Eisenpress, "Seasonal adjustments by electronic computer methods," *J. Am. Statist. Assoc.* (Dec. 1957), reprinted as *Technical Paper No. 12* by the National Bureau of Economic Research, New York, N.Y.; and (iii) J. Shiskin, "Seasonal adjustments of economic indicators," *Proc. Business and Econ. Sect. Am. Statist. Assoc.* (1957). The project described in this article has been carried on at the Bureau of the Census since the spring of 1954. During the academic year 1956–57, however, I extended and refined the electronic computer program during a year's leave of absence spent at the National Bureau of Economic Research. The tests of the program and the analysis of the relations among different types of economic fluctuations described were both made during this academic year. This work was financed by National Science Foundation and Rockefeller grants. Important contributions to this project have been made by Henry Eisenpress and Geoffrey H. Moore. Michael J. Conlon provided valuable assistance in the preparation of this article.

2. J. Shiskin, "New measures of recession and recovery," in preparation.
3. The method of time series decomposition described here follows the general plan formulated by early analysts of economic time series, particularly Warren M. Persons [see W. M. Persons, "Indices of business conditions," *Rev. Econ. and Statistics* (Jan. 1919); "An index of general business conditions," *ibid.* (Apr. 1919)].
4. For other tests, see the sources cited in (*1*).
5. The results for six additional series are shown in J. Shiskin and H. Eisenpress, "Seasonal adjustments by electronic computer methods," *J. Am. Statist. Assoc.* (Dec. 1957), pp. 432–433, chart 6.

Radiation Dose Rate and Mutation Frequency

The frequency of radiation-induced mutations is not, as the classical view holds, independent of dose rate.

W. L. Russell, Liane Brauch Russell, Elizabeth M. Kelly

It is usually considered to be one of the basic tenets of radiation genetics that variation in radiation intensity—that is, dose rate—does not affect mutation rate. However, the experimental results upon which this conclusion is based were obtained only from certain cell stages, particularly *Drosophila* spermatozoa. The bulk of the radiation dose causing genetic hazards in man will be accumulated not in spermatozoa but in spermatogonia and oocytes. It was therefore of both practical and fundamental importance to question whether mutation rates observed following irradiation of these cell stages would also prove to be independent of radiation intensity.

Two major considerations that prompted such a question, in the face of the general acceptance of the absence of a radiation intensity effect on induced mutation rate, may be outlined. First, there has been increasing evidence that induction of mutation may not be as direct an action as had often been supposed, and that the mutation process in the gene may not be entirely independent of variation in its cellular environment. Consequently, there was room for speculation that even though the mutation process in spermatozoa is apparently independent of dose rate, it might not be so in metabolically active cells like spermatogonia. Second, it was reasoned that even if the actual mutation process in spermatogonia should prove to be, as in spermatozoa, independent of radiation intensity, nevertheless the mutation rate, as measured by mutations transmitted to the offspring, might still be dependent on dose rate, because of cell selection due to killing or other interference with the dynamics of the cycle of the seminiferous epithelium (*1, 2*).

With these two considerations in mind, experiments to determine mutation rates induced by chronic gamma irradiation in spermatogonia in mice were started. The first data from these experiments, and a comparison of them with mutation rates obtained earlier with acute x-irradiation, were presented at the April 1958 annual meeting of the National Academy of Sciences (*1*). They had been submitted earlier for a publication still in press (*2*), and they have also been discussed briefly elsewhere (*3*). The results showed a much lower mutation rate from chronic gamma than from acute x-irradiation. It was pointed out that, without further analysis, it could not be definitely decided whether the difference was attributable to intensity or to quality of radiation (although the latter seemed unlikely in view of the magnitude of the effect), and whether it was the mutation process itself that was involved or some secondary process, such as cell selection.

Since the time of the early reports, the data have been approximately doubled. Also, a number of new experiments, undertaken specifically for the purpose of analyzing the observed effect, have already thrown additional light on the problem. Because of the wide interest in this field, the present interim report has been prepared, bringing tabulation of the spermatogonia results up to date and presenting preliminary results from the new experiments.

