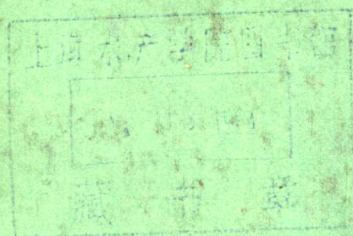




GENETICS TODAY

Volume 1



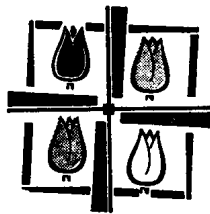
GENETICS TODAY

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Edited by
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in collaboration with

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PREFACE

This volume contains the abstracts of contributed papers, demonstrations and films presented at the XIth International Congress of Genetics. The editorial policy has been to assemble the abstracts into sections not corresponding completely with the titles of the symposia (see vol. II and III). Within each section we have endeavoured to arrange the abstracts in a practical manner which may, however, vary from section to section. Those abstracts delayed in some way are printed at the end of their respective sections. The abstracts are numbered within each section; D refers to demonstrations and F to films. In the alphabetical index of authors at the end of this volume references are given to these numbers. Full addresses of the authors may be found by consulting the membership list supplied at the Congress and printed at the back of volume III.

S. J. GEERTS

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SECTION I

COMPLEX LOCI

1.1. Genetic Fine Structure of the rosy Cistron in *Drosophila melanogaster*. A. CHOVNICK, A. SCHALET, and R. P. KERNAGHAN (Storrs, U.S.A.).

A highly efficient crossover selector system⁽¹⁾ has been used to examine the fine structure of a single cistron in *Drosophila*. This system permits routine large-scale sampling approaching that used in microbial studies, and permits distinction between rare crossovers and spontaneous mutations. Such studies, involving a large number of mutants of the rosy cistron, permit the following general conclusions: (a) The mechanism of recombination in higher organisms, like that of microorganisms, does permit crossing over within a cistron. (b) The resolving power of such analysis of genetic fine structure in *Drosophila* approaches that of microbial systems.

1. CHOVNICK *et al.*, *Amer. Nat.* **96**, 281, 1962.

1.2. On the Phenotypic Manifestation of Mutations in the rosy Region of *Drosophila melanogaster*. A. SCHALET, R. P. KERNAGHAN, and A. CHOVNICK (Storrs, U.S.A.).

According to the suggestion of Welshons (1962) discontinuous mapping of complex loci in *Drosophila* may be a function of the type of mutant (recessive visibles) usually used. His evidence shows that *Drosophila* results may be reconciled with continuous mapping of mutant sites in microorganisms when methods are used to detect mutable sites for recessive lethals, recessive visibles with atypical phenotypes and possibly wild-type iso-alleles. While Welshons' proposal may apply to many of the complex loci in *Drosophila*, evidence from analysis of the rosy region reveals that within a single functional unit defined on the basis of a visible and enzymatic phenotype, recessive visibles give continuous recombination mapping of mutable sites upon appropriate sampling. The following considerations suggest that the range

of phenotypes exhibited by mutations in the rosy region does not include complementary recessive visibles and lethals or non-complementary lethals: 1. Lack of complementation among all mutants at separable sites and failure to detect mutants complementing rosy² among 24 X-ray-induced rosy mutants selected over a deficiency for the entire rosy region. 2. Absence of rosy mutants which are lethal and unassociated with genetically detectable alterations involving closely neighboring loci among a sample of 13 rosy mutants with lethal effects localized to the vicinity of the rosy region. 3. Absence of complementary recessive mutations within the rosy cistron among a sample of 11 lethals and visibles selected within a small (0.5 map units) chromosomal segment which included the rosy region.

1.3. Notch Pseudoalleles in *Drosophila melanogaster*. W. J. WELSHONS, E. S. VON HALLE, and B. J. SCANDLYN (Oak Ridge, U.S.A.).

The Notch locus is a complex one composed of pseudoallelic recessive visibles superimposed upon an array of pseudoallelic recessive lethal Notch (N) mutants. The linear order from left to right on the X chromosome of all pseudoalleles is *fa* - *fa*^{no} - *N*⁴⁰ - *N*^{Nic} - *spl* - *N*¹⁰³ - *N*^{j24} - *N*^{Co} - *N*^{el1} - *nd*. The order of *N*^{el1} and *nd* is tentative but both are to the right of *N*^{Co}. The genetic length of the region has been estimated to be 0.11 units.

It was noted previously that no *N*'s had been discovered in the region to the left of *fa*^{no}, but it was presumed that they would eventually be found there. Recent experiments performed with *N*^{el1}, *spl*, and *fa* yielded results which placed *N*^{el1} 0.03 units to the left of *fa*, and increased the genetic length of this complex locus to 0.14 units.

Similar experiments utilizing *N*³⁹, *fa* and *spl* localized *N*³⁹ around *fa*^{no}. Since *N*³⁹ was known to be cytologically deficient for salivary band 3C7, 85,600 chromosomes were tested from *N*³⁹/*fa*^{no} heterozygotes, but no recombinants were found. It is probable that deficiency *N*³⁹ includes the locus of *fa*^{no}.

Obviously the region left of fa^{70} is capable of producing recessive lethals as indicated by the localization of N^{ell} . Furthermore, the N^{39} experiments indicate that the single salivary band C37 cannot contain all the pseudoallelic loci comprising the functional Notch locus since mutants like N^{ell} and N^{Co} are localized at appreciable distances to the left and right of the deficiency.

as found by Green at the white locus, in which event the resulting duplication in the present case might be spectacle and the complementary deficiency a lethal.

This investigation was supported by U.S. Public Health Service Research Grant GM-8889-02 from the Division of General Medical Science.

