and the other listed suppressors of td2 were not tested on later *trp-3* alleles that fall in the same complementation group as td2. Although suppressed mutants grow on minimal medium, growth is stimulated by addition of tryptophan. Suppressed *trp-3; su* colonies are morphologically distinguishable from those of the wild type. Tryptophan synthetase is formed at levels below that of the wild type. Originated in *trp-3* allele td2 (originally numbered S1952) (1166, 1169). Called *su-2* (388) and su_2 (1169).

***su(trp-3^{td2})-2a*: suppressor-2a of *trp-3* allele td2**

I. Linked to *al-2* (15%) (*581*).

Isolated (1170) as one of four additional suppressors of td2, numbered su_{2a}, su_{2b}, su_{2c}, and su_{2d}. All are nonallelic with su_2 and with each other, with the possible exception of su_{2b} and su_{2c} (1170). None was tested for allelism with *su(trp-3^{td6})*, which also suppresses td2. su_{2b}, su_{2c}, and su_{2d} are unmapped. Erroneously printed as $su\text{-}2_a$ (581). The *a* does not refer to mating type.

***su(trp-3^{td3})*: suppressor of *trp-3* allele td3**

Not mapped.

Allele specific. Suppresses *trp-3* alleles td3, td24, and td71, but not any other alleles from td1 through td34 (580, 1169). Originated in *trp-3* (td3). Probably allelic with su24. Called su_3 (1169).

***su(trp-3^{td6})*: suppressor of *trp-3* allele td6**

Not mapped. Different locus from *su(trp-3^{td2})-2* and *su(trp-3^{td3})*.

Allele specific. Suppresses *trp-3* alleles td6 and td2, but does not suppress any other allele from td1 through td34 (580, 1169). Although suppressed mutants grow on minimal medium, growth is stimulated by the addition of tryptophan and is morphologically distinguishable

from that of wild type. Tryptophan synthetase is formed at levels below that of the wild type. Originated in *trp-3* (td6). Called su_6. (1169).

$su(trp\text{-}3^{td201})$-1: suppressor-1 of *trp-3* allele td201

VIIR. Between *met-7* (18%) and *arg-10* (7%) (*1174*). Right of *ssu-1* (10 to 13%) (954).

Allele specific. Suppresses missense allele td201, but not eight other alleles (td1, td6, td7, td16, td37R, td71, td138R, and td141) (1174). Also does not suppress td2, td3, or td24 (910). A suppressed mutant has a low level of tryptophan synthetase activity, allowing slow growth on minimal medium. Enzyme activity is due to a protein physically like the wild-type enzyme (1174, 1175). Strains carrying the suppressor alone, without td201, grow slightly slower than wild type (910). The suppressor is effective when in another nucleus from td201 in a forced heterokaryon between noncomplementing alleles (*td16; su* plus *td201; su*$^+$) (910). Called *Su-1* or $Su\text{-}1_{td201}$ in reference 954, *su-YS* in reference 910, and $su_1(201)$ in reference 1173.

$su(trp\text{-}3^{td201})$-2: suppressor-2 of *trp-3* allele td201

VII. Linked but not allelic with $su(trp\text{-}3^{td201})$-*1*; probably closer to the centromere (*910*).

Allele specific. Suppresses missense allele td201, but does not suppress six other alleles (td1, td2, td3, td16, td24, and td71) (910). The suppressed mutant has a low level of tryptophan synthetase activity, allowing slow growth on minimal medium. Enzyme activity is due to a protein physically distinguishable from wild-type tryptophan synthetase, unlike suppressors 1 and 3 (844). Suppressor alone, without td201, grows slightly more slowly than wild type (910). The suppressor is effective when in another nucleus from td201 in a forced heterokaryon between noncomplementing alleles (*td16; su* plus *td201; su*$^+$) (910). Called *su-R* in reference 910 and *su-2* in reference 844.

$su(trp\text{-}3^{td201})$-3: suppressor-3 of *trp-3* allele td201

Unmapped. Not linked to $su(trp\text{-}3^{td201})$-*1* or other VII markers.

Allele specific. Suppresses missense allele td201, but not four other alleles (td1, td6, td71, and td141). The one allele tested gave less powerful suppression of td201 than did the known alleles of $su(trp\text{-}3^{td201})$-*1*. The suppressed mutant has a low level of tryptophan synthetase activity, allowing slow growth on minimal medium. Restored enzyme activity is due to a protein physically like that of the wild type. Called su_3 and $su_3(201)$ (1173).

su(trp-5): suppressor of *trp-5*

VIL. Closely linked to *aro-6* (*J. A. Kinsey, personal communication*).

Possibly a feedback-negative allele of *aro-6*, which specifies 3-deoxy-D-arabino-heptulosonic acid-7-phosphate synthase (Tyr) (J. A. Kinsey, personal communication).

$su(ure\text{-}1^9)$: suppressor of *ure-1* allele 9

Unmapped, but not linked to *ure-1* or *ure-2*.

Does not suppress *ure-2* (allele 47). Suppressed only *ure-1* (allele 9) strains from a certain lineage, suggesting that suppression requires a cosuppressor closely linked to *ure-1* (9). (121).

suc: succinate

IL. Right of *acr-3* (2%). Left of *In(H4250)* and *phe-1* (1%) (578, 816). (*482*).

Uses acetate, succinate, or any of numerous related compounds (608). Most but not all strains grow better on acetate than on succinate (576). Lacks pyruvate carboxylase activity (72, 578, 1032). Numerous alleles are CO_2 remediable (108). Leaky, but less so on higher ammonium concentrations (1031). A special medium has been devised for selective plating (K. D. Munkres, cited in reference 816, p. 248). Most ascospores are poorly pigmented in homozygous *suc* × *suc* crosses. Allele KG163 shows greatly reduced recombination in the region between *leu-4* and *suc*, suggesting inversion heterozygosity (576). *suc* would be named *ace-6* except for priority of nomenclature (578).

sup: suppressor

See *su*.

sw: snow white

See *per-1*.

t: thermophobic

See *scot*.

T: tyrosinase

IR. Between *ad-3A* (18%) and *al-2* (*474*).

Tyrosinase structural gene (Fig. 11). Prototrophic. Multiple alleles distinguished electrophoretically and by thermolability (473–477). Null allele not known. Primary structure (407 amino acids) determined for the product of allele T^{L} (599). Scored by color reaction with DL-3,4-dihydroxyphenylalanine as a substrate (475, 477). Two forms of enzyme demonstrated in heterokaryons (474). For regulation, see references 332, 472, and 475 and references therein. Also see tyrosinase regulatory genes, symbolized *ty*.

T: (used as prefix to allele [isolation] numbers)

Has been used in allele numbers for mutants isolated at the Universities of Tokyo and Texas.

***T( ):* translocation**

Translocations are listed here only if they were used for mapping genes, centromeres, or chromosome tips. They can often be used for mapping by duplication coverage (analogous to deletion mapping in phages and other organisms). When an insertional or terminal translocation is crossed by normal sequence, independent segregation produces recombinant meiotic products that are duplicated for the translocated segment. Such a translocation involving linkage groups D (donor) and R (recipient) is symbolized *T(D→R)*. Crosses between two different reciprocal translocations that have breakpoints in the same two chromosome arms can produce recombinant meiotic products duplicated for segments between the displaced breakpoints. Simple reciprocal translocations involving linkage groups B and C are symbolized *T(B;C)*. For theory, diagrams, methods, review, and description of many additional translocations, see reference 808. Translocations listed as terminal are thought to be really quasiterminal, i.e., reciprocal translocations in which one breakpoint is very close to a tip and distal to all essential genes.

***T(AR18)*: insertional translocation**
T(IIL→IIIR)AR18

An interstitial segment of IIL is inserted in IIIR. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *het-6* but not *cys-3*, *het-c*, or *pyr-4* (729, 808).

***T(AR30)*: reciprocal translocation**
T(IIL;VL)AR30

A distal segment of IIL, with breakpoint mapping left of *pi* (30% recombination), is interchanged with a distal segment of VL, with breakpoint between *NO* and *caf-1* (19%) (600, 808, 817).

***T(AR33)*: terminal translocation**
T(VL→IVL)AR33

A distal segment of VL is translocated to the left tip of IV. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes the nucleolus organizer and *caf-1* but not *lys-1* or *at* (808, 817). Used to demonstrate demagnification of genes specifying rRNA (887).

***T(T39M777)*: terminal translocation**
T(VIL→IR)T39M777

A distal segment of VIL is translocated to the right tip of I. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *un-4* and markers distal to it, but does not include *cys-1* (808).

***T(T54M50)*: terminal translocation**
T(VIIL→IVR)T54M50

A distal segment of VIIL is translocated to the right tip of IV. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *thi-3* and *csp-2* but not *met-7* (808).

***T(STL76)*: reciprocal translocation**
T(IR;IIR)STL76

A distal segment of IL with a breakpoint between *cyh-1* and *os-5* is interchanged with IIR between *arg-12* and *ace-1*. When translocation *T(STL76)* is crossed with overlapping translocation *T(4637)*, viable duplication progeny result that contain two copies of the intervals between breakpoints of the two translocations. See *T(4637)* for included markers (808).

***T(NM103)*: terminal translocation**
T(IR→VIR)NM103

A distal segment of IR is translocated to the right tip of VI. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *met-6* and markers distal to it, but does not include *thi-1* (808, 1091).

T(NM149)*: terminal translocation *T(IIL→VR)NM149

A long segment of IIL is translocated to the right tip of V. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *ro-3* but not *thr-2*, *thr-3*, or *tng* (808, PB).

T(NM152)*: insertional translocation *T(IVR→I)NM152

An interstitial segment of IVR is inserted in I. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *pyr-3* through *pyr-2* and *mat*, but does not include *arg-14* or *cys-4* (238, 745, 808).

