

AN ESTIMATE OF THE MUTATIONAL DAMAGE IN MAN FROM
DATA ON CONSANGUINEOUS MARRIAGES*

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In a diploid, outbreeding organism like man the deleterious mutants carried by the population are only partly expressed in each generation, being largely concealed by heterozygosis with more favorable alleles. However, the total hidden mutational damage carried by the population can be estimated indirectly from the detrimental effects of consanguineous marriage.

This method, applied to mortality data of Arner,¹ provided the basis for the statement that "a calculation from . . . results of inbreeding in man . . . leads to the conclusion that every person on the average contains heterozygously at least one lethal gene or group of genes which [homozygously would] . . . kill an individual . . . between birth and maturity."² The calculation itself was not given, however, and the stated figure of one lethal equivalent per person represents a conservative estimate, being a good deal lower than the most probable value (nearly two) actually indicated by the data. Recently Slatis³ has used a similar procedure for estimating the number of heterozygous genes that, if homozygous, would cause detectable rare abnormalities and has arrived at a tentative estimate of eight such genes per person. His conclusions are qualified by the fact that the subjects were selected for having abnormalities, some of which may not have been simple recessives.

In this paper we shall present calculations whereby, using death rates both from Arner's and from two other published studies of consanguineous marriages, we have attempted to measure the total mutational damage. We shall also show how, by making some assumptions about the manner in which the mutations are expressed (and hence with less assurance), we have estimated the amount of mutational damage actually expressed each generation. Finally, we shall use the data to estimate the total mutation rate in man.

Human Consanguinity Data.—The selection of families on the basis of the consanguinity of the parents has both advantages and disadvantages. The disadvantages of this approach are that a large sample is required and that the sociological concomitants of consanguineous marriage (rural-urban differences, etc.) may be confounded with the genetic effects. The advantages are that it is not biased by selection of particular genetic entities and that homozygosity for two or more deleterious genes with possibly synergistic effects is unlikely at the low levels of inbreeding found in man.

Three published studies on consanguineous marriage fulfil the condition of a large sample. It is questionable whether they also meet the requirement for separation of genetic effects from the sociological correlates of inbreeding. The most recent and useful data (summarized in Table 1) were obtained by Sutter and Tabah⁴ from

Catholic marriage dispensations issued during 1919-1925 in two French departments. These authors visited about two-thirds of the families and took histories of births and deaths, with notes on conspicuous abnormalities. The same information was obtained from town clerks for a control sample of unrelated parents married during the same period and selected without regard to fertility or medical history.

Arner¹ obtained his data by going through early American genealogies and recording, among other things, the number of deaths before age 20 in the children of consanguineous marriages. Nonconsanguineous marriages of the parents' siblings served as controls. He does not give the number of deaths for each kind of consanguineous marriage beyond first cousins, but only an average. However, elsewhere in his paper he gives data from which the approximate composition of this group may be inferred, and from this we have estimated the average inbreeding coefficient. The relevant data are given in Table 2.

TABLE 1
CHILD MORTALITY: DATA OF SUTTER AND TABAH
(Data Given as Deaths/Total, with Proportion of Deaths Below)

	First Cousins ($F = .0625$)	$1\frac{1}{2}$ Cousins ($F = .0312$)	Second Cousins ($F = .0156$)	Not Related ($F = 0$)
<i>Morbihan:</i>				
Stillbirths and neonatal deaths	51/461	3/78	23/309	72/1,628
	.111	.038	.074	.044
Infantile and juvenile deaths	64/410	17/25	32/286	138/1,556
	.156	.227	.112	.089
<i>Loir et Cher:</i>				
Stillbirths and neonatal deaths	18/282	6/105	11/240	36/1,117
	.064	.057	.046	.032
Infantile and juvenile deaths	32/264	1/99	17/229	60/1081
	.121	.010	.074	.056

TABLE 2
CHILDREN DYING UNDER THE AGE OF 20: DATA OF ARNER

First Cousins ($F = .0625$)	Other Cousins ($F = .0112$)	Not Related ($F = 0$)
113/672	211/1,417	370/3,184
.168	.149	.116

The third source of data is a very old study by Bemiss⁵ based on correspondence with physicians, with consequent unintentional selection of families with conspicuous abnormalities. We have included his data on "children dying young" in Table 3. Although the abnormalities are undoubtedly selected, few of them were incompatible with life, and in fact the incestuous group (parent-child and sib matings, not included in Table 3), which recorded 29 of 31 as defective, included no

TABLE 3
CHILDREN DYING YOUNG: DATA OF BEMISS

Uncle-Niece ($F = .1250$)	Double First Cousins ($F = .1250$)	First Cousins ($F = .0625$)	Second Cousins ($F = .0156$)	Third Cousins ($F = .0039$)	Not Related ($F = 0$)
23/53	55/154	637/2,778	85/513	8/59	134/837
.434	.357	.229	.166	.136	.160

deaths. It is likely, therefore, that the data on deaths are not seriously biased by selection. Yet it is to be expected that these data would be less reliable than those from the other studies, especially the modern careful studies of Sutter and Tabah.