1.4. An Apparent Non-equivalence in Crossover Rates between Lozenge Alleles in Trans and Cis Arrangement in *Drosophila*. LUOLIN S. BROWNING and EDGAR ALTENBURG (Houston, U.S.A.).

The rate of recovered crossovers between lz^{BS} and lz^{46} in *Drosophila* was found to be significantly greater from females heterozygous for these two lozenge alleles in *trans* arrangement as compared with *cis* as follows. Among 350,000 F_1 from the *trans* arrangement ($lz^{BS} + / + lz^{46}$), 116 crossovers (46 $lz^{BS} lz^{46}$ and 70 $+ +$) were recovered, or about 1 crossover in 3000 offspring; among 436,500 F_1 from the *cis* arrangement ($lz^{BS} lz^{46} / + +$) 75 crossovers were recovered (75 being the combined number of $lz^{BS} +$ and $+ lz^{46}$, both of lozenge phenotype as contrasted to the spectacle phenotype of the double mutant non-crossover class on the one hand and the complementary normal on the other), or about 1 crossover in 5820 offspring. It is not considered very likely that this difference in rates could be due entirely to the relative viabilities of the crossover classes as compared with the non-crossovers in the *trans* and *cis* arrangements. Work earlier reported indicated that the crossover rate between apricot (w^a) and white (w) was also greater for the *trans* than the *cis* arrangement. In explanation of these results, it is suggested that alleles might somehow differ structurally. One series of lz -locus experiments (involving a question as to the identity of the alleles) gave an unexpected result in that the two lz alleles in *trans* arrangement gave the apparent double mutant class (10 phenotypic spectacles in 70,000 offspring) but none of the complementary normal phenotype. Judd reported a result which we interpret as similar to this, insofar as in the F_1 from apricot and buff in *trans* arrangement, the only "exceptions" found were of white phenotype (no normals). We have no explanation for this kind of result except perhaps that it is due to some type of non-homologous pairing such

1.5. A Study of Recessive Lethals on the Dot-like Fourth Chromosome in *Drosophila melanogaster*. BENJAMIN HOCHMAN (Knoxville, U.S.A.).

A three-year study of 1352 fourth chromosomes, extracted from natural populations of *D. melanogaster*, has uncovered 15 which are lethal in the homozygous state. Allelism tests of 14 of the lethals (one was lost) demonstrate that they occupy eleven different loci. One locus is represented three times; a second locus twice; and nine have a single representative each. One of these nine is allelic to a lethal which has arisen spontaneously in two different laboratory stocks.

Crosses of the lethals to the six existing chromosome 4 dominant visibles have thus far localized one of them to the bt^D locus and a second to that section of the microchromosome delimited by the Minute-4 deficiency. None of the lethals permits pseudodominance of any of the seven non-allelic recessive visibles tested.

Several X-ray-induced lethal fourth chromosomes, generously provided by Drs. M. M. Green and H. Gloor, have been examined for interactions with the lethals of spontaneous origin. Three heterozygous combinations which are lethal have been observed to date.

If all of the spontaneous microchromosomal lethals are "point" mutations, the number of loci identified on chromosome 4 has been approximately doubled over the figure based heretofore solely on the known visible mutations. The total number of "potentially-lethal" loci to be expected on the microchromosome, as well as the developmental period during which each lethal acts, will be discussed.

This research was supported by Grant RG 9845, United States Public Health Service, National Institutes of Health.

1.6. A "Mutable" Gene in *Drosophila melanogaster*. WILLIAM M. HEXTER (Amherst, U.S.A.).

Attached-X females properly marked and heterozygous for a specific allele of garnet (g^{53d}) and any other garnet allele (g^x) were singly mated by appropriately marked males, and the female progeny were screened for the presence of wild type (non-garnet-eyed) females. In a total of 282,000 females, 15 were wild type. In a similar test differing only in the homozygosity of g^{53d} , 0 wild type females were recovered in 539,000 females. Of the 15 wild type females, 6 were non-diagnostic as to generating mechanism; of the remaining 9, 6 could be interpreted as due to single crossover between pseudoallelic loci. The remaining 3 are not so easily explained. If the pseudoallelic hypothesis were correct it should be possible to demonstrate a double mutant. Four of the presumptive 6 females were tested in a manner to reveal one of the two garnet alleles should the double mutant exist. The sum of these 4 experiments was 428,000 flies with 0 single garnet mutants recovered. An additional and independent test of a presumptive double mutant was to place the supposed double mutant chromosome ($g^{53d} g^x$) in apposition to an appropriately marked g^{53d} free-X chromosome. Due to homozygosity of g^{53d} such a test would not be expected to yield any wild type progeny. In fact, however, 22 wild types were recovered in 685,000 flies; of these 22, 11 were associated with recombination and could be simply interpreted as a crossover between pseudoallelic loci. The remaining 11 were non-recombinants for outside markers and are not as easily explained. The conclusion based on these results is that some, if not all, of the wild types were due to mutability of g^{53d} when heterozygous with other garnet alleles. Further genetic tests have not suggested, nor ruled out, the possibility that g^{53d} is a duplication.

Detailed data are in press in *Proc. Nat.-Acad. Sci. Wash.*

Supported by a grant from The National Institutes of Health (CA-03114).

1.7. Evidence from Rod-ring Experiments for the Duplicational Origin of f^{3N} Reversions in *Drosophila*. EDGAR ALTENBURG and LUOLIN S. BROWNING (Houston, U.S.A.).