T(ALS159)*: terminal translocation *T(IVR→VIR)ALS159

A distal segment of IVR is translocated to the right tip of VI. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *pyr-1* and all IVR markers distal to it, but does not include *psi* (745, 808).

T(NM169d)*: terminal translocation *T(IR→VL)NM169d

A distal segment of IR is translocated to the left end of V. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *un-18* but not *R* (808, PB; B. C. Turner, personal communication).

T(AR173)*: complex duplication-generating translocation *T(IR,IR;V;VII)AR173

An interstitial segment of IR behaves as though inserted in V or VII. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *un-2*, *cyt-4*, and *his-2* but not *sn*, *nuc-1*, or *rg-1* (670, 808).

T(ALS176)*: terminal translocation *T(IIR→VL)ALS176

All or most of IIR is translocated to the left tip of V (nucleolus satellite). Viable duplication progeny from *T* × normal sequence contain two copies of the IIR segment, which includes *arg-5* and all markers distal to it, but does not include *bal* (808, 809).

T(NM177)*: insertional translocation *T(IIR→IL)NM177

An interstitial segment of IIR is inserted in IL. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which contains *nuc-2* through *arg-12*, but does not include *aro-3* or the *aro* cluster gene (671, 808; A. Kruszewska, personal communication).

***T(ALS179)*: translocation *T(VIIL→IVR)ALS179* (probably terminal)**

A far-left segment of VIIL is translocated to the right end of IV. Viable duplication progeny from *T* × normal sequence are inferred to contain two copies of the segment, which includes no known genic markers (808).

T(AR179)*: complex duplication-generating translocation *T(IIL;IIL;IVR;VL)AR179

A long segment of IIL is duplicated in one class of progeny from *T* × normal sequence. The segment includes *thr-2* and markers distal to it, but does not include *bal* or *arg-5* (808).

T(ALS182)*: terminal translocation *T(IR→VL)ALS182

A distal segment of IR is translocated to the left tip of V (nucleolus satellite). Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *met-6* and markers distal to it, but does not include *thi-1* (670, 809).

T(AR190)*: terminal translocation *T(IR→VL)AR190

Most of IR is translocated to the nucleolus satellite at the left tip of V. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *his-2* and markers distal to it, but does not include *un-2* (670, 808).

T(AR209)*: terminal translocation *T(VIR→IVR)AR209

All or most of VIR is translocated to the right tip of IV. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *pan-2* and probably *rib-1*, but does not include *ad-1* or *ylo-1* (808).

T(D305)*: complex duplication-generating translocation *T(IIIR;IIIR;VIL;X?)D305

A distal segment of IIIR is duplicated in one class of progeny from *T* × normal sequence. These duplication progeny contain two copies of the segment, which includes *phe-2* and *dow* but not *ro-2* (808, 809).

T(OY320)*: terminal translocation *T(VIR→IIIR)OY320

A distal segment of VIR is translocated to the right tip of III. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *ws-1* but not *trp-2* (808).

T(OY321)*: nearly terminal translocation *T(IL→VL)OY321

A distal segment of IL is translocated to the nucleolus organizer in VL. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *cyt-1* and markers distal to it, but does not include *leu-4*. The viable duplications contain only the proximal portion of the nucleolus organizer (808; D. D. Perkins, N. B. Raju, and E. G. Barry, in preparation).

T(S1229)*: insertional translocation *T(IVR→VII;IL;IIR;IVR)S1229 arg-14

An interstitial segment of IVR is inserted in VII. The right breakpoint in IV is inseparable from *arg-14*. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *pt*, *cys-15*, and *mtr* through *arg-2*, but does not include *pdx-1* or *pyr-3*. (Reference 238 is incorrect in showing *pdx-1* included.) (54, 55, 238, 808).

T(P2869)*: insertional translocation *T(IIL→VI)P2869

An interstitial segment of IIL is inserted in VI. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *ro-7*, *pi*, and *het-6* but not *het-c* or *pyr-4* (808).

T(S4342)*: insertional translocation *T(IVR→IIIR)S4342

An interstitial segment of IVR is inserted in IIIR. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *arg-14* through *uvs-2*, but does not include *arg-2* (238, 808).

T(4540)*: insertional translocation *T(IR→IIIR)4540 nic-2

An interstitial segment of IR is inserted in IIIR. The left IR breakpoint is inseparable from *nic-2*. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *cr-1*, *cys-9*, and *un-1* but not *ad-3B* or *thi-1* (808, 908).

T(4637)*: reciprocal translocation *T(IR;IIR)4637 al-1

A distal segment of IR with breakpoint at *al-1* is interchanged with IIR (left of *arg-12*). When *T(4637)* is crossed with overlapping translocation *T(STL76)*, viable duplication progeny result that contain two copies of the intervals between breakpoints of the two translocations. The duplicated segment includes IR markers *un-7* and *os-5* through *hom* but does not include *cyh-1* or *lys-3* (808; D. D. Perkins, unpublished data).

T(5936)*: terminal translocation *T(VIIR→IL)5936

A distal segment of VIIR is translocated to the left tip of I. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *arg-11* and markers distal to it, but does not include *dr* (808, PB).

T(39311)*: insertional translocation *T(IL→IIR)39311

An interstitial segment of IL is inserted in IIR. Viable duplication progeny from *T* × normal sequence contain two copies of the segment, which includes *nit-2* through *csp-1* but does not include *un-5*, *sn*, or *os-4* (798, 808, 809).

***ta*: tufted aerial**

IL. Between *un-16* (2%) and *acr-3* (<3%) (*816*, PB).

Rapid-spreading colonies. Morphology is distinguishable from that of the wild type, but varies with growth conditions (816). Conidiation best at 34°C. Possible maternal effect, with ta^+ sometimes resembling *ta* in initial cultures from ascospores. (D. Newmeyer, unpublished data).

***td*: tryptophan desmolase (synthetase)**

See *trp-3*.

***tet*: tetrazolium**

IL. Right of mating type (7 to 8%). Linked to *acr-3* (2%) and *ad-3B* (1%) (*395*).

Tetrazolium dye reduction. Detected as a difference between *A* and *a* laboratory strains (74-OR23-1A colonies fail to reduce dye and thus remain white; 74-OR8-1a colonies reduce dye to red) (395). Called *Tet-R* and *Tet-W* ("red," "white"). See reference 395 for tests on other wild types. *cya, cyb*, and *cyt* mutants all fail to reduce 2,3,5-triphenyl-tetrazolium chloride, and this is used as a test in the initial identification of such mutants. *cya-1, cyt-3*, and *cyt-4* are all located near the mating type locus (87); their relationship to *tet* is not known.

Probably (but not certainly) the same gene is responsible for the mating-type-linked difference in resistance to 2,3,5-triphenyl-tetrazolium chloride. 2,3,5-triphenyl-tetrazolium chloride-resistant strains include 74-OR23-1A, Em A FGSC no. 691, Em A 5256, Lindegren 1A, Lindegren 25a, and all RL wild types tested. 2,3,5-Triphenyl-tetrazolium-sensitive strains include 74-OR8-la, Em a FGSC no. 692, and Em a 5297 (1116, 1117). Oak Ridge *a* wild-type strain ORSa, derived by backcrossing to 74-OR23-1A, is resistant (731). The gene for 2,3,5-triphenyl-tetrazolium chloride resistance maps left of *mei-3*, probably between *mt* and *arg-1* (D. R. Galeazzi, personal communication).

***thi-1*: thiamine-1**

IR. Right of the *T(4540)* right breakpoint and *cys-9* (13%). Left of *T(NM103), T(ALS182)*, and *met-6* (7 to 14%) (721, 808, 816, 1091). (*482*).

Uses thiamine or precursors pyrimidine plus thiazole (1059). Adaptation to growth on minimal medium occurs after a lag; growth tests should, therefore, be scored early. Adaptation is not carried over via ascospores, conidia, or small mycelial fragments. Adaptive growth is paralleled by attainment of wild-type thiamine pyrophosphate and carboxylase levels. Apparently concerns utilization of intact thiamine rather than its biosynthesis. (302, 303). Allele 17084 is inseparable from translocation *T(IR;VII)17084* (808).

***thi-2*: thiamine-2**

IIIR. Between *his-7* (1 to 2%) and *ad-2* (1 to 3%) (219, 814). (*504*).

Requires thiamine. Cannot use pyrimidine plus thiazole (1059). Does not undergo growth adaptation on minimal medium (302).

***thi-3*: thiamine-3**

VIIL. Between *nic-3* (9 to 18%) and *T(T54M50)* (808, 812, 816). (*482*).

Uses thiamine or thiazole (1059). Does not undergo growth adaptation on minimal medium. Growth on minimal medium is leaky at first, but becomes tight with exhaustion of endogenous thiazole (302), so scoring is best done late.

***thi-4*: thiamine-4**

III. Linked to *acr-2, spg*, and *sc* (<1%). Left of *pro-1* (3%) and *ace-2* (4 to 9%) (812). (*482*).

Requires thiamine (301). Anomalous in condensation of the pyrimidine and thiazole precursors. Very leaky (301).

A probable allele called "*thi-lo*" greatly increases the thiamine requirement of *thi-1* strains and decreases the ability to synthesize thiamine from pyrimidine and thiazole. *thi-lo* strains have no detectable nutritional requirement in the absence of *thi-1*. *thi-lo* failed to recombine with *thi-4* among 55 scorable progeny (301).

***thi-5*: thiamine-5**

IVR. Linked to *pan-1* (1%) (*812*).

Uses thiamine (482). Probably 2-methyl-4-amino-5-aminomethyl pyrimidine can also be used (799).

***thi-lo*: thiamine-low**

See *thi-4*.

***thr-1*: threonine-1**

Name changed to *ile-1*, q.v. Because this locus specifies threonine dehydratase, the original name *thr-1* is inappropriate (549, 552). See Fig. 15. Called *thre-1* in reference 240.