Chronic Gamma Irradiation of Spermatogonia

Young mature male mice were exposed, in polystyrene cages of 3.0 to 3.5-millimeter wall thickness (more than adequate for secondary electron equilibrium), to a 5-curie Cs^{137} source. Dose rate was regulated by distance. Exposure was continuous (except for occasional interruptions of a few minutes) until the total dose had been accumulated (*4*). The males were mated to test females (see below) immediately following removal from the radiation field. However, only mutations induced in spermatogonia are considered in this section of this article. Unirradiated males were tested simultaneously with the irradiated.

Mutation rates were determined by the specific locus method. Irradiated and control males are mated to females homozygous for seven autosomal recessive visibles. The offspring are then examined for mutations at the seven loci. Details of the experimental procedure have been described earlier (*5*).

The results from the chronic gamma irradiation experiments are given in

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Table 1. The mutations listed in the table have not yet all been tested for allelism. However, classification by phenotype has proved remarkably reliable in our experience with well over 100 tested mutants at these loci, so there is little likelihood of error.

For comparison with the chronic gamma irradiation data listed in Table 1, a summary is presented in Table 2 of the results of three of our acute x-ray experiments (*2*). The radiation intensity in these experiments was approximately 80 to 90 roentgens per minute.

Results from the chronic gamma and acute x-ray experiments are compared in Fig. 1. All the points for the chronic gamma-ray mutation rate curve are considerably below the acute x-ray curve. However, a comparison of the two sets of results over the whole range of doses cannot be reduced to a simple statistical test of significance because the mutation rate curve following acute x-irradiation shows a clear departure from linearity, already discussed elsewhere (*2, 3, 6*), while the present mutation rate data from chronic gamma irradiation show no evidence of a similar departure. Three statistical tests have been made (*7*) which attempt to avoid this difficulty in different ways.

In view of the possible special reasons for the departure of the acute x-ray curve from linearity (the drop in the mutation rate at the 1000-roentgen dose being attributed to cell selection), one test of the significance of the difference between the chronic gamma and acute x-radiation induced mutation rates was made with the 1000-roentgen x-ray point excluded. The two sets of data, with a combined control point, were fitted simultaneously to two straight lines by the method of least squares, with weights based on the Poisson assumption. The ratio of the slopes is 4.1 (95-percent confidence interval 2.36, 12.5), and the slopes differ significantly ($P < 1 \times 10^{-9}$). A similar test, but one that excludes both the acute x-ray 1000-roentgen point and the chronic gamma-ray 861-roentgen point, also yields a significant difference ($P < 1 \times 10^{-7}$). The third statistical test was made between just two points. In view of the lack of data at closely comparable doses in the lower part of the dose range, and because of the presumed complexity at the 1000-roentgen x-ray point, the two points that seemed to offer the most meaningful single comparison were the 600-roentgen point for acute x-rays and the 516-roentgen point for chronic gamma rays. A test of the significance of the difference between the mutation rates per roentgen at these two points gave $P = 0.0008$ for a one-tailed test (*8*).

Table 1. Mutations at specific loci induced in spermatogonia of mice by chronic gamma irradiation.

Dose (r)	Intensity (r/wk)	Offspring (No.)	Mutations at 7 loci (No.)	Mean No. of mutations per locus, per gamete ($\times 10^5$)
0		105,403	8	1.08
86	10	48,500	6	1.77
516	90	20,752	4	2.75
861	90	20,993	9	6.12

Table 2. Mutations at specific loci induced in spermatogonia of mice by acute x-irradiation.