Definitions.—We wish to distinguish between *total* mutational damage and *expressed* mutational damage, both measured for the purposes of this paper in *lethal equivalents*.

A *lethal equivalent* is a group of mutant genes of such number that, if dispersed in different individuals, they would cause on the average one death, e.g., one lethal mutant, or two mutants each with 50 per cent probability of causing death, etc. The concept will be illustrated in a later section.

The *total mutational damage* per gamete is the average number of lethal equivalents in the zygote that would result from doubling the chromosomes of this gamete.

The *expressed mutational damage* per gamete is the average number of lethal equivalents in this gamete that would be expressed if it were combined with another gamete to form a zygote according to the mating system actually prevailing among the individuals being considered.

An Estimate of the Total Mutational Damage.—Considering a single locus, the probability of a particular zygote surviving the detrimental effects of mutants at this locus is⁶

$$1 - \underbrace{qF^2s}_{\substack{\text{Probability of} \\ \text{death due to} \\ \text{homozygosity} \\ \text{from consanguinity}}} - \underbrace{q^2(1-F)s}_{\substack{\text{Probability of} \\ \text{death due to} \\ \text{homozygosity not} \\ \text{from consanguinity}}} - \underbrace{2q(1-q)(1-F)sh}_{\substack{\text{Probability of} \\ \text{death in} \\ \text{a heterozy-} \\ \text{gote}}}$$

where s is the probability of death in the mutant homozygote and h is a measure of dominance, being 0 for a completely recessive factor and 1 for a gene causing the same probability of death in a heterozygote as in a homozygote. F is Wright's⁷ coefficient of inbreeding and measures that fraction of loci that are homozygous as a result of consanguinity, being $1/16$ for children of first cousins, $1/32$ for those of first cousins once removed ($1\frac{1}{2}$ cousins), $1/64$ for those of second cousins, etc.

We make the assumption that different causes of death, genetic or environmental, are independent in action ("nonsynergistic" in the sense used by Muller⁸). On this model the fraction of survivors is

$$S = \Pi(1 - x)\{1 - qF^2s - q^2(1 - F)s - 2q(1 - q)(1 - F)sh\},$$

where x is the probability of a particular environmental cause of death, and the product is taken over all environmental causes and over all loci with mutant alleles. Since the number of causes is large and the separate probabilities are small, this is equivalent to

$$S = e^{-\Sigma x - F\Sigma q^2s - (1-F)\Sigma q^2s - 2(1-F)\Sigma q(1-q)sh} \\ = e^{-(A+BF)},$$

or

$$-\log_e S = A + BF,$$

where $A = \Sigma x + \Sigma q^2s + 2\Sigma q(1 - q)sh$ and $B = \Sigma qs - \Sigma q^2s - 2\Sigma q(1 - q)sh$. The summation is over all environmental factors, or over all loci having mutant alleles.

In a randomly mating population ($F = 0$) the amount of expressed damage is measured by A . B is a measure of the hidden genetic damage that would be expressed fully only in a complete homozygote ($F = 1$). We take as a measure of total genetic damage per gamete the quantity Σqs , this being the amount ex-

pressed in a zygote formed by doubling the chromosomes of this gamete. This quantity is equal to the sum of B and the genetic component of A and hence lies between B and $B + A$.

Estimates of A and B were obtained from the weighted regression on F of the natural logarithm of the number of survivors. According to maximum-likelihood theory, the appropriate weights are $nS/(1 - S)$, where S is the expected fraction of survivors and n is the total number. The weights were obtained by iteration, starting with the observed value of S as a trial value. The values of A and B computed in this way are shown in Table 4. Because of the low levels of inbreeding found in man and the small number of deaths in the noninbred groups, virtually the same estimates of A and B are obtained from the simple approximation $S = 1 - A - BF$. Furthermore, since the effects of these factors as actually felt by future populations are dispersed over many individuals, the estimate of B is not greatly influenced by the way in which a large number of lethal effects (genetic and environmental) interact in an individual.