***thr-2*: threonine-2**

IIL. Right of *T(NM149)* and *ro-3* (6 to 25%). Left of *T(AR179)*; hence, left of *bal* and *arg-5* (3 to 18%). Adjoins *thr-3* (<0.1%) (808, 812, PB).

Requires threonine. Cannot use other amino acids (1061). Lacks threonine synthetase (352) (Fig. 17). Strongly inhibited by methionine (320, 1061). Known alleles are not heat sensitive, unlike all known *thr-3* alleles.

***thr-3*: threonine-3**

IIL. Linked to *thr-2* (<0.1%) (*812*).

Requires threonine. Also responds slightly to α-aminobutyric acid or isoleucine. Known alleles are heat sensitive (25°C versus 34°C), requirement is tight at 34°C. Not inhibited by methionine (320, 1061).

***ti*: tiny**

IL. Between *arg-3* (1%) and the *T(39311)* right breakpoint (PB) (*789*).

Heat sensitive. Spreading colonial morphology at 25°C or below; more restricted at 30°C; no growth at 34°C (789, PB). Can be scored microscopically after ascospore germination. Reduced amount of cell wall peptides (1165).

timex

Symbol formerly used to designate *bd; inv* double mutants. See *bd*.

Tip: (chromosome tip)

Eight chromosome tips have been defined by mapping the breakpoints of terminal translocations relative to gene loci. Because a class of meiotic segregants from these translocations are viable that would necessarily be deficient for any chromosomal segment distal to the terminal breakpoint, it is known that no essential gene loci are located beyond the site of translocation. Although these rearrangements behave genetically as terminal, they are thought to be reciprocal translocations in which the telomere is translocated. They would, therefore, more accurately be called quasiterminal. See references 467, 756, and 808. The VL tip has also been mapped by an independent method, using the heteromorphic satellite as a terminal cytological marker. Remaining tips are simply listed as located beyond the most distal known gene marker.

Tip IL: left tip of linkage group I

Marked by terminal translocation *T(5936)*, which is linked to *ro-10* (0/38) and *fr* (11%) (808, PB).

Tip IR: right tip of linkage group I

Marked by terminal rearrangements *In(NM176)*, *In(H4250)*, and *T(T39M777)*, which are closely linked to *R* and *un-18* (808, 1093; H. R. Cameron, personal communication).

Tip IIL

Left of *T(AR30)*, which is left of *pi* (~30%) (600). An extended unmarked segment left of *pi* is also suggested by data from *T(NM149)* (D. D. Perkins, unpublished data).

Tip IIR

Right of *rip-1* and *un-15*.

Tip IIIL

Left of *r(Sk-2)-1*, *cum*, *acr-7*, *acr-2*, and *thi-4*.

Tip IIIR

Marked by *T(OY320)*, which is linked to *dow* (6%) (PB).

Tip IVL

Marked by *T(AR33)*, which is linked to *cys-10* (0/221) and left of *acon-3* (<1%) (817, PB).

Tip IVR

Marked by *T(AR209)*, *T(T54M50)*, and *T(ALS179)*, which are right of *uvs-2* (2 to 6%) and *cys-4* (817).

Tip VL

Marked by *sat*, *T(ALS176)*, *T(ALS182)*, and *T(AR190)*, which are left of *T(AR30)*, *caf-1* (>11%), and *lys-1* (20 to 35%) (60, 600, 808, PB).

Tip VR

Marked by *T(NM149)*, which is linked to *his-6* (0/499) (808; D. D. Perkins, unpublished data).

Tip VIL

Left of *chol-2*.

Tip VIR

Marked by *T(NM103)* and *T(ALS159)*, which are right of *trp-2* (13%) (808, 1091; D. D. Perkins, unpublished data). Right of *ws-1* and *un-23*.

Tip VIIL

Left of *T(ALS179)*, *het-e*, and *su([mi-1])-5*.

Tip VIIR

Right of *sk*.

***tng*: tangerine**

II. Right of *T(NM149)* and *pyr-4* (16%). Left of *arg-5* (6 to 14%) and probably left of *thr-2* (2%) (*PB*).

Irregular spreading growth. Hyphae not curled (unlike *ro*), and not as densely branched as *col-4*. Conidia formed in irregular patches. Many conidia are large, with 5 to 20 nuclei (allele P4474) (N. B. Raju, personal communication). Resembles *ro-9* (R2526) strains in gross appearance, and the genes map in similar locations. Both are female infertile. (PB).

***tol*: tolerant**

IVR. Linked to *trp-4* (~1%), probably to the left (*755*).

Suppresses the vegetative (heterokaryon) incompatibility associated with mating type alleles *A* and *a*, but does not affect sexual compatibility. (*tol; A + tol; a*) heterokaryons are fully compatible and stable if other *het* loci are homokaryotic, and *A/a* duplications grow normally when *tol* is present (755). Recessive (252); see reference 746, however, for a stable mixed-mating type heterokaryon that is (*tol a + tol*$^+$ *slime A*). *tol* does not suppress the vegetative incompatibility of differing alleles at *het-c* or *het-e* (755, 803). Mutation or deletion of *tol*$^+$ restores normal growth rate to slow-growing, unstable, mixed-mating-type (*tol a + tol*$^+$ *A*) heterokaryons (252). *tol* is present in some isolates from nature and has arisen at least twice by mutation in laboratory stocks (755, PB; O. C. Yoder, personal communication). Double-mutant *tol trp-4* stocks are convenient because the closely linked *trp-4* tags the *tol* allele, which otherwise requires progeny tests for scoring. Used to maintain stable *A + a* heterokaryons, allowing the desired component to be used as the parent in a cross (746). Homozygous *tol* may partially restore fertility to the mutant *fmf-1* (531).

***Tp*( ): transposition**

Used to designate rearrangements having a segment transposed from one interstitial position to another in the same chromosome. In crosses of *Tp* by normal sequence, crossing over in the segment between the two positions can generate recombinant meiotic products that are duplicated for the transposed segment. These can be used for mapping by duplication coverage (808).

Tp(T54M94)*: transposition *T(IR→IR)T54M94

An interstitial segment of IR is transposed proximally and inserted in inverted order. Viable duplication progeny produced by crossing over in crosses of *Tp* × normal sequence include *nit-1* through *al-2*, but do not include *ad-9* or *arg-6* (808, 809; B. C. Turner, personal communication).

***tr*^{u}: transport of uracil**

See *uc-5*.

Transport mutants

Mutations affecting transport have been obtained by many techniques, using metabolites, antimetabolites, etc. Nomenclature has consequently been chaotic. Transport mutations are listed under the following entries.

(i) Amino acid transport: Basic (*pmb*, = *bat*); neutral (*mtr*, = *pmn*); and general [*pmg*; see also *su(mtr)-1*]. Individual genes have been symbolized (e.g., references 248 and 1152) as *pm b* or *Pm*$^-$ *B*, and compound mutants, such as *pmn; pmb*, have been symbolized *pm nb* or *Pm*$^-$ *NB*. Symbols in the present article are changed from this nomenclature so as to show that *pmn* and *pmb* are separate genes. Other mutant genes that may involve amino acid transport are *arg*^{R}, *lys*^{R}, *hlp-1*, and *hlp-2*.

(ii) Transport or possible transport mutations for other metabolites or ions: *acp*^{i}, *car, cys-13, cys-14, fpr, glt, ipm-1, ipm-2, mea-1, sit, sor, trk, tys, uc-5*, and *ud-1*.

(iii) Mutations that appear to affect more than one transport system: *hgu-4, nap, fpr-6, mod-5, mts*, and *un-3*; see especially reference 1149.

(iv) There are also general regulatory loci that control many related enzymes including the relevant permeases, e.g., *nit-2, cys-3, pcon (nuc-2), preg, pgov*, and *nuc-1*. Transport of various ions and compounds is reviewed in references 406 and 921; amino acid transport is reviewed in reference 1150, and peptide transport is reviewed in reference 1151.

***tre*: trehalase**

IR. Between *met-6* (7%) and *al-2* (20 to 37%); near *mig* (≤1%) (*1045*, 1176). Shown between

met-6 and *ad-9* (7%) in reference 466, with only two-point data given.

Unable to use trehalose as carbon source. Lacks trehalase activity. (Also, the invertase level is reduced 50% and the amylase level is increased.) In (*tre* + *tre*$^+$) heterokaryons, the trehalase level is reduced to 10% of the wild-type level, and in *tre*/*tre*$^+$ duplications the level is reduced to 1% of the wild-type level. *tre* codes for trehalase inhibitor, probably a protein (1045). For a possibly related regulatory mutation, see reference 663. For putative structural gene, see *mig*.

trk: transport of potassium

IIIR. Linked to *leu-1* (0/92) (*988*).

Requires high K^+ concentration for growth. Na^+ cannot substitute. Cation transport system maximum velocity is normal; K_m is three times normal. Recessive. Obtained by inositol death enrichment on low potassium. (988).

tRNA synthetase

See *leu-5* and *trp-5*.

trp: tryptophan

Tryptophan is required in much higher concentrations than its precursors, anthranilic acid and indole. A 0.01-mg/ml amount of the precursor is sufficient, but up to 0.1 or 0.2 mg of tryptophan per ml is required by some mutants. High concentrations of anthranilic acid are toxic. Most *trp* mutants grow better on 0.2 mg of tryptophan per ml plus 0.2 mg of phenylalanine per ml than on tryptophan alone. For the biosynthetic pathway, see Fig. 11. Also called *tryp* and *try*. *trp-3* called *td*. See also *nt*.