It was previously suggested that the reversion of f^{3N} (an allele located in the left sub-

segment of the forked locus) might be due to duplication of the right forked sub-locus at a pre-meiotic division, a suggestion which would account not only for the relatively high reversion rate of f^{3N} (to f^{3N+}), but also for the restriction of the reversions in large measure to diploid cells (oogonia and spermatogonia) in irradiated material. Since a ring X would get lost much more often than a rod as the result of a duplication in a dividing chromosome, the recovered reversion rate of f^{3N} would expectedly be higher for a rod than a ring X, provided the reversions were due to duplications (whether X-ray induced or spontaneous). In accordance with the above suggestion, it has been found that the spontaneous reversion rate of f^{3N} is much higher in a rod X than in a ring as follows. Among 380,000 females heterozygous for a rod and a ring (but homozygous for f^{3N}) there were 12 reversions recovered in the rod and one in the ring. Insofar as these results would indicate that the forked locus arose as the result of a duplication, they would support the Lewis theory of pseudoallelism in *Drosophila*.

This investigation was supported by U.S. Public Health Service Research Grant GM 08889-02 from the Division of General Medical Science.

1.8. The Genetic Fine Structure of the Mutants z^m and z^l in *Drosophila melanogaster*. B. H. JUDD (Austin, U.S.A.).

The zeste locus in *Drosophila melanogaster* is located at 1.0 on the X chromosome. The mutant z^m (zeste-mottled) was found by Green as a single male ($sc z^m$) from the cross $sc z ec ct / w^{bf} \times sc z ec ct$. Later z^l (zeste-light) was found by Becker as a single male in the $sc z^m$ stock.

Analysis of z^m leads to the hypothesis that it does not represent a change at the zeste locus, but is the result of an asymmetrical exchange at the white locus. The white locus is very closely linked to zeste (1.5 on the X chromosome) and is functionally related. It is postulated that z^l arose as a similar asymmetrical exchange from z^m .

The change in the white locus can be localized within the two rightmost recombination sites of the locus. The phenotypic expression of this change leads to the conclusion that a portion of the white locus has been duplicated. Recombination experiments give support to this interpretation.

The duplicated nature of the two mutants

leads to further asymmetrical pairing and exchange within the locus when these chromosomes are used for crossover studies. A variety of unusual recombination products presumably resulting from asymmetrical exchanges have been analyzed.

1.9. Biochemical Division of the White Locus in *Drosophila melanogaster*. HUGH S. FORREST (Austin, U.S.A.).

Despite the fact that the *white* locus in *Drosophila melanogaster* has been known for some time to be divided into subloci separable by crossing over and other genetic tests, there is remarkably little knowledge of its fundamental biochemical role. It is believed by some workers, for example, that mutation at this locus results in an "incomplete" or "non-functional" pigment-synthesizing granule, resulting in loss of synthetic ability for both the red and brown pigments.

In a new attempt to investigate the biochemical function of the locus, it has been shown that all of the alleles at the locus so far tested can be divided into two groups—those that accumulate the compound xanthurenic acid, albeit in smaller amounts than wild type, and those that do not contain this compound at all. It is reasonable to assume that xanthurenic acid can be used as an index of the production of 3-hydroxykynurenine, an intermediate in the biosynthesis of the brown pigments. Thus the *white* locus may be separable into two functional areas, one of which, in the mutant condition, is unable to produce 3-hydroxykynurenine (measured as xanthurenic acid) and the other of which can produce limited amounts of this material. A theory relating this finding with the failure or partial failure of mutants of the *white* locus to synthesize the red eye pigments will be presented.

indistinguishable; they produce similar syndromes of abnormalities at similar times in embryogeny, and have similar effects on male transmission ratio and recombination. Combinations of members of different complementation groups are of course viable to some extent, but the proportion of viable compounds found varies greatly depending on the alleles concerned. The three new alleles reported here were found to belong to three different previously established complementation group. Their characteristics regarding embryonic effects, transmission ratio, and recombination followed the general pattern of being in each case indistinguishable from other members of their group. However, one of the new alleles was found to be clearly distinguishable from other members of its group on the basis of degree of complementation with another lethal allele. This suggests that alleles in the same complementation group may not have identical structures, and that quantitative measurements of the degree of complementation amongst different alleles may be used as a basis for assessing their similarity or dissimilarity.

1.11. Genetical Analysis of the R Chromonemal Region of *Mormoniella*. P. W. WHITING and DORIS J. BUSH (Philadelphia, U.S.A.).

Two eye-color factors, *O* and *S*, mutating with relatively high frequency to oyster-white and to scarlet respectively, make the "*R*" region of *Mormoniella* especially favorable for genetical analysis. These colors serve as markers for many other *R*-locus changes including semi-lethals and lethals, steriles and near-steriles. Complementation tests identify factor homologies. For factors such as lethals and male-steriles which cannot be transmitted through haploid males, diploid males heterozygous for these deleterious factors are used. Two different female-steriles have been found, masked in lethal-bearing genes. Two different lethals, complementary to each other and, therefore, non-homologous, have proved non-complementary, "homologous", to a third lethal. It is suggested that the last impairs some process essential to the normal functioning which is impaired by each of the other two lethals. Formula of each *R*-locus gene is given in terms of its factor states. By use of the gene formulae it is possible to predict the phenotypes and breeding behavior of the different compounds. It is postulated that the complexity of the *R* region is not greater than that of other chromonemal regions. Its complexity has been explored and revealed by

1.10. Complementation Groups at a Complex Locus in the House Mouse. DOROTHEA BENNETT and L. C. DUNN (New York, U.S.A.).

The study of three newly arisen alleles has provided the opportunity to compare the effects of lethals of independent origin, and especially to compare members of the same complementation group with respect to secondary effects. Complementation groups are defined as consisting of alleles from different sources which are lethal in combination. Alleles in the same complementation group are in general

use of oyster and scarlet mutations, acting as markers of each pleiotropic gene of the series. Data in full are at present being published in *Genetics*.