TABLE 4
ESTIMATES AND HOMOGENEITY TEST

	A	B	B/A	B_{FF}	B_{FO}	B_{CO}	χ^2
Sutter and Tabah (1953), Morbihan:							
Stillbirths and neonatal deaths	.0460	1.124	24.41	.901	1.233	1.163	.36
Infant and juvenile deaths	.0950	1.431	15.06	.937	1.665	1.222	.88
Total	.1410	2.555	18.12
Sutter and Tabah (1953), Loir et Cher:							
Stillbirths and neonatal deaths	.0335	.574	17.12	.398	.662	.538	.24
Infant and juvenile deaths	.0558	.908	16.26	1.201	.759	1.141	.36
Total	.0893	1.482	16.60
Arner (1908)	.1300	1.032	7.94	.446	1.803	.970	4.94*
Bemiss (1858)	.1612	1.734	10.75	2.193	1.392	1.371	2.73

It is possible that in any of these studies the consanguineous and nonconsanguineous groups are not comparable in some respect. For example, in the data of Sutter and Tabah the consanguineous group was interviewed by the authors, but the control data were gotten more indirectly. Also, especially in the Bemiss study, there is doubt about the accuracy of ascertainment of the more distant degrees of relationship. Further, there may be undetected environmental differences between the inbred and control groups. For these reasons we have made several tests for internal consistency of the data.

In Table 4 the regression coefficients B_{FF} were obtained from the different degrees of consanguinity, omitting the noninbred group entirely. This is to be compared with B_{FO} , which is based on the comparison of the noninbred group with the average of all the inbred groups. The χ^2 values are for the comparison of these two regression coefficients. The only data that show any evidence of inconsistency ($B_{FF} = .45$, $B_{FO} = 1.80$, $\chi^2 = 4.94$, $P \sim .03$) are Arner's, yet his material was collected with the greatest attention to an adequate control, and the computed values of A and B are in reasonable agreement with the other data. Possibly we have been misled in our indirect estimate of F for his "other cousins" group. An additional value is also given in the table, B_{CO} , based on comparisons of the outbred group with those from first-cousin marriages only. This is of special interest in the Bemiss study, since there is some doubt as to his definition of second and third cousins, and in the Arner study for the reason just given.

The Sutter and Tabah data include stillbirths and recorded late miscarriages, whereas the Arner and Bemiss data include mostly postnatal deaths. All these studies include deaths up to early adulthood. Making allowance for the incomplete stillbirth data in the Arner and Bemiss studies, we conclude that B for stillbirths plus juvenile deaths probably lies between 1.5 and 2.5, with $A + B$ only slightly larger. That is, the average gamete carries a group of detrimental factors that, if dispersed in separate individuals and made homozygous, would result in 1.5–2.5 deaths of that age group. Thus the total genetic damage here measured is 1.5–2.5 lethal equivalents per gamete, or 3–5 per zygote.

At loci with complete or partial dominance the genetic damage measured by this procedure is due to mutation. But overdominant loci where the heterozygote is fitter than either homozygote make a contribution to inbreeding decline, and hence to B , that is not related directly to mutational damage. It can be shown (Crow, unpublished) that if the two homozygous types have a selective disadvantage of s and t relative to the heterozygote, the genetic damage in a randomly mating population (i.e., the amount by which the population is less fit than if it were made up entirely of the optimum heterozygous type) is proportional to $st/(s + t)$, whereas if this population is made completely homozygous it is proportional to $2st/(s + t)$. Thus the contribution to B of such a locus is exactly equal to its contribution to A . In our data B is some fifteen times the value of A , and the latter includes non-genetic deaths, so we conclude that overdominant loci are not making any substantial contribution to B and that the genetic damage we are measuring is mutational.

These data omit abortions, early adult deaths, and cases of infecundity. Moreover, B would be still higher if genetic impairments that influenced the survival or reproduction of offspring or other relatives of inbred individuals were included. Thus the value of B taken to include these cases is probably at least twice as great as we have given. Furthermore, one lethal equivalent probably comprises several detrimental mutants. Therefore, every individual must be heterozygous for many genes which would be seriously deleterious if homozygous and which together probably produce an appreciable loss of fitness even in the heterozygote.