Tryptophan feedback inhibits anthranilate synthase, anthranilate phosphoribosyltransferase, and one of three isozymes of 3-deoxy-D-arabinoheptulosonic acid-7-phosphate synthase (the first step in aromatic biosynthesis). Tryptophan stimulates chorismate mutase, directing chorismate to prephenate rather than to anthranilate synthesis. All four genes of the tryptophan pathway are derepressed by starvation for tryptophan. High indoleglycerol-phosphate levels also cause derepression. Derepression may involve inhibition of Trp-tRNA synthetase. (40, 152, 258, 436, 603, 742, 1004, and references therein). Genes for tryptophan biosynthesis are derepressed coordinately with those for histidine, arginine, and lysine biosynthesis; this is called "cross-pathway regulation" (137, 1131); reviewed in reference 642; see *cpc-1*.

trp-1: tryptophan-1

IIIR. Between *ad-2* (1 to 7%) and *ro-2* (2 to 12%) (11, 219, 812). Linked to *fpr-3* (<1%) (550). (*504*).

Uses tryptophan or indole (1060); strains carrying some alleles can also use anthranilate; others cannot (4). *trp-1*$^+$ and *trp-2*$^+$ gene products together form an enzyme aggregate with three activities: anthranilate synthetase, phosphoribosyl-anthranilate isomerase, and indoleglycerol-phosphate synthetase (181, 260) (Fig. 11). *trp-1* codes for the beta subunit of the aggregate (546); it specifies phosphoribosyl-anthranilate isomerase, indoleglycerol-phosphate synthetase, and collaboratively the glutamine amino transferase activity of anthranilate synthetase (29, 181, 502). Strains carrying different alleles differ in lacking one or more of the three activities, e.g., *trp-1* (allele 15) lacks all three activities; *trp-1* (20) lacks only phosphoribosyl-anthranilate isomerase, *trp-1C* (1) lacks only anthranilate synthetase, *trp-1* (25) lacks both phosphoribosyl-anthranilate isomerase and indoleglycerol-phosphate synthetase, etc. (259). (To avoid confusion, note that in reference 259 and related papers, the same "allele number" may be used for a *trp-2* mutation, a *trp-1* mutation [non-anthranilate-utilizing], and a *trp-1C* mutation [anthranilate utilizing]; mutations of the last class are listed by FGSC as *trp-1* with the allele number prefixed by C.) Strains carrying different alleles differ in their ability to form aggregates (181, 259). Association between *trp-1* and *trp-2* products is essential for glutamine-dependent anthranilate synthetase activity but not for the other two activities (181). The *trp-1* gene has been cloned (545, 925), sequenced (925), and reintroduced into *Neurospora* by transformation (925). It is only partially expressed in *E. coli*. Fine-structure maps (10, 259). Complementation maps (10, 163). Reviewed as example of gene fusion (218). Nonsense allele used to demonstrate restoration of normal enzyme aggregate by supersuppressors (183). Alleles that accumulate anthranilate are scorable by blue fluorescence under long-wave UV after 2 to 5 days of growth on minimal medium plus indole (10 μg/ml), 34°C (814, 816). Aging cultures may produce brown pigment; blue fluorescence disappears as pigment forms.

trp-2: tryptophan-2

VIR. Right of *del* (0 to 13%). Left of *un-23* (5 to 27%), *T(OY320)*, and *ws-1* (38%) (818, *822*, 1019, PB).

Uses kynurenine, anthranilic acid, indole, or tryptophan (96). Kynurenine is utilized by con-

version to anthranilate (447). Inferred to be the structural gene for the alpha subunit of the anthranilate synthetase complex (546). The gene product catalyzes anthranilate synthesis with ammonia but not with glutamine as the amino donor (29). Specifies anthranilate synthetase (glutamine linked) in collaboration with *trp-1* in trifunctional *trp-1*$^+$–*trp-2*$^+$ enzyme aggregate (181, 259) (Fig. 11); see *trp-1*. Nonsense allele used to isolate supersuppressors (954) and to study enzyme complex restored by supersuppressors (183).

***trp-3*: tryptophan-3**

IIR. Right of *fl* (2 to 6%). Left of *rip-1* (9%) and *un-5* (10%) (816, PB). (*1166*).

Uses tryptophan (685); strains carrying some alleles also use indole (4). Structural gene for tryptophan synthetase (1167), called tryptophan desmolase in early literature. Tryptophan synthetase catalyzes three reactions: indoleglycerol-phosphate → tryptophan, indole → tryptophan, and indoleglycerol-phosphate ⇄ indole (Fig. 11). In *Neurospora*, all three reactions are catalyzed by a single protein, which is specified by a single gene (645, 1167). Mutants lack indoleglycerol-phosphate → tryptophan activity but differ with respect to the other activities; e.g., strains carrying *trp-3* allele (td141) are blocked in indoleglycerol-phosphate utilization but can use indole; *trp-3* (td100) can synthesize indole but not convert it to tryptophan; *trp-3* (td140) lacks all three activities. (See references 582 and 1049 for citations and characteristics of other mutants.) Used extensively for studies of gene structure in relation to enzymatic activity (257, 582 and references therein, 1167). The active enzyme is a homooligomer (645) thought to have two domains (644 and references therein). Biochemical studies of complementation between alleles: in vivo (582, 583) and in vitro (1048 and references therein). Complementation maps (4, 5, 9 and references therein, 582). Fine-structure maps (5, 540, 582, 1049). Reviewed as example of gene fusion (218). *trp-3* mutant C83 provided the first proved example in *Neurospora* of gene-controlled loss of enzyme activity (685); *trp-3* mutant S1952 provided the first example of allele-specific suppression restoring functional wild-type-like enzyme (1166). Allele td140 is supersuppressible (954, 955). Certain classes of *trp-3* mutants are osmotic remediable (583). Called *td* and *tryp-3*.

***trp-4*: tryptophan-4**

IVR. Between *his-5* (3 to 7%) and *leu-2* (1 to 2%) (633, 991). (*47*).

Uses tryptophan or indole (750). Deficient in anthranilate phosphoribosyl transferase (1126) (Fig. 11). Scorable by blue fluorescence (anthranilate) in medium under long-wave UV after 2 to 5 days of growth on minimal medium plus indole (10 μg/ml), 34°C. Initial stocks of the first *trp-4* mutant were inhibited by suboptimal concentrations of tryptophan (750), but derivatives have been obtained that are free of this problem (909).

***trp-5*: tryptophan-5**

VR. Right of *pab-1* and *inl* (4%). Left of *met-3* (4%) (6, PB). (*14*).

Uses anthranilate, indole, or tryptophan (14). Tryptophanyl-tRNA synthetase activity is <5% that of the wild type (742). Anthranilate synthetase and tryptophan synthetase derepressed (742). Not temperature sensitive.

***try, tryp*: tryptophan**

Changed to *trp*.

***ts*: tan spore**

VR. Linked to *inl* (4%) (*734*).

Ascospores slow to mature, remaining light brown when wild-type ascospores have blackened. Expressed autonomously, allowing visual scoring in heterozygous asci. Only a small minority of *ts* ascospores, that have darkened with age, are capable of germination. Photograph of asci (734). Used to study multiple (737) and selective (314) fertilization, preferential segregation (735), and factors affecting crossing-over frequency (586, 736).

***tu*: tuft**

IIR. Between *pe* (8%) and *fl* (19%) (*613*).

Conidia mostly in clusters at top of slant (613). (Stock lost.)

***ty*: tyrosinase**

This symbol is used for regulatory genes *ty-1*, *ty-2*, *ty-3*, and *ty-4*. Strains carrying these genes are prototrophic, growing on unsupplemented minimal medium. The structural gene is symbolized *T*.

***ty-1*: tyrosinase-1**

IIIR. Linked to *tyr-1* (0 to 6%) and *dow* (21%) (*1115*, PB).

Tyrosinase repressed. Recessive (475, 477). Uninducible by protein synthesis inhibitors in the sexual phase but inducible in vegetative culture (472, 477). Prototrophic. Velvet-like vegetative morphology. Female infertile (no or few perithecia) but fertile as male. Infertility is recessive in heterokaryons (475). Low ascospore viability (459). Scored by color reaction with DL-3,4-dihydroxyphenylalanine as the substrate (475, 477) or by morphology and female sterility.

_ty-2_: tyrosinase-2

IR. Right of *al-2* (*459*).

Tyrosinase repressed. Recessive (475, 477). Uninducible by protein synthesis inhibitors, e.g., cycloheximide, in the sexual phase, but inducible in vegetative culture (472, 477). Short aerial hyphae (459), although described earlier as morphologically normal (475). Female infertile but fertile as the male. Infertility is recessive in heterokaryons (475). Prototrophic. Score by color reaction with DL-3,4-dihydroxyphenylalanine as the substrate (475, 477) or by morphology or female sterility.

_ty-3_: tyrosinase-3

IIIR. Near the centromere; left of *ad-4* (*W. L. Chan, Ph.D. thesis, University of Malaya, Kuala Lumpur, 1977, cited in reference 460*). Not allelic with *T* or *ff-3* (193; N. H. Horowitz and H. Macleod, cited in reference 332).

Tyrosinase repressed. Uninducible by protein synthesis inhibitors, e.g., cycloheximide, in vegetative culture (322, 460; N. H. Horowitz and H. Macleod, cited in reference 332). Originally reported to be female sterile and morphologically abnormal; subsequently, these properties were shown due to a second, nonallelic mutation, *ff-3*. The *ty-3* single mutant is female fertile and morphologically normal (193). Score by color reaction with DL-DOPA as the substrate (475, 477). The original strain containing both *ty-3* and *ff-3* is called T22; it also contained *ty-4*, q.v.

_ty-4_: tyrosinase-4

Unmapped.

Tyrosinase repressed. Uninducible by total starvation in sodium phosphate buffer, but can be induced by cycloheximide. Found in strain T22, which also contains *ty-3* and *ff-3* (460; W. L. Chan, Ph. D. thesis, University of Malaya, Kuala Lumpur, 1977, cited in reference 460); also present in some Emerson wild types (460).

tyr-1: tyrosine-1

IIIR. Right of *vel* (3 to 5%) and *phe-2* (2 to 4%). Left of *un-17* (4%) (316, 816, PB; R. L. Metzenberg, personal communication). (*47*).