1.12. Partial Complementation of Six Multiple Alleles in a Chlorophyll Mutation Locus in Barley.
GERHARD HOLM (Lund, Sweden).

A locus in chromosome 1 contains at least 6 recessive vital viridis alleles which, when intercrossed, give F_1 's exhibiting different degrees of partial complementation judging from the visually examined leaf colours. A spectrophotometrical analysis of 8 of the F_1 's verified this interpretation and also showed that the degree of complementation in an F_1 apparently depends upon the relative position in the locus of the two alleles involved in the cross. The 6 alleles probably belong to the same cistron and overlap each other in a linear order. The less two alleles overlap, the higher the complementation in their F_1 .

The pigment content in 5 of the alleles is the same, 1.23 mg/g fresh weight, but in the 6th (no. 4 in the linear order) it is significantly higher, 1.44 mg/g fr.w. Normal plants contain 2.00 mg/g fr.w.

The 6 multiple alleles appeared in a material of 82 vital viridis mutations of which the resulting 76 most probably belong to single loci. 865 diallelic crosses were made in the group and the result indicates a very low frequency of multiple allele loci among the vital viridis mutations.

1.13. Fine Structure of Genes Conditioning Resistance to Erysiphe graminis hordei at the MI_a Locus in Hordeum vulgare. JOHN G. MOSEMAN (Beltsville, U.S.A.).

Several genes conditioning the resistant reaction of barley *Hordeum vulgare* L. to infection with the ascomycetous fungus *Erysiphe graminis* DC. f. sp. *hordei* Em. Marchal have been found at the MI_a locus on chromosome 5. Genes MI_a , MI_{a2} , and MI_{a3} at this locus were studied. When genes MI_a and MI_{a3} were homozygous they conferred a similar degree of resistance, but when heterozygous with a recessive gene conditioning susceptibility they conferred a different degree of resistance. Gene MI_{a2} conditioned a lesser degree of resistance than genes MI_a and MI_{a3} . The degree of resistance conferred when the genes were MI_aMI_{a2} , MI_aMI_{a3} , or $MI_{a2}MI_{a3}$ also was determined. For each gene conditioning

the reaction of the host, a corresponding gene has been found conditioning the pathogenicity of the pathogen. Pathogenically different cultures differentiated the genes at the MI_a locus. The relation of the pathogen genes corresponding to host genes was determined by the pathogenicity of haploid progeny cultures derived from crosses between selected cultures. Pathogen gene A_a corresponding to host gene MI_a was inherited independently of pathogen genes A_{a2} and A_{a3} corresponding to host genes MI_{a2} and MI_{a3} , respectively. Pathogen genes A_{a2} and A_{a3} were found to be at or near the same locus. The utilization of degree of resistance conferred, relation of corresponding pathogen genes, and other host-pathogen relations in studying fine structure of genes conditioning resistance to *E. graminis* f. sp. *hordei* at the MI_a locus in *H. vulgare* were discussed.

1.14. Complementation, Recombination, and Biochemical Relationships within the Td locus in Neurospora crassa. ANN MATTHEWS LACY (Baltimore, U.S.A.).

The present study of non-indole-utilizing (NIU) tryptophan synthetase deficient mutants indicates that the complementation and recombination maps of the Td locus are not co-linear. The clustering on the recombinational map bears some relationship to the biochemical characteristics of the mutants; however, the biochemical relationships between mutants grouped together by complementation appears somewhat obscure.

The twenty NIU complementing mutants tested are distributed among three of the four complementing groups previously established for NIU mutants (the fourth group is still represented by $Td7$ only).

The ability to form cross-reacting material (CRM) is not a prerequisite for complementation. Four CRM⁻ mutants are in the $Td71$ complementation group. These mutants, however, are not located near $Td71$ on the recombination map, but are located in the vicinity of $Td3$, 7, and 24 (i.e. "profound region").

The ability to catalyze the formation of indole from indole-glycerolphosphate is not limited to members of the $Td71$ complementation group, but can be detected at varying levels in crude extracts of at least 90% of the CRM⁺ NIU mutants tested (including $Td3$, 7, and 24). CRM⁻ mutants tested exhibit no detectable activity in this reaction.

Of three temperature sensitive mutants studied (mutant phenotype at 25°C, not at 37°C),

two are in different complementation groups and exhibit different levels of ability to form indole; the third neither complements nor forms indole.

The relationships between the NIU mutants will be described in detail and their possible significance in terms of gene structure and function will be discussed.

Supported by grants from the National Science Foundation.

1.15. Further Analysis of Interallelic Complementation at the *leu-2* Locus of *Neurospora crassa*. S. R. GROSS (Durham, U.S.A.).

A previous analysis of the complementation behavior of a large number of *leu-2* mutants of *Neurospora crassa* yielded a complementation map that was linear, overlapping and continuous.⁽¹⁾ Evidence was presented that indicated that the polypeptide whose structure was determined by the *leu-2* gene was at least one of two different polypeptide structural units of an enzyme β -carboxy- β -hydroxyisocaproic acid isomerase, an enzyme which catalyzes the isomerization of β -carboxy- β -hydroxyisocaproate and α -hydroxy- β -carboxyisocaproate, intermediates in leucine biosynthesis. An analysis of the complementation interaction between *leu-2* and *leu-3* alleles as well as enzymological analyses indicated that both the *leu-2* and *leu-3* loci were involved in the determination of the structure of β -carboxy- β -hydroxyisocaproic acid isomerase. The evidence obtained was interpreted as indicating that complementation between *leu-2* mutants resulted from a protein-protein interaction in the formation of a polymer consisting of α chains coded by the *leu-2* gene and β chains coded by the *leu-3* gene, and that at least in the case of enzymes synthesized as a result of complementation, the isomerase consisted of at least two α and two β polypeptide chains. A recent study of the physical chemistry of the isomerase obtained from several complementing pairs of *leu-2* mutants has revealed that the molecular weight of the normal enzyme as well as that of the heteroallelic enzymes are the same, ca. 80,000. The enzymes obtained by complementation are remarkably similar to the normal enzyme with respect to the functional area of the protein. Several physical properties of the heteroallelic enzymes differ markedly from that of the isomerase enzyme. These differences, however, are probably confined to areas not directly involved in substrate binding.