An Estimate of the Expressed Mutational Damage.—Given the observation that there are the equivalent of 3–5 lethals acting in late fetal to early adult stages per zygote, we can estimate the amount of damage expressed in a single generation. The probability of a particular mutant being eliminated by death due to homozygosity through inbreeding is Fs ; that of elimination due to the mutant meeting a pre-existing allele is $(1 - F)qs$; and that of elimination due to the mutant in a heterozygote is $(1 - F)(1 - q)sh$. Neglecting products of small quantities, the total probability of elimination is approximately $(F + q + h)s$. We shall designate $F + q + h$ by z .

The mutant genes actually found in the population will be determined in part by the number of generations that each mutant persists before elimination, and therefore the more completely recessive genes will contribute disproportionately to the inbreeding effect. The mean persistence of a mutant gene is the reciprocal of its probability of being eliminated in any particular generation and is therefore $1/zs$. Therefore, the number of mutant genes per gamete in the population is $\Sigma(\mu/zs)$, where μ is the mutation rate and the summation is over all relevant loci.

The total number of lethal equivalents per gamete is $\Sigma[(\mu/zs)s]$, or $\Sigma(\mu/z)$, whereas the number of expressed lethal equivalents per gamete is $\Sigma[(\mu/zs)zs]$, or simply $\Sigma\mu$. If z and μ are uncorrelated, $\Sigma(\mu/z)$ may be written as $(\Sigma\mu)(1/z)$. In that case, if estimates of $1/z$ and of lethal equivalents are obtainable, this formula can be used to find $\Sigma\mu$, the total mutation rate. Now although q and μ are clearly correlated, the correlation between z and μ is not likely to be large. For q has probably comprised but a small part of z either in early times or at present, and although Muller⁸ has suggested that selective processes might cause some correlation between h and μ , their effectiveness is not expected to be great. At any rate, to whatever extent z and μ may be positively correlated, this method will underestimate $\Sigma\mu$.

On the assumption that $\Sigma(\mu/z) = (\Sigma\mu)(1/z)$, the expressions for total lethal equivalents and for expressed lethal equivalents have a factor, $\Sigma\mu$, in common, which cancels out, leaving the following simple relationship: *The number of expressed lethal equivalents per gamete is the total number of lethal equivalents multiplied by the harmonic mean of z .*

There are several reasons for thinking that most elimination of deleterious genes is now in heterozygotes rather than in homozygotes. Direct measurements on *Drosophila* lethals and semilethals have shown a mean dominance of about 4–5 per cent.^{9, 10} This was foreshadowed by Sturtevant's observation¹¹ that the number of lethals carried by wild populations of *Drosophila* is smaller than would be predicted with random mating (the occurrence of which was, however, questionable) and the observed rate of mutation. It was also indicated by the fact that most deletions of any magnitude have a depressing effect on viability as heterozygotes,^{8, 9} by dosage compensation,¹² and by numerous observations of incomplete recessivity in various organisms, including especially those of Levit¹³ on man.

To get the harmonic mean of z , which for the reasons just given we consider to be in large measure determined by the heterozygous effect of the mutants, we used the *Drosophila* data of Muller and Campbell (unpublished). Their values of h for 16 autosomal lethals range from .091 to -.026. The variance of these values (.00185) does not differ significantly from the variance of repeated observations on the same lethal (.00102), so it is possible that the values simply represent random deviations a true mean value of .042, constant for all lethals. In this case the harmonic mean of h would be the same as its arithmetic mean, or about .04. However, it seems more likely a priori as well as from the evidence given by visible mutants that the true value differs from mutant to mutant, but by less than the observed range would indicate because of measurement errors. We take the observed values and arbitrarily regress each toward the mean by the ratio of the "true" standard deviation (obtained by subtracting the variance of replications of tests on the same lethal from the variance among means of different lethals) to the observed standard deviation, compute for each gene the value of z , and take the harmonic mean. For this purpose the mutation rate per locus was assumed to be 10^{-5} , but it makes very little difference what value is chosen. With $F = 0$ the harmonic mean of z is .013, with $F = .001$ it is .014, with $F = .005$ it is .022, and with $F = .01$ it is .030.

There is a difficulty here in that the gene frequencies may have been largely determined at a time when F was large, but the present expressed damage is in populations with very little inbreeding. If we compute the equilibrium frequency of each mutant in a hypothetical population with $F = .01$, then compute the expressed

damage in a population changed to $F = .001$, the result is .023 of the total damage.

It should be noted that mutants with nearly neutral or slightly favorable heterozygotes dominate this value out of all proportion to their initial rate of occurrence. For example, if only two mutants are omitted from the data of Muller and Campbell, the value is raised to nearly .04.