Requires tyrosine (1055). Lacks prephenate dehydrogenase activity (40, 316) (Fig. 11). Shows phenotypic adaptation after a lag, attaining the wild-type growth rate on minimal medium. Adaptation is not carried through conidia (1055). Allele UT145 formerly called *tyr-3* (316).

tyr-2: tyrosine-2

IR. Between the *T(4540)* breakpoints; hence, between *nic-2* and *thi-1* (10%). Probably left of *cr-1* (2%) (*812*, PB).

Requires tyrosine (812). Decreased prephenate dehydrogenase activity (316); *pe*-like morphology. Female sterile. tyr^- ascospores not fully pigmented. Growth is suboptimal even on fully supplemented medium. Scoring by growth on slants is treacherous; adapts to growth on minimal medium after several days. Can be scored by darkening of tyrosine-supplemented minimal medium.

tyr-3: tyrosine-3

See *tyr-1*.

tyr-s

See *tys*.

tys: tyrosine sensitive

I. Right of mating type (6%) (PB) (*1156*).

Growth inhibited 80% by 0.07 mM L-tyrosine. Growth of *tys* strains on minimal medium is also inhibited by glycyl-leucyl-tyrosine and by various tyrosine analogs. Uptake of L-*p*-[^{14}C]tyrosine is increased slightly, but this is not proposed as the cause of the inhibition. Primary defect unknown. (1156). Used to obtain the oligopeptide transport mutant *glt*, which is resistant to glycyl-leucyl-tyrosine but not to tyrosine (1155). Called *tyr-s* (1155, 1156).

_uc_: uracil salvage or uracil uptake

Symbol used in references 808b and 810 for mutations affecting the thymidine salvage pathway (*uc-1, -2, -3, -4*) or transport of pyrimidine bases (*uc-5*) (Fig. 23). Not used for biosynthetic

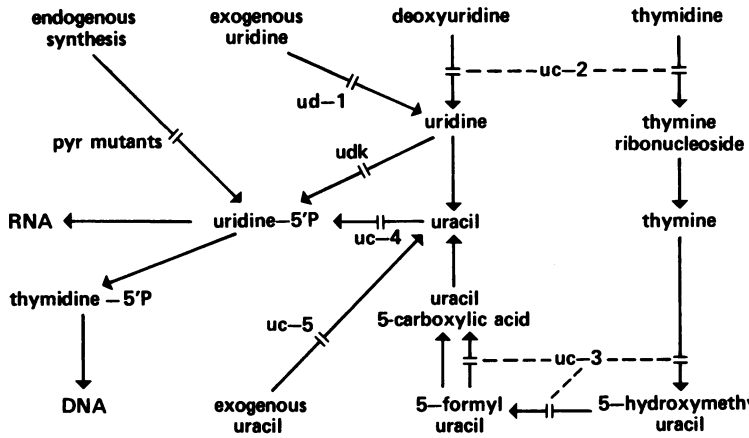


FIG. 23. Uracil salvage pathway, by which many exogenously supplied pyrimidines are cycled via uracil to uridine 5'-phosphate and, thus, into the latter part of the pyrimidine biosynthetic pathway. The figure shows the sites of action of the salvage pathway *uc* genes, the *ud* and *uc-5* genes, which control uptake of exogenous uridine and uracil, and *udk*, which controls uridine kinase (126, 420, 616, 977, 1141). For endogenous pyrimidine synthesis, see Fig. 20.

pathway mutants (see *pyr* for uracil biosynthesis).

***uc-1*: uracil salvage-1**

II. Linked to *pyr-4* (31%) (*1139*, 1141).

Altered thymidine salvage pathway (Fig. 23). Able to use thymidine, thymine, 5-hydroxymethyluracil, or 5-formyluracil as the sole pyrimidine source in germinating conidia of *pyr-4* mutants, which are blocked in pyrimidine biosynthesis. (In *uc-1*$^+$background, *pyr-4* can use these compounds only if a primer of uridine or cytidine is supplied [1141]). Causes elevated activities of enzymes which oxidatively demethylate thymine (420).

***uc-2*: uracil salvage-2**

I. Linked to *mt* (0/12 asci) (*1139*, 1141).

Thymidine salvage pathway defect (Fig. 23). Unable to use thymidine or deoxyuridine, but can use thymine, 5-hydroxymethyluracil, 5-formyluracil, uracil, or uridine as the sole pyrimidine source for *pyr-4* when *uc-1* is also present (1141). Reduced activity of the 2'-hydroxylase reactions thymidine → thymine ribonucleoside and deoxyuridine → uridine (977).

***uc-3*: uracil salvage-3**

Not mapped.

Thymidine salvage pathway defect (Fig. 23). Unable to use thymidine, thymine, or hydroxymethylcytosine, but can use deoxyuridine, 5-formyluracil, uracil, or uridine to support growth of *pyr-4* when *uc-1* is also present. Excretes thymine into the medium when grown in the presence of thymidine (1141). Lacks thymine 7-hydroxylase, which catalyzes the reactions thymine → 5-hydroxymethyluracil → 5-formyluracil → uracil-5-carboxylic acid; apparently, there is an alternate enzyme for 5-formyluracil → uracil-5-carboxylic acid, which explains the growth on 5-formyluracil (616, 977).

***uc-4*: uracil salvage-4**

VR. Between *inl* (12%) and *his-6* (11%) (*126*). Not in I as reported in references 1139 and 1141 on the basis of one ascus in eight recombinant with *mt*.

Thymidine salvage pathway defect (Fig. 23). Unable to use thymidine, thymine, 5-hydroxymethyluracil, 5-formyluracil, or uracil to support *pyr-1*, even in the presence of a uridine primer (1141). Deficient in phosphoribosyl transferase (127). The *uc-4* single mutant converts 50% of the supplied uridine to uracil and excretes it into the medium (1141). Resistant to 5-fluorouracil (126).

***uc-5*: uracil uptake-5**

IV. Right of *cot-1* (32%) (126). (*1139*, 1141).

Apparently defective in transport of pyrimidine bases (Fig. 23). Unable to use any free

pyrimidine base to support the growth of the pyrimidine auxotroph *pyr-1*, although it can use both ribose- and deoxyribose-nucleosides (1141). Resistant to 5-fluorouracil (126). The *pyr-1 uc-5* double mutant has been used to study uptake inhibition of structural analogs (225). Selected by inability to use uracil to supplement *pyr* auxotrophs, or as 5-fluorouracil resistant on ammonia-free minimal medium (126). Uracil uptake is decreased by NH_3 (629) or by other good nitrogen sources, and by a *nit-2* mutation (128). Called tr^u.

_ud-1_: uridine uptake-1

VIIR. Between *met-7* (27%) and *arg-10* (10%) (126). (*463*).

Unable to use pyrimidine nucleosides to support the growth of a pyrimidine auxotroph (*pyr-1*) although any free pyrimidine base can be used. Probably defective in pyrimidine nucleoside transport (1141) (Fig. 23). Apparently also defective in purine nucleoside transport (224). Resistant to 5-fluorodeoxyuridine and 5-fluorouridine (126). Resistance is recessive in heterokaryons. Shows interallelic complementation (127). Scored by spotting conidial suspension on medium containing 4×10^{-5} M 5-fluorodeoxyuridine, filter sterilized (463). Mutant gene CIF-dUrd 7, selected by resistance and called *fdu-1* (463), is allelic (126). Uridine uptake is decreased by good nitrogen sources and by *nit-2* (128).

udk: uridine kinase

VR. Left of *uc-4* (29%) (*126*).

Deficient in uridine kinase (127) (Fig. 23). The *udk uc-4* double mutant is resistant to 5-fluorodeoxyuridine and 5-fluorouridine (126), but the *udk* single mutant is not resistant to any analog.

ufa-1: unsaturated fatty acids-1

IV or V. Linked to *inl* in a cross with *alcoy* (944). Data do not distinguish linkage group because *T(IVR;VR)R2355* was present.

Requirement satisfied by C_{16} or C_{18} fatty acids having a double bond in *cis* at either the Δ^9 or Δ^{11} position, by C_{16} fatty acids with double bond in *trans* at Δ^9, or by fatty acids with multiple *cis* double bonds interrupted by methylene bridges (944, 945). Reverts readily, and revertants overgrow culture if grown on suboptimal supplement (S. Brody, personal communication). Tween 80 (0.1%) is satisfactory for maintenance. Stock viability is better on Tween 80 than on fatty acids suspended in detergent Tergitol Nonidet P-40 (944). For biosynthetic pathway see reference 944.

ufa-2: unsaturated fatty acids-2

IV or V. Linked to *inl* in a cross with *alcoy* (944). Data do not distinguish linkage group because *T(IVR;VR)R2355* was present.

Requirement similar to that of *ufa-1* mutants. Linkage similar to that of *ufa-1*. No intercross data. Designation as second *ufa* locus is based solely on complementation with *ufa-1* (944). Highly revertible, and stocks are readily lost on suboptimal medium (S. Brody, personal communication).

_un_: unknown

Unknown function. Temperature-sensitive (heat-sensitive) conditional mutants, irreparable by supplementation at the restrictive temperature (usually 34 to 37°C). Originally referred to as "unknown requirement" on the initial hypothesis that such mutants would prove to be auxotrophs. Several heat-sensitive genes have been sufficiently characterized to be assigned more specific names than *un* (e.g., *ndc, rip, psi, eth-1*, and *fs-2*). At least some *un* mutants are deficient in amino acid transport (543, 1075). For most *un* loci, however, little is known of the molecular or cellular basis. Because of their map locations, several are useful as genetic markers. Scoring may require growth comparisons at two temperatures, preferably with small conidial inocula. Some heat-sensitive mutants with altered morphology at restrictive temperature are called *cot* or *scot*.