1. GROSS. *Proc. Nat. Acad. Sci.* **48**, 922, 1962.

1.16. Homology Tests on X-ray-induced Recessive Lethal Mutations in the *ad-3* Region of *Neurospora crassa*. F. J. DE SERRES (Oak Ridge, U.S.A.).

Forward-mutation experiments on a balanced heterokaryon⁽¹⁾ have shown that 76 per cent of the *ad-3* mutations are recessive lethal (*ad-3^{RL}*) on adenine-supplemented medium and that 40 per cent of these were *ad-3A ad-3B^{RL}* double mutants. Atwood and Mukai⁽²⁾ have shown that such recessive lethal mutations can be analyzed by homology tests. Such tests on the *ad-3^{RL}* mutations show that they form an inclusive complex; all combinations show homology except some of the *ad-3A^{RL} + ad-3B^{RL}* combinations. The apparent homology of certain *ad-3A^{RL} + ad-3B^{RL}* combinations is of particular interest since the trikaryon involving two such mutations in combination with a viable double mutant (from a cross of two viable mutants *ad-3A^V × ad-3B^V = ad-3A^V ad-3B^V*) is capable of growth on minimal medium. The behavior of these trikaryons shows that there is an intact essential region (designated region X) present in the *ad-3A^V ad-3B^V* double mutant that is non-functional in any of the *ad-3A^{RL}* or *ad-3B^{RL}* mutations showing homology. The simplest assumption is that region X is located between the *ad-3A* and *ad-3B* loci, and present data are consistent with this hypothesis. The interaction patterns of such *ad-3^{RL}* mutations in homology tests offer a unique opportunity for the analysis of the genetic composition of the *ad-3* region, and the results of these tests will be described in terms of a complementation map.

1. DE SERRES and OSTERBIND, *Genetics* **47**, 793-796, 1962.
2. *PNAS* **39**, 1027-35, 1953.

1.17. Complementation and Genetic Mapping of *pan-2* Mutants Induced in a Reversion of a Primary *pan-2* Mutant. MARY CASE (New Haven, U.S.A.).

Twenty-nine secondary *pan-2* (pantothenic acid-requiring) mutants have been obtained following X-irradiation of a reversion induced by X-rays in a complementing primary *pan-2* mutant *B5*. Thirteen of the 29 mutants complement with at least one primary mutant, and all 13 can be positioned on a linear complementation map of the locus. The behavior of the secondary mutants establishes the existence of an additional complementation unit at the *pan-2* locus. All

mutants were crossed to *B5* to determine whether the mutants are identical to *B5*. In addition, crosses were made to five other *pan-2* alleles located at various points on the genetic map of the locus. Two of the mutants map at the same site as *B5*; however, one of these mutants is a non-complementing type while the other exhibits a different complementation pattern from *B5*. Fourteen of the mutants are located near *B5* in the proximal region of the genetic map. Ten are located near the distal end of the map, while the three others are located at different sites near the middle of the map. The relationship between complementation and genetic maps of primary and secondary mutants will be discussed further.

Supported in part under a contract, AT (30-1)-872, with the U.S. Atomic Energy Commission.

1.18. Allelic Recombination and Complementation in Adenine-requiring Mutants of *Schizosaccharomyces pombe*. C. RAMIREZ, J. FRIIS and U. LEUPOLD (Bern, Switzerland).

Allelic recombination and complementation have been studied in several groups of mutants blocked in adenine biosynthesis, including two groups accumulating a red pigment (*ad₆* and *ad₇*) and one group blocked in an early reaction preceding the pigment blocks (*ad₁*).

In the *ad₇* group, 152 u.v.-induced allelic mutants have been demonstrated to represent mutations at 33 different but closely linked sites, recombination frequencies ranging from 1 to 77⁰ prototrophic recombinants per 10⁶ ascospores. The mutant distribution is characterized by "hot spots", one particular site having contributed as many as 26 per cent of the mutants. Complementation has not been observed in any of the pairwise mutant combinations tested.

In the *ad₁* group, however, complementation leading to a more or less prototrophic phenotype has been found in many of the diploid combinations tested. The complementation pattern of 138 mutants of u.v.-induced, diethylsulfate-induced and spontaneous origin may be represented by a linear complementation map in which eight types of complementation behaviour

define five different "complementation units". The complementation and recombination maps show a considerable degree of co-linearity.

A more complex complementation pattern has been found in allelic combinations of 158 u.v.-induced mutants of the constitution *ad₆*. The results of complementation tests involving 21 different types of complementation behaviour may be represented in a complementation map in which both ends are closed to circles. With a few notable exceptions, the complementation and recombination maps show again a significant degree of co-linearity.

1.19. Studies on the Genetic Fine Structure of the *ad₇* and *ad₆* loci of *Schizosaccharomyces pombe*. H. GUTZ (Berlin, Germany).

Leupold has done a very extensive intragenic recombination analysis in the *ad₇* and *ad₆* loci of *Schiz. pombe*. The *ad₆* locus shows the phenomenon of allelic complementation. In addition to the intragenic recombination analysis Leupold has constructed a complementation map of this locus. All mutants analysed by Leupold have been induced by ultraviolet irradiation. The *ad₇* and *ad₆* mutants are not randomly distributed in the maps. There are a few "hot spots", and the mutation sites appear to be concentrated in several parts of the map.