The data of Stern¹⁰ and ten of the sixteen cases studied by Muller and Campbell were based on complete lethals, which, as pointed out by the latter workers, may be more dominant in their effect on survival than mildly deleterious genes, since the lethal homozygotes may be more than sufficient to kill the embryo. For example, Seto¹⁴ showed that lethals that kill in the egg stage of *Drosophila*, which are presumably therefore more drastic, have a greater amount of heterozygous lethality than those killing at later stages. Muller and Campbell, however, do not show a conspicuously or significantly lower dominance for the near-lethals than for the complete ones (.039 versus .044). Moreover, there is some ground for the opposite inference that mutants at loci giving rise mainly to slight detrimentals would have a higher dominance than marked detrimentals because of a lower selective pressure acting to stabilize the expression of the loci.

We shall take the harmonic mean of z as .02. With 1.5–2.5 lethal equivalents per gamete, this corresponds to 3–5 per cent of expressed lethality per gamete, or nearly 6–10 per cent per zygote. The zygotic value is somewhat less than twice the gametic, since with homozygous deaths two lethals lead to only one zygotic death. However, this correction is small if most eliminations are in heterozygotes, as we judge them to be in modern populations. Synergism between different loci would also cause this estimated value to be too high, but for reasons given earlier this is not likely to introduce any sizable error.

Comparison with the values of A in Table 4 suggests that a substantial fraction of deaths in nonconsanguineous marriages may be attributed to heterozygous effects of the same factors that cause deaths as homozygotes in consanguineous marriages; that is, these deaths are in a large measure genetically selective.

An Estimate of the Rate of Occurrence of Detrimental Mutation.—From the relation given in the last section that the total number of lethal equivalents per gamete is $(\Sigma\mu)(1/z)$, we can compute the mutation rate as the total number of lethal equivalents multiplied by the harmonic mean of z . Taking 1.5–2.5 as the total number per gamete and .02 as the harmonic mean of z , the total mutation rate of lethals and detrimentals causing deaths from late fetal to early adult states is .03–.05 per gamete per generation. This corresponds to an inbreeding coefficient of .005; if F in the past were as much as .01, the estimated mutation rate would be about 50 per cent greater.

If we assume that the total lethal and detrimental mutation rate, including that causing early embryonic deaths not detected in these studies, is 2–3 times the above values, we have a total mutation rate of .06–.15 per gamete. *Drosophila* data give 10^4 as the ratio of total detrimentals per gamete to single locus rate.¹⁵ Using this value, we obtain a rate of $6\text{--}15 \times 10^{-6}$ detrimental mutations per locus per generation, a value in good agreement with the rates of visible mutations at selected loci.¹⁶ Both types of estimates depend on a number of unverified assumptions and should therefore not be accepted uncritically. However, the agreement between the two

essentially independent methods increases in some measure our confidence in each separately.

Discussion.—Besides the lethal effects considered here, there is mutational damage expressed as anatomical defect and nonlethal disease. Sutter and Tabah also give data on the increase of these in consanguineous marriages, from which it can be estimated that the average person carries about 4–5 genes which, if homozygous, could cause conspicuous abnormality. Such abnormality is likely to reduce reproductive potential. Other mutational damage affecting eventual reproductive potential has less conspicuous expression. For these reasons our figures probably underestimate the over-all mutational damage.

The data of Bemiss and Arner are for American populations a century or more ago, and those of Sutter and Tabah for rural French populations born about thirty-five years ago; yet the values of *A* and *B* are quite similar. Current United States rates for stillbirths (5 months' gestation or longer) are 0.016; for neonatal deaths (under one week), 0.017; and for deaths from 1 week to age 25, about 0.004. This total of .037 is about a third of that in the noninbred populations we have considered. Presumably the value of *B* would also be lower now, since many of the deaths in consanguineous families were known to be from infectious diseases that are now much rarer and for which genetic susceptibility is now less serious. In this connection it is noteworthy that Sutter and Tabah's data show a sharp rise in the incidence of tuberculosis with consanguinity. (A large body of carefully collected consanguinity data may be expected from the Japanese studies of the Atomic Bomb Casualty Commission.)

Provided that genetic selection is not suspended or reversed by improvements in environment, but merely attenuated, the mutational damage at equilibrium will be the same under mild or rigorous selection. However, recent improvements in the environment have taken place very rapidly relative to the time over which present gene frequencies were established. We believe, therefore, that mutational damage and mutation rates are more realistically measured in the data we have considered than from contemporary death rates, the genetic component of which is certainly not at equilibrium. For this reason it is of great importance that comparable studies be carried out on populations under primitive or rigorous conditions while they still exist. Such studies, by affording a comparison with those on modernized populations, would also provide a measure of the relaxation of selection under modern conditions.