Temperature-sensitive auxotrophs with certain requirements have sometimes been classed initially as *un* because complex complete medium is either inadequate or inhibitory. Thus, complex-medium-irreparable temperature-sensitive mutants no. 3, 13, and 14 of reference 507 proved to be temperature-sensitive *thr* auxotrophs; no. 6, 20, and 30 were *his*; and no. 19, 24, and 35 were *asn* (T. Ishikawa, personal communication). Many *un* mutants do not achieve normal growth rates even at the permissive temperature (usually 25°C). Genes *un-1* to *un-8* were assigned locus numbers in reference 813. "*T*" in allele numbers of several *un* mutants designates Tokyo, not translocation.

_un-1_: unknown-1

IR. Between *nic-2* and the *T(4540)* right breakpoint. Linked to *cys-9* (0/72), *cr-1* (5 to 9%), and *bs* (9%) (816, 818, PB). (*482*).

Unknown function. Heat sensitive. Growth at 25°C but not at 34°C (484). Formerly called *un(44409)*.

***un-2*: unknown-2**

IR. Between the *T(AR173)* left breakpoint and *T(AR190)*; hence, right of the centromere and *arg-3* (1 to 2%) and left of *his-2* (1%). Included in duplications from *T(AR173)* but not from *T(AR190)* (808). (*482*).

Unknown function. Heat sensitive (482). Growth at 25°C but not at 39°C. Scorable but leaky at 34°C. Formerly called *un(46006)*.

***un-3*: unknown-3**

IL. Right of *In(NM176)*; hence, right of *ser-3* (1%) (1093). Left of *mt* (0.04 to 0.1%) (488, 758). Closest bracketing marker left of *mt* (482).

Unknown function. Heat sensitive (484). Growth drops off sharply between 28.5°C and 30°C (R. L. Metzenberg, personal communication). Multiply transport deficient at permissive temperatures with increased fragility of protoplasts (543); reduced rate of uptake of citrulline (1075) and aspartate (543, 1149). Resistant to ethionine and to *p*-fluorophenylalanine at 25°C (542, 543). Used to tag mating type (487) and as a flanker in an attempt to resolve the mating type region by recombination (758). Strains with probable *un-3* alleles are selected as citrulline-resistant mutants of *pyr-3 arg-12*^{s}; most mutants selected in this way show complementation between alleles (1075). A possible functional relation to mating type is discussed in reference 543. Growth at 25°C aided by 0.3 mg of sodium acetate per ml. May be scored by slow growth at 25°C if acetate is not added to minimal medium (487). Formerly called *un(55701)*.

***un-4*: unknown-4**

VIL. Right of *lys-5* (2%). Left of *T(T39M777)* and *cys-2* (4%) (808, 1012). (*482*).

Unknown function. Heat sensitive, 34°C versus 25°C (481). Formerly called *un(66204)*.

***un-5*: unknown-5**

IL. Right of *fr* (6%). Left of *T(39311)* and *nit-2* (2%) (816, 798). (*574*).

Unknown function. Heat sensitive, 34°C versus 23°C. Inhibited by histidine and tryptophan; osmophilic (26 to 28°C) (574, 575). Formerly called *un(b39)*.

***un-6*: unknown-6**

IIIR. Right of *sc* (21%) and *acr-2* (6 to 20%). (*816*, PB).

Unknown function. Heat sensitive, 34°C versus 25°C (484). Formerly called *un(83106)*.

***un-7*: unknown-7**

IR. Left of *al-1* (3%) (818, *813*). Between *T(STL76)* and *T(4637)*; hence, right of *cyh-1* (808).

Unknown function. Heat sensitive, 34°C versus 25°C (506). Dies slowly at restrictive temperature (508). Allele *T*35M50 is called 31 or TS31 in references 507 and 508.

***un-8*: unknown-8**

IVR. Right of *T(ALS159)*; hence, right of *psi-1*. Left of *col-4* (5%) and *T(S1229)*. Linked to *pyr-1* (0/47). (*813*, PB).

Unknown function. Heat sensitive (507). No growth at 34°C. Morphology abnormal at 25°C, unlike that of strains carrying closely linked *psi*. Allele *T*27M9 is called 1 in reference 507.

***un-9*: unknown-9**

VR. Between *pyr-6* (3%) and *his-6* (5 to 9%) (*818*).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*54M96 is called 42 in reference 507.

***un-10*: unknown-10**

VII. Right of *wc-1* (7%). Left of *frq* (9%) and *for* (12%) (*818*; J. F. Feldman, personal communication).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*42M45 is called 11 in reference 507.

***un-11*: unknown-11**

VR. Linked to *al-3* (0/48) (*818*).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*42M30 is called 10 in reference 507.

***un-12*: unknown-12**

IVR. Linked to *col-4* (0/73) and *pdx-1* (5%), left of *T(S1229)* (*818*, PB).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*51M118 is called 17 in reference 507.

***un-13*: unknown-13**

VI. Linked to *ylo-1* (2%), *un-4* (3%), and *lys-5* (4%) (*818*).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*42M24 is called 9 in reference 507.

***un-14*: unknown-14**

IIIR. Right of *acr-2* and *thi-4* (8 to 20%). Left of *leu-1* (5%) (*818*, PB).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*54M55 is called 36 in reference 507.

***un-15*: unknown-15**

IIR. Linked to *rip-1* (1%). Right of *trp-3* (10%). (*813*, PB).

Unknown function. Heat sensitive, 34°C versus 25°C (T. Ishikawa, personal communication). Grows poorly at the permissive temperature; *rip-1* is, therefore, preferred to *un-15* as a marker for the right end of II (811). One allele, *T*54M50.

***un-16*: unknown-16**

IL. Right of *mt* (<1%). Left of *ta* (1%) and *acr-3* (<3%). Closest flanking marker right of the mating type locus (*818*, PB).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*42M69 is called 16 in reference 507.

***un-17*: unknown-17**

IIIR. Between *tyr-1* (4%) and *dow* (23 to 28%) (*818*).

Heat sensitive, 34°C versus 25°C (507). Ascospores containing *un-17* are white or slow to mature (D. D. Perkins, unpublished data). Shows rapid, exponential death at 35°C, which is averted by cycloheximide or conditions allowing no protein synthesis. Altered phospholipid synthesis (508). Also cold sensitive and osmotic remediable at 11°C (701). Allele *T*51M171 is called 25 or TS25 in references 507 and 508.

***un-18*: unknown-18**

IR. Right of *T(NM169d)* and *R* (11%) (*808*).

Unknown function. Heat sensitive (507). No growth at 34°C. Growth at 25°C is substantial but not wild type, and better on complete medium than on minimal medium. Allele *T*54M94 is called 41 in reference 507.

***un-19*: unknown-19**

VR. Linked to *al-3* (9%), *un-9* (22%), and *un-11* (14%) (*D. D. Perkins, unpublished data*).

Unknown function. Heat sensitive, 34°C versus 25°C (D. D. Perkins, unpublished data).

***un-20*: unknown-20**

IIR. Between *ff-1* (4%) and *ace-1* (15%). Right of *aro-1* (5 to 9%). (*816*, 1052, PB).

Unknown function. Heat sensitive, 39°C versus 25°C. Best scored at 39°C on minimal medium. Leaky. Some flat, aconidiate unpigmented growth occurs even at restrictive temperatures (PB). Called *mo(P2402t)* (816, 1052).

***un-21*: unknown-21**

IIIR. Between *acr-2* and *un-6* (*PB*).

Unknown function. Heat sensitive, 34°C versus 25°C (507). Allele *T*53M26 is called 29 in reference 507.

***un-22*: unknown-22**

VII. Linked to *met-7* (1%) and *un-10* (>20%) (*1019*, PB).

Unknown function. Heat sensitive, growing at 20 to 28°C but not at 37°C. Called *un(61C)* and *un(62C)*. (1019).

***un-23*: unknown-23**

VIR. Right of *trp-2* (5 to 27%). Left of *ws-1* (*1019*; D. D. Perkins, unpublished data).

Unknown function. Heat sensitive, growing at 20°C but not at 28°C. Called *un(64D)*. (1019).

un(STL6)

See *fls*.

un(b39)

See *un-5*.

un(44409)

See *un-1*.

un(46006)

See *un-2*.

un(55701)

See *un-3*.

un(66204)

See *un-4*.

un(83106)

See *un-6*.

***upr-1*: UV photoreactivation-1**

IL. Between *mt* (2%) and *arg-1* (7%) (*1094*).

Sensitive to UV (273, 1096), nitrous acid (1094), ionizing radiation (940), and nitrosoguanidine, 4-nitroquinoline 1-oxide, and ICR-170 (509). Insensitive or marginally sensitive to methyl methane sulfonate (536). Unable to excise dimers (1164). Normal spontaneous mutation (275). High UV-induced mutation (273). For mutation induction by other agents, see references 509 and 940. Defective photoreactivation in vivo, but photoreactivation enzyme functions in vitro (1094). No homozygous effect on meiosis or crossing over (1094). Recessive in heterokaryons (979). Double mutant *upr-1; uvs-3* is more sensitive than either single mutant (1095). Double mutant *upr-1; uvs-2* is no more sensitive than the *uvs-2* single mutant (506).