Dose (r)	Offspring (No.)	Mutations at 7 loci (No.)	Mean No. of mutations per locus, per gamete ($\times 10^5$)
0	42,833	1	0.33
300	40,408	25	8.85
0	106,408	6	0.81
600	119,326	111	13.29
0	33,972	2	0.84
1000	31,815	23	10.33

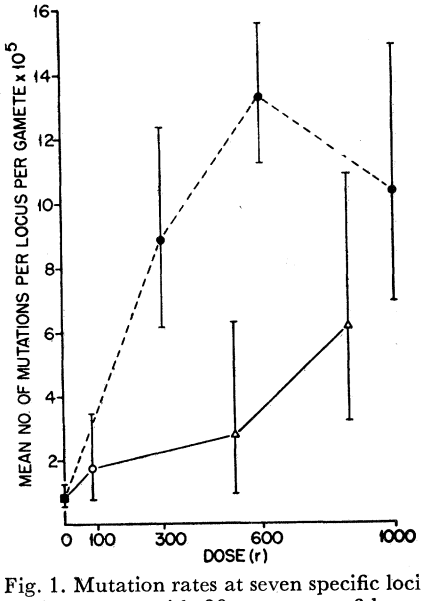

Fig. 1. Mutation rates at seven specific loci in the mouse, with 90-percent confidence intervals. Solid circles represent results with acute x-rays (80 to 90 r/min). Open points represent chronic gamma-ray results (triangles, 90 r/wk); (circle, 10 r/wk). The point for zero dose represents the sum of all controls.

In the statistical tests, the fitted curves show no evidence of departure from linearity. The question of whether or not the data may be expected to be truly linear is discussed below. The actual ratio of effectiveness of chronic gamma and acute x-irradiation found may, of course, be valid only for the particular combination of doses and intensities tested. The important point is that the data now available adequately confirm the earlier report (*1–3*) that chronic gamma radiation is significantly less effective than acute x-radiation in inducing specific locus mutations in spermatogonia.

The conclusions of the preceding paragraphs—that chronic gamma irradiation of mouse spermatogonia is mutagenically less effective than acute x-irradiation—is in sharp contrast to the findings for *Drosophila* spermatozoa, reviewed by Muller (*9*), which have heretofore been considered to have general applicability and have entered into the basic concepts of radiation genetics.

It is, therefore, of great importance to attempt to determine what factors are responsible for the present result. For this reason, a number of experiments, designed to throw light on this question, have been initiated. In Table 3, preliminary results of these new experiments, as well as older findings already reported elsewhere, are compared with the present data.

Intensity versus Quality

The difference in mutation rate between spermatogonia subjected to chronic gamma irradiation and those subjected to acute x-irradiation could be due to differences either in quality or in intensity of radiation. In order to differentiate between these two factors, the effect of a change in quality alone has been investigated in three separate comparisons (see Table 3). No appreciable differences were found in the effectiveness of acute gamma rays (from Co^{60}), on the one hand, and acute x-rays, on the other, in inducing dominant lethal mutations in spermatozoa, specific locus mutations in spermatozoa and other post-spermatogonial stages, or specific locus mutations in spermatogonia. (It appears safe to assume the same result also for oocytes, for which no direct quality com-

Table 3. Semiquantitative comparison of mutation rates presented in this article with those obtained earlier and with preliminary results from experiments in progress. Each plus symbol in the table stands for a mutation rate of approximately 5×10^{-8} per roentgen, per locus. The check marks represent arbitrary values that are valid for comparative purposes among the dominant lethal results. They cannot be quantitatively compared with the specific locus mutation rates.

Gametogenic stage irradiated	Genetic effect measured	Type of irradiation		
		Chronic Gamma (Cs^{137})	Acute	
			Gamma (Co^{60})	X-ray
Postspermatogonia	Dominant lethals*†	√ √ √	√ √ √	√ √ √
Postspermatogonia	Specific locus mutations	(++++++)‡	++++++++++§	++++++++++‖
Spermatogonia	Specific locus mutations	+	++++§	++++
Oocytes	Specific locus mutations	+¶		+++++++**

* Paper in preparation.
† Chronic and acute gamma rays give approximately equal rates, although comparison is not exact because of difficulty in matching particular postspermatogonial stages irradiated. In the comparison of acute gamma with acute x-rays, the former were found slightly less effective.
‡ Value is based on only 1 mutation in 1613 young, so the mutation rate is not yet reliable.
§ Value based on 4 mutations.
‖ From Russell *et al.* (*17*).
¶ From Russell *et al.* (*12*); see also Carter (*13*) for chronic Co^{60} gamma data.
** From Russell *et al.* (*18*).

parison was made.) These results show