Taking the maps of Leupold as a basis, I have analysed the intragenic distribution and the complementation pattern of mutants which were induced by nitrous acid and X-rays. In both loci (*ad₇* and *ad₆*) the intragenic distribution of the nitrous acid-induced mutants is quite different from that of the u.v.-mutants. They show some pronounced "hot spots". Above half of the sites induced by nitrous acid are not present on the u.v.-map. From the non-random distribution of the mutants it is concluded that a direct deamination of the DNA-bases is not to be considered as mutation mechanism. All X-ray mutants map on single sites, and no intragenic deletions have been found.

According to the results of Leupold, the complementation map of the *ad₆* locus is non-linear. Seventy-nine per cent of the nitrous acid-induced mutants show complementation. Among these mutants at least 6 complementation types were found which are not present in the u.v.-mutants.

SECTION 2

RECOMBINATION

2.1. Factors Affecting Distributive Pairing between Nonhomologues in *Drosophila melanogaster*. RHODA F. GRELL (Oak Ridge, U.S.A.).

Meiotic associations that lead to the regular separation of nonhomologous chromosomes occur with high frequencies in *Drosophila melanogaster* females of particular genotypes. The relation of the size of the nonhomologues to the regularity of their association has been investigated by measuring the nondisjunction frequencies between a free fourth chromosome and a series of X-duplications in the progeny of females of the genotype $y^2/y^2/Dp(1;f), y^+; T(3;4)86D, red/T(3;4)86D, red; ci^{10}$ mated to $xy, yB/Y$ males. The duplications were obtained and characterized by Cooper and Krivshenko and vary in size from ~ 0.3 to 3.0 times the metaphase length of chromosome four. The percent of nondisjunction between chromosome four and the duplication, given in ascending order of duplication size, are, $/ \sim 0.3 = 4.49 \pm 0.19, / 0.5 = 0.39 \pm 0.07, / 0.7 = 0.12 \pm 0.06, 0.9 = 0.08 \pm 0.03, 1.0 = 0.13 \pm 0.05, 1.1 = 0.02 \pm 0.02, 1.1 = 0.08 \pm 0.03, 1.4 = 0.38 \pm 0.08, 1.6 = 0.39 \pm 0.08, 2.0 = 0.42 \pm 0.07, / 2.5 = 2.03 \pm 0.39, 3.0 = 1.89 \pm 0.24 /$. The lowest frequencies of nondisjunction (~ 0.08 per cent) occur with those duplications closest in size to chromosome four (0.7 - 1.1) whereas the highest frequencies occur with the smallest (~ 0.3) and largest (2.5 and 3.0) duplications.

The results indicate size alone is not responsible for pairing efficiency since the progressive change in duplication size is not associated with correlated changes in nondisjunction frequency. Instead, the duplications fall into five discrete classes (designated by slashes in the sequence above) within which size variation causes no apparent related change in segregation behavior. This suggests the possibility of sites that participate in nonhomologous associations as has been postulated by Gershenson and Cooper for homologous associations.

2.2. Nonrandom Assortment of Compound Chromosomes with Nonhomologous chromosomes in Oocytes of *Drosophila melanogaster*. E. H. GRELL (Oak Ridge, U.S.A.).

The requirements for nonrandom assortment of nonhomologous chromosomes was studied by R. F. Grell (1962). For noncompound chromosomes it was determined that a chromosome must not have been a crossover. (The term compound chromosome is intended to include chromosomes formed by the joining of two homologous chromosome arms to one centromere.) If two nonhomologous chromosomes are noncrossovers they may pair and pass to opposite poles of the first meiotic division spindle. The pairing in which nonhomologous elements may participate has been termed *distributive pairing* to distinguish it from *exchange pairing* which occurs prior to crossing over and only involves homologous chromosomal regions.

The studies reported here deal with distributive pairing of nonhomologous chromosomes when at least one member of the pair is a compound chromosome. A familiar compound chromosome is the attached X. The two arms are attached in reverse order on either side of a centromere. Crossing over occurs between the two arms with almost the same frequency as for normal free X chromosomes. Yet, from females of the genotype $xx/O; SMI, Cy/T(2;3)A$, 90 per cent of the recoverable gametes have the attached X or SMI, Cy and only 10 per cent have both or neither chromosome. Despite the fact that more than 95 per cent of the tetrads contain an exchange, the attached X shows a very highly nonrandom assortment with a second chromosome. Experiments that compare compound X's with an exchange and those without an exchange indicate that in contrast to free X's, exchange has no influence on the extent of nonhomologous pairing. All compound chromosomes tested (various compound X's and the attached 4's) have shown nonrandom assortment with nonhomologous chromosomes.

2.3. On the Nature of the Event that leads to Recombination of Pseudoalleles of the *m-dy* locus in *Drosophila*. ALLAN B. BURDICK, ROBERT A. SHLESER, EVELYN BARBOUR BENDBOW, and ROSANNE ABBADESSA (Lafayette, U.S.A.).

The recombination map of the *m-dy* locus

shows an unambiguous sequence of five separable mutant elements extending over about 0.07 map units. The sequence is $v \dots m^{61e}$, m^{59a} , m , dy , $dy^{60k} \dots g$ with several other elements being inseparable from one or more members of the sequence. A number of insertional-type crossover products (double and triple crossovers with respect to the outside markers) have been obtained from free-X recombination tests. These have involved nine different elements of the locus, and their explanation poses a problem as to the nature of the event that leads to recombination of the elements of the locus. Although the *cis-trans* test of the most distal elements (m^{61e} and dy^{60k}) indicates that they occupy separate functional sites, complementation tests among the various elements indicate that they could all belong to the same cistron. The $m^{61e} dy^{60k}$ *cis*-phase double mutant has been tested in *trans*-phase with all of the internal elements of the locus and has never yielded a wild-type recombinant—that is, no double crossing over takes place within the locus—although $m^{61e} dy^{60k}$ has broken down several times to yield dy^{60k} types. Attached-X recombinations of m^{61e} and m yielded 31 events of which 14 were reciprocal. One of the $m^{61e} m$ *cis*-phase double mutants has been *trans*-phase tested to m^{61e} , m^{59a} , and m and has not given any wild-type recombinants. Our present opinion is that recombinations within this locus are of the same classical reciprocal kind that have been postulated for interlocus recombinations, and that the insertional-type crossovers we have obtained can be explained on a classical basis.