***ure*: urease**

Strains carrying mutations at four distinct loci, *ure-1, -2, -3, -4*, lack all detectable urease activity (Fig. 10 and 24). Urease from other organisms comprises numerous distinct subunits; possibly all four *Neurospora* loci are structural genes (78). *ure* mutation D2, which is tightly linked to *ure-1*, fails to complement mutations at any of the four loci, suggesting a regulatory role (452). Strains carrying mutations of another type possess partial activity but are readily scorable by poor growth on urea as the nitrogen source. These probably represent additional loci in V (A7, S3), IV (E3, E7), or elsewhere (C5, K3, R2) (452; H. B. Howe, Jr., personal communication). They are not given separate entries here. *ure* mutants have been isolated by methods based on the inability to generate ammonia from urea, using pH indicators (78, 452, 569). The following methods for scoring isolates are probably generally applicable, but all have not been tested on mutations at all loci. Method 1: Little growth on filter-sterilized urea as the sole nitrogen source (452) (not good if amino acids must be added). Method 2: Five- to twenty-minute scoring test, touching bits of filter paper dipped in urea-bromthymol blue buffer to conidia or aerial growth (570). Method 3: Color change when grown on slants of synthetic crossing medium (1134) containing phenol red (90 μg/ml) and urea (3 mg/ml) as the sole nitrogen source, added before autoclaving; scored after 4 to 5 days at 34°C (PB; modified from the method of J. A. Kinsey).

***ure-1*: urease-1**

VR. Right of *ace-5* (<1%), *am* (1%), and *ure-2* (3%). Left of *his-1* (<1%) (*570*, 578).

No urease activity (452, 569) (Fig. 10 and 24). Some revertants show altered heat stability of urease, suggesting a structural role (78). Used to study the metabolic fate of arginine by measuring urea accumulation (592). Used for arginine tracer experiments (217); flux (355). Used to determine the relative contributions of arginine and purines in urea formation (235). For the role of urease in purine catabolism, see Fig. 24. For scoring methods, see *ure*. Possible allele D2 fails to complement *ure-1, -2, -3*, or *-4* (452). Called *ure(9)* (570).

***ure-2*: urease-2**

VR. Left of *am* (2%). Probably right of *sp*. Linked to *ure-1* (3%) (*570*).

No urease activity (452, 569) (Fig. 10 and 24). Reversion and complementation data (78). Enzyme from interallelic complementation and from some revertants shows altered heat stability, suggesting a structural role (78). Shows hyperinducibility of purine catabolic enzymes uricase, allantoicase, and allantoinase (872). For scoring methods, see *ure*. Called *ure(47)* (570).

***ure-3*: urease-3**

IIR. Between *arg-12* (7 to 12%) and *ace-1* (14%) (*PB*). Allele B1 is closely linked to translocation *T(IR;II)B1* in the original strain. Consequently, *ure-3* was at first assigned incorrectly to IR (78, 452). Point-mutant allele F29 shows linkage in IIR, not in IR (PB).

No urease activity (452) (Fig. 10 and 24). Some revertants show altered urease thermostability, suggesting a structural role (78). For scoring methods, see *ure*.

***ure-4*: urease-4**

IR. Left of *ad-3B* (3%). Probably right of *his-3* (1%) (78). (*452*).

No urease activity (452) (Fig. 10 and 24). Some revertants show altered heat stability of urease, suggesting a structural role (78). For scoring methods, see *ure*.

***ure(D2)*: urease**

VR. At or near *ure-1* (0/45) (*452*).

No urease activity. Fails to complement representatives of all four *ure* loci; possible regulatory gene (452).

***uvs*: UV sensitive**

uvs and other radiation-sensitive mutants are highly pleiotropic, with phenotypic spectra that may (or may not) include: sensitivity to ionizing radiation, radiomimetic chemicals, mitomycin C, or histidine; impairment of meiosis; increased frequency of deletion or mitotic recombination; increased spontaneous or induced mutation; defective DNA repair; altered secretion of deoxyribonuclease. Sensitivity is typically recessive. The most sensitive *Neurospora* mutant, *uvs-2*, is only 20 times more sensitive than the wild type to UV (935).

The more general name, *mus* (mutagen sensitive), has been adopted for mutant loci beyond *uvs-6* (537). Several UV-sensitive mutants have names other than *uvs* or *mus*: see *gs(6), Mei-2, mei-3, nuh-4, upr-1*.

Scoring is most readily accomplished by spot tests, i.e., spotting conidial suspensions on the surface of prepoured plates that contain sorbose (536, 932, 1023) and comparing growth on exposed and control plates. Properties of the *uvs* mutants are summarized in references 509, 537, 936, and 938. For properties of double mutants, see references 506, 539, and 1095.

***uvs-1*: UV sensitive-1**

Not mapped.

Slightly increased sensitivity to UV (187, 273). Spontaneous and UV-induced mutation probably normal (273, 275). Homozygous fertile and without effect on crossing over. Ascospore viability and early growth severely impaired in homozygous crosses (187). Reduced rate of dimer excision (1164). Difficult to score; spot tests best read early (24 h).

***uvs-2*: UV sensitive-2**

IVR. Right of *cys-4* (5%) (*1023*). Left of *pmb* (8%) (S. Ogilvie-Villa, cited in reference 248; R. Sadler and S. Ogilvie-Villa, personal communication) and the *T(S4342)* right breakpoint. Linked to *T(AR209)*, *T(T54M50)*, and *T(ALS179)* (2 to 6%), which mark the IVR tip (808).

Sensitive to UV (273, 1023), ionizing radiation (537, 935, 940), methyl methane sulfonate (536, 537), nitrosoguanidine (509, 935), mitomycin C (537), 4-nitroquinoline 1-oxide (509), nitrous acid (D. R. Stadler and E. Crane, personal communication), and ICR-170 (509). Slight or no sensitivity to histidine (537, 759). No dimer excision (1164). Normal spontaneous mutation (275). High UV-induced mutation rate (273); for mutation induction by other agents, see references 509 and 940. Homozygous fertile; no effect on meiosis or crossing over. Recessive in heterokaryons (1023). *uvs-2* is the most UV sensitive of *Neurospora* mutants (15 to 20 times wild type) (537, 938). Used to show that DNA repair is induced by a small dose of UV (1022). Used to demonstrate postreplication repair (130). Only known allele was discovered in several Seattle stocks of mixed ancestry, and thus may be present in lab stocks elsewhere (1023; D. R. Stadler, personal communication). Not to be confused with a cytoplasmically determined UV-sensitive mutation called *uvs-2* in reference 187, but now called [*uvs(cyt)*].

***uvs-3*: UV sensitive-3**

VL. Linked to *cys-10* (3 to 7%), probably to the left (538, *932*).

Allele ALS11 is sensitive to UV (273, 932, 933), ionizing radiation (537, 933, 940), methyl methane sulfonate (536), nitrosoguanidine (509,

933), mitomycin C (195, 537), histidine (932), and 4-nitroquinoline 1-oxide and ICR-170 (509). Reverts spontaneously (932). No UV-induced mutation (273). For mutation induction by other agents, see references 509 and 940. Increased spontaneous mutation (275, 537). Dimer excision delayed and at reduced rate (1164). Defective photoreactivation in vivo, but photoreactivation enzyme functions in vitro (934). Defective in extracellular nuclease, giving reduced halo around colonies on DNA agar (538). Apparently deficient in proteolytic conversion of nuclease precursor to active intra- and extracellular deoxyribonucleases, but this effect could be indirect (360). Increased stability of CPS(Pyr) and ACT activities in vitro also suggests that protease activity may be reduced in *uvs-3* mutants (882). Causes increased duplication instability (mitotic recombination or deletion or both) (932). Conidial viability is low (275, 932). Double mutant *upr-1; uvs-3* is much more sensitive to UV than is either single mutant (1095). Double mutant *uvs-3, uvs-6* is inviable (506). Homozygous barren, with block before karyogamy (860, 932). Shows high level of repair of genetic damage without induction in rescuing a heterokaryotic component that carries potentially lethal mutagen-induced damage (1022). Probable allele FK016, isolated as halo mutation *nuh-4*, resembles *uvs-3* allele ALS11 but is less extreme in some properties, e.g., it shows better conidial survival (538; see reference 537).

***uvs-4*: UV sensitive-4**

IIIR. Left of *ad-4* (4%) (*932*).

Sensitive to UV (273, 932, 933) and histidine (932). Moderately sensitive to methyl methane sulfonate (537). Insensitive or only slightly sensitive to nitrosoguanidine (537, 933); no increased sensitivity to ionizing radiation (940) or mitomycin C (195, 537). Probably normal spontaneous mutation (275). Reduced UV-induced mutation (273). For mutation induction by other agents, see reference 940. Homozygous fertile; no effect on intragenic or intergenic recombination. Ascospore viability and early growth severely impaired in homozygous crosses (932). Recessive in heterokaryons (979).

***uvs-5*: UV sensitive-5**

IIIR. Linked to *vel* (1%) (*932*).

Sensitive to UV (273, 932, 933), nitrosoguanidine and ICR-170 (509), and histidine (932); not sensitive to ionizing radiation (940) or 4-nitroquinoline 1-oxide (509). Slow growth. Normal dimer excision (1164). Spontaneous mutation normal (275); UV-induced mutation reduced (273). For mutation induction by other agents, see references 509 and 940. Homozygous barren (932) with meiosis blocked at pachytene (860).

***uvs-6*: UV sensitive-6**

IR. Between *thi-1* (3 to 8%) and *ad-9* (4%) (538; E. Käfer, personal communication; D. Newmeyer, unpublished data). Very close to *met-6* (<1%) (D. Newmeyer, unpublished data). (*937*).

Increases sensitivity to UV (273, 937), ionizing radiation (537, 937, 940), nitrosoguanidine (509, 537), histidine (755), methyl methane sulfonate (536), ICR-170 and 4-nitroquinoline 1-oxide (509), and possibly mitomycin C (537). Normal UV-induced mutation (273). For mutation induction by other agents, see references 509 and 940. Increased spontaneous mutation not evident in the *ad-3* system (275), but is found for recessive lethals in a heterokaryon test system (E. Käfer, personal communication). Normal dimer excision (1164). Defective in extracellular nuclease, giving reduced halos around colonies on DNA agar (538). Increased duplication instability due to mitotic recombination, deletion, or both (759). Homozygous barren (759), with a block at crozier differentiation (860). Reduced conidial viability (537). Switches to stop-start growth after initial normal growth (D. Newmeyer, unpublished data). Not completely recessive in heterokaryotic conidia (979). Increased stability of CPS (Pyr) activity in vitro suggests that protease activity may be reduced in the mutant *uvs-6* (882); see also reference 360. Double mutant *uvs-3; uvs-6* is inviable (506).

uvs(FK104)

See *mus-9*.