Summary.—From studies of the increased mortality in children of consanguineous marriages it is estimated that the average person carries heterozygously the equivalent of 3–5 recessive lethals acting between late fetal and early adult stages. Assuming that the most important effect of detrimental "recessive" mutations in populations that undergo little present inbreeding is through heterozygous damage, and using *Drosophila* data to estimate the amount of this, the frequency of deaths in the populations studied due to the same factors as those causing the additional deaths in consanguineous marriages is estimated as 6–10 per cent. From this we estimate a total mutation rate of .03–.05 per gamete per generation to such genes. Since the total, including those causing early undetected embryonic deaths and detrimental effects after maturity, is probably 2–3 times as high as that accounted for here, the total mutation rate to lethals and detrimentals is estimated as

.06-.15 gamete per generation or, with 10^4 loci per gamete, $6-15 \times 10^{-6}$ per locus.

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¹ G. B. L. Arner, *Columbia Univ. Studies in History, Economics, and Public Law*, 31, No. 3, 1-99, 1908.

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³ H. M. Slatis, *Am. J. Human Genet.*, 6, 412-418, 1954.

⁴ J. Sutter and L. Tabah, *Population*, 7, 249-266, 1952; 8, 511-526, 1953.

⁵ S. M. Bemiss, *Trans. Am. Med. Assoc.*, 11, 319-425, 1858.

⁶ Here and later we are making the simplifying assumption that a pre-existing mutant allele has the same effect on viability as the new mutant. This is justified as a first approximation by the observation that compounds of mutants are often near the average of the mutant homozygotes. This formula is given for only two alleles, but the extension to a larger number is obvious and the linearity in F remains. In applying this to a real population, we assume $F = 0$ for all individuals not known to come from a consanguineous marriage.

⁷ S. Wright, *Am. Naturalist*, 56, 330-338, 1922; *Ann. Eugen.*, 15, 323-354, 1951.

⁸ H. J. Muller, *Am. J. Human Genet.*, 2, 111-176, 1950.

⁹ H. J. Muller, *J. Cellular Comp. Physiol.*, 35, suppl. 1, 205-210, 1950, and unpublished data of Muller and Campbell.

¹⁰ C. Stern, G. Carson, M. Kinst, E. Novitski, and D. Uphoff, *Genetics*, 37, 413-449, 1952.

¹¹ Referred to on p. 41 of Th. Dobzhansky and S. Wright, *Genetics*, 26, 23-51, 1941.

¹² H. J. Muller, *Harvey Lectures, Ser.* 43, pp. 165-229, 1947-1948.

¹³ S. G. Levit, *J. Genetics*, 33, 411-434, 1936.

¹⁴ F. Seto, *Am. Naturalist*, 88, 373-378, 1954.

¹⁵ The "single locus" rate in *Drosophila* may, of course, be the total rate among a series of pseudoalleles. In man there is not only this difficulty but the fact that mutants at different loci with phenotypically similar effects may be counted as mutants at a single locus.

¹⁶ J. Neel and W. Schull, *Human Heredity* (Chicago: Univ. Chicago Press, 1954), p. 144.

TWO LATE-GLACIAL DEPOSITS IN SOUTHERN CONNECTICUT

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One of the recent trends in Pleistocene research has been the study of pollen in deposits that might record late-glacial vegetation changes. To my knowledge there are now only two North American localities that clearly provide such evidence. Not including the type locality in Wisconsin for the Two Creeks interstadial flora,¹ these are George Reserve, Michigan,² and Aroostook County, Maine.³

Evidence from these sections suggests that during the Two Creeks interstadial the central parts of Wisconsin and Michigan were forested with spruce and fir, while at the same time northern Maine had only local stands of spruce. During the ensuing ice advance, eastern Michigan and northern Maine suffered great alterations of climate and may have been characterized by steppe-like vegetation.

The New Evidence.—The late-glacial deposits described briefly here (to be more fully detailed in a later publication) lie less than 20 miles south of the late-Wisconsin drift border described by Flint,⁴ near Middletown, Connecticut. One of these is a kettle, Totoket Bog, earlier analyzed by Deevey,⁵ and the other is Durham Meadows, a formerly lacustrine basin which lies in a small valley tributary to the Connecticut River. The pollen sequences obtained from these are different from