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2.4. Genetic Ultrafine Structure of the T4rII Region.

IRWIN TESSMAN (Lafayette, U.S.A.).

Recombination frequencies as low as 10^{-6} per cent have been measured in the *rII* region of phage T4. At this sensitivity, hot spots in the *rII* region have been resolved into two or more genetic sites, separable by recombination. No lower limit to recombination frequencies is apparent. Therefore, lack of recombination cannot guarantee identity of genetic sites.

The largest recombination frequencies in the *rII* region are greater than 1 per cent and the lowest less than 10^{-6} per cent. The ratio, 10^6 , is greater than the number of nucleotides in the entire phage. Therefore, in this range there

must be extreme deviations from even rough additivity of recombination frequencies. Within the range of very low recombination frequencies, marked deviations from additivity are, in fact found.

2.5. Effect of Negative Interference on Genetic Map

Concepts and Its Consequences for Intrallelic Recombination Studies. W. D. HANSON (Raleigh, U.S.A.).

Experimental studies involving intra-allelic recombination have yielded results which are not entirely compatible with classical recombination concepts. Negative interference noted in intra-allelic recombination studies suggested that given a genetic recombination at a point one could expect additional points of recombination or a "cluster" of points of recombinations (one or more) within some interval. The products of meiotic division were described in a probability space so that the probability of chromosome types could be formulated. The products of meiosis for linked loci were formulated for classical recombination studies and for intra-allelic recombination studies assuming that cluster recombinations could occur in some interval. The results involved the probability of an "odd cluster", length of cluster interval, and genetic map distance between linked loci. Cluster recombinations within a relatively short interval should not upset classical recombination measures. The frequency of chromosome types (with respect to marker genes) with selection for intra-allelic recombinations reflected a bias identified with cluster effects. Relative reverse mutation rates could also be a factor; however, for crosses involving a locus bounded by two markers a contradiction existed for linear order of alleles vs. relative mutation rates. A positive bias in map distance involving a non-selected marker was associated with cluster recombinations which was a maximum for a closely linked marker and decreased with increased map distance. Published data supported cluster recombination concepts, as compared to mutation, with a positive bias in recombination being a consistent feature in these data.

2.6. Polarized Intragenic Recombination in *Neurospora crassa*. NOREEN E. MURRAY (Stanford, U.S.A.).

Methionine-independent progeny from crosses between many pairs of combinations involving

nineteen alleles at the *me-2* locus were classified with respect to the markers present on both sides of the *me-2* gene. One of the two classes of methionine prototrophs having markers recombined occurred in excess over the other; when the markers entered the cross in the opposite phase, a similar excess was found in the reciprocal class. The linear order of *me-2* alleles determined from these asymmetries was consistent with a map based on prototroph frequencies.

Pronounced asymmetries were also observed in the numbers of the two parentally-marked classes of prototrophs; when the markers entered the cross in the opposite phase these asymmetries were reversed. The asymmetries between the two parentally-marked classes were correlated in direction with the asymmetries between the two classes having markers recombined.

The results may be interpreted in terms of multiple exchanges within small, discontinuously distributed, regions of effective pairing—the asymmetries would result from a reduction of coincident exchanges in the region proximal to the selected interval.

The coincident exchange frequency in the proximal region is influenced by the position of the more proximal of the two *me-2* alleles, but is independent of the recombination frequency between the two alleles. The reduced coincidence in the proximal region could be explained if the *me-2* region is situated immediately distal to some discontinuity which imposes a nonrandomness of exchanges by reducing the chance that the effectively paired region could be proximal to, yet extend into, the *me-2* region.

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2.7. Conversion and Crossing-over as Recombination Mechanisms in *Ascobolus immersus*.

W. GAJEWSKI, A. KRUSZEWSKA, A. MAKAREWICZ, A. PASZEWSKI, S. SURZYCKI and H. BIELAWSKA (Warsaw, Poland).

Crosses between mutants with white ascospores from one series give wild type, dark recombinants due predominantly to conversion. The frequencies of conversion-type asci are roughly proportional to the distances between the sites involved which enables one to map them. When mutants from one series are crossed with wild type strain the frequencies of 2:0 asci increase in percentage from one end of the series to the other approaching the value of recombination between the extreme sites of the series.

This is interpreted that the frequency of 6:2 asci in crosses between two white mutants represents only that part of copy choices which begin or end in between two sites. It seems that the beginning of the switches is random along the analysed unit whereas the return to the original matrix is rather nonrandom.

One mutant, namely 186, crossed with wild strain shows very high frequencies (10-12 per cent) of 6:2 and 2:6 asci of conversion type. Besides, less frequent asci 5:3, 3:5, 7:1 and 1:7 also appear. They are not due to aberrant chromosome segregation or mutation. The asci 5:3 and 3:5 result probably from copy errors of half-chromatids. The 7:1 and 1:7 asci may be the result of two copy errors or two successive replications. The tetrad analysis of visible ascospore mutants in *Ascobolus immersus* on sufficiently large scale reveals the predominant role of conversion as a mechanism of recombination within a small piece of genetic material.

2.8. Effect of Crossing-over on the Process of Spontaneous Mutation. G. E. MAGNI (Pavia, Italy).

It has been recently observed that the rates of spontaneous mutations occurring during meiotic division are higher than those occurring during vegetative reproduction (mitosis) in the same type of cells (yeast).⁽¹⁾

Further experiments have shown that most spontaneous back-mutations at locus *hi₁₋₁* during meiosis, in either one of the two identical alleles carried by diploid yeast cells, are connected with crossing-over in the specific locus.

Data regarding the amount of negative interference in the adjacent chromosome regions and the polarization of the intragenic crossing-over will be presented.

The hypothesis of unequal crossing-over within the locus leading to back mutant chromatids will be discussed.

1. MAGNI and VON BORSTEL, *Genetics* **47**, 1097, 1962.

2.9. Further Investigations on Somatic Gene Conversion in the Tomato. RUDOLF HAGEMANN (Gatersleben, Germany).

In heterozygous *sulf⁺sulf* plants a green yellow variegation occurs (*sulf* means any mutant allele of the chlorophyll deficient *sulfurea* series either of the *sulf^{pura}* or of the *sulf^{vsg}* group)

The genetic analysis demonstrated that the *sulf*⁺ allele, stable in homozygous condition, becomes unstable in presence of a *sulf* allele. This type of allele-induced instability in somatic cells is termed somatic gene conversion.⁽¹⁾

Further experiments have shown:

(1) A two-element-system (as found in maize) is not involved.

(2) In hybrids between *sulf* homozygotes and taxa of the subgenus *Eulycopersicon* the percentage of variegation is only determined by the "conversion activity" of the special *sulf* allele present. The environmental conditions (temperature, nutrition, illumination) do not significantly alter the percentage of variegation. Conversion-table *sulf*⁺ alleles have not yet been found.

(3) In hybrids between *sulf* homozygotes (*Lycopersicon esculentum*) and *L. hirsutum* (subgenus *Eriopersicon*) or *Solanum pennellii* variegation occurred very seldom, thus indicating an influence of the genotypic milieu.

(4) The somatic conversion is confined to the *sulf* locus. But as to the different alleles, conversion is not highly directed. The *sulf* allele, converted from *sulf*⁺, need not be identical with the *sulf* allele already present; e.g. in a *sulf*⁺ *sulf*^{pura-90} per cent plant the *sulf*⁺ allele is converted to *sulf*^{vag} more frequently than to *sulf*^{pura}, and even the *sulf*^{pura} alleles arisen may differ in their conversion activity from *sulf*^{pura-90} per cent.

1. Z. Vererbungsl. 89, 587, 1958.

2.10. Preliminary Experiments on the Chemistry of Crossing-Over. SHELDON WOLFF and FREDERICK J. DE SERRES (Oak Ridge, U.S.A.).

Experiments with metabolic inhibitors have indicated that protein synthesis is necessary for the rejoining of radiation-induced chromosome breaks. Because of these experiments Wolff has postulated that the bonds formed at rejoining are in protein. Although crossing-over may in some ways be likened to breakage and rejoining there are some differences. We, therefore, treated *Neurospora crassa* with specific metabolic inhibitors at the time of crossing-over to see if recombination is affected when certain cellular synthetic processes are blocked. In particular, the experiments were designed to see whether or not protein synthesis inhibitors which can inhibit the rejoining of radiation-induced breaks can affect crossing-over.

Several experiments of two different types were

performed. In the first type the marker asco that gives rise to white ascospores was used. The asci were dissected and second division segregations scored. In the second type biochemical markers that were separated by various distances were used and recombinants scored.

The protein synthesis inhibitor, chloramphenicol which inhibits rejoining and also inhibits growth of *Neurospora*, did not affect recombination. We interpret this to mean that protein synthesis is not involved in crossing-over.

The only inhibitor that gave consistently higher recombination values when added at the time of meiosis was 5-fluorodeoxyuridine (FUdR) a compound that, in some systems, has been shown to be a specific inhibitor of DNA synthesis.

Bromodeoxyuridine (BUdR) a compound that is structurally similar to FUdR but is a thymidine analog that becomes incorporated into the DNA did not have any effect on recombination nor did uridine which is usually added with FUdR to prevent effects on RNA.

The simplest explanation for these experiments is that genetic recombination, which entails the exchange of chromatids, is specific for DNA and that a functional mechanism for DNA synthesis is required when the bonds rejoining the two pieces are synthesized. This is unlike the rejoining or repair of X-ray induced breaks that are non-polarized and are inhibited by chloramphenicol.

2.11. Tetrad Analysis of Crosses of *Neurospora crassa* Grown on Media Containing Various Concentrations of 5-bromouracil and Cytidine Sulfate. S. F. H. THRELKELD (Hamilton, Canada).

Crosses of two *Neurospora crassa* strains *A*, *arg-3* (30300), *cr*, *tryp-2* (75001), *ylo* (Y30539y) and *a*, *pyr-1* (H263), *pdx-1* (37803) were made on various media, by conidiating 5-day-old cultures of the protoperithecial parent, *a pyr-1 pdx-1*, with conidia from the other strain.

Standard *Neurospora* reproductive medium was supplemented in the following ways to give four types of media: (i) cytidine sulfate 100 mg/l., 5-bromouracil 0 mg/l.; (ii) cytidine sulfate 100 mg/l., 5-bromouracil 100 mg/l.; (iii) cytidine sulfate 45 mg/l., 5-bromouracil 100 mg/l.; (iv) cytidine sulfate 45 mg/l., 5-bromouracil 0 mg/l.

Crosses grown on media (i) and (ii) showed no significant differences with respect to ascus patterns or recombination frequencies. Com-