***val*: valine**

VR. Right of *at* (10%). Linked to *ilv-2* (0/135) (*PB*).

Strains carrying alleles 33026 and 33050 appear to be valine auxotrophs, not requiring or responding to added isoleucine (482, PB). Not defective in valyl-tRNA synthetase (J. Evans, personal communication via J. A. Kinsey), as are the only known *val* mutants of bacteria. Requirement somewhat leaky; not temperature sensitive. Relation to *ilv-1* and *ilv-2* is uncertain. An *ilv* mutant strain with an incomplete isolation

number, *ilv* (?6201), was incorrectly designated *val* (45201) and was the source of linkage data for a locus erroneously shown as *val* on maps made before 1980.

***van*: vanadate resistant**

VIIL. Left of *nic-3* (4%) (*B. J. Bowman, personal communication*).

Resistant to vanadate (0.1 to 1.0 mM) in Vogel minimal medium with 0.15 mM phosphate and 20 mM HEPES (*N*-2-hydroxyethylpiperazine-*N'*-2-ethanesulfonic acid) buffer, adjusted to pH 7.5 (B. J. Bowman, personal communication).

***var-1*: variant-1**

Unmapped. Single-gene difference.

Slow growth (62% normal). Aerial hyphae decreased to give shaven appearance. Functional protoperithecia absent. Lysed areas appear and spread in old cultures. Not rejuvenated in heterokaryons. Originated in experiment on uninterrupted growth. (86).

***vel*: velvet**

IIIR. Linked to *phe-2* (1%). Right of *T(D305)* and *ro-2* (6%). Left of *tyr-1* (3 to 5%) (*814*, 816, PB).

Soft, colonial growth habit (789). Flat at first; then becomes elevated. Eventually conidia may be formed in aerial puffs. Good female fertility. *col-13* (R2471) and *col-15* (R2531) are putative alleles.

***vis(3717)*: visible**

Linked to the right of the centromere in linkage group I (*482*, 486), but inseparable from translocation *T(I;III)3717* (808). No corresponding point mutation is known.

Aconidial flat morphology.

***wa*: washed**

VR. Linked to *inl* (6%) (*812*).

Thin, spreading surface growth and conidiation (812). Called *spco-2*.

***wc-1*: white collar-1**

VIIR. Right of *met-9* (1 to 4%). Left of *un-10* (7%) and *for* (6%) (724, *812*, 816).

Carotenoids absent from mycelia; conidia become pigmented with some delay. Named because nonconidiating rim at top of agar slant remains white. A double mutant with *fl* or other nonconidiating mutant would be classed as albino. Regulatory mutants for photoinduced carotenogenesis via blue light receptor might be expected to have a similar phenotype (444, 445). A blue light treatment (given in vivo), which increases the activity of soluble and microsomal enzymes required for phytoene biosynthesis in the wild type, does not do so in the mutant *wc-1* (445). Fails to show phototropism of perithecial beaks when used as the female (protoperithecial) parent, but not when used as the male (fertilizing) parent (R. W. Harding, personal communication). Useful genetic marker (725, 800). Scoring clearest at high temperatures (34°C).

***wc-2*: white collar-2**

IR. Right of *T(NM103)*, *T(ALS182)*, and *thi-1* (8%). Left of *ad-9* (7 to 20%) (*PB*).

Resembles *wc-1*. Detected in a *glp-1* strain (J. B. Courtright, personal communication); *wc-2* is separable from *glp-1* by crossing over (PB).

***ws-1*: white spore-1**

VIR. Right of *trp-2* (38%) (*822*).

Delayed ascospore maturation; autonomous. Ascospores fail to darken or do so slowly. Black spots appear on some *ws-1* ascospores. In aged crosses, a few percent of the *ws-1* ascospores darken and are capable of germination. Fertile and prototrophic, with normal tyrosinase activity vegetatively. Photograph of asci. (822). Second-division segregation frequencies may be as high as 80 to 96% (586, 822), providing evidence for chiasma interference.

***ws-2*: white spore-2**

VI. Linked to *ylo-1* (16%), *trp-2* (2%) (A. Kruszewska, personal communication), and the centromere (9 to 24% in ordered asci) (586). Recombines with *ws-1* (R. L. Phillips, personal communication). (*816*).

Ascospores white initially, browning with age. Autonomous (816).

***xdh-1*: xanthine dehydrogenase-1**

II. Linked to *pe* (14%) and *alc-1* (24%) (*872*).

Defective in purine catabolism. Unable to use hypoxanthine as the sole nitrogen source. Lacks xanthine dehydrogenase (xanthine oxidase) (872) (Fig. 24). *nit-1*, *-7*, *-8*, and *-9* mutants are also deficient in xanthine dehydrogenase activity because of a defect in the molybdenum-containing cofactor that is common to xanthine dehydrogenase and nitrate reductase (741, 1080, 1081).

ylo: yellow

Yellow carotenoids. In addition to strains with mutations at loci designated *ylo*, strains with some *al-1* alleles (especially ALS4 and RES-25) produce lemon-yellow carotenoids, although in reduced quantity (1039, 1042, 1071). *age-3* mutants are also yellow. Many morphological mutants appear to be abnormal in carotenoid metabolism and may appear yellowish or pale rather than the wild-type orange.

ylo-1: yellow-1

VIL. Between *cys-1* (8%) and *ad-1* (6%). Probably right of *Bml* (2%) (1012, *PB*). (*381*).

Yellow carotenoids (381). Affects synthesis of neurosporaxanthin (4'-apo-β'-caroten-4'-oic-acid); citations in reference 398. Lesion probably involves the conversion of lycopene to 3,4-dehydrolycopene or the conversion of either torulene or γ-carotene to neurosporaxanthin (398 and references therein) (Fig. 9). Resembles the orange wild type in young cultures, but color differences become clear with age. Expressed in both conidia and mycelia. Undefined modifiers affect intensity. Fails to complement with many of the *al-1* and *al-2* albino strains (R. E. Subden, personal communication).

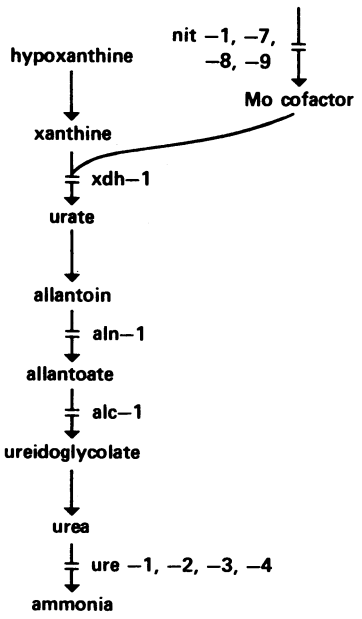


FIG. 24. Purine catabolic pathway, showing the sites of action of the *xdh*, *aln*, *alc*, and *ure* genes (452, 569, 872). The molybdenum cofactor is essential to both xanthine dehydrogenase and nitrate reductase (1081).

ylo-2: yellow-2

IL. Right of *In(H4250)*; hence, of *suc*. Left of *arg-1* (1%) (*816*).

Rather sparse yellow conidia (816). No information on carotenoids; possibly the yellowish color is a secondary effect of abnormal development. Inferior to *ylo-1* as a marker. First shown distinct from *ylo-1* by A. M. Kapular (via FGSC).

ylo-3: yellow-3

IIR. Probably allelic with *fl* (0/56) (D. D. Perkins, unpublished data). Listed as IIIR; no data given (1040). Not linked to *ylo-1* or *ylo-2* (A. M. Kapular, via FGSC).

Pale yellow. Slow to conidiate. (A. M. Kapular [via FGSC], D. D. Perkins, unpublished data).

SOURCES OF STOCKS

The preferred source is the Fungal Genetics Stock Center (FGSC), Humboldt State University, Arcata, CA 95521. FGSC specializes in *Neurospora* stocks for research. Nearly 4,000 *Neurospora* stocks are maintained, including key alleles at most of the known loci. These are available free of charge to individuals and nonprofit institutions. Stock lists are published biennially in the *Neurospora Newsletter* under the following headings: single mutants; multiple mutants; nonchromosomal mutants; wild type and wild-collected strains (representing all known species); chromosome rearrangements; reference strains, testers, and stocks for special purposes. The last category includes linkage testers, standard *N. crassa* wild types and mating type testers, species diagnostic testers and reference strains, heterokaryon incompatibility testers, strains for mutant enrichment and replication, strains that produce only microconidia, strains for obtaining protoplasts or spheroplasts, Spore-killer testers, strains for mutagenesis, nonsense suppressors, and transport mutants.

The American Type Culture Collection maintains about 200 *Neurospora* stocks.

SUMMARY

Information on all the known gene loci of *N. crassa* is gathered in a compendium. Over 500 loci have been mapped to the seven linkage groups. Linkage maps have been revised, resolving many ambiguities of gene order. A separate entry for each locus gives documented information on linkage, phenotype, biochemistry, scoring, interactions with other loci, regulation, fertility, stability, complementation, fine structure, and uses in research. Entries are also given for the nucleolus organizer, for individual centromeres and chromosome tips, and for chromosome rearrangements that have been used in mapping. Diagrams of biosynthetic and catabolic pathways show sites of gene action. Brief summaries are given of the history of *Neurospora* research and of current genetic nomenclature and stock sources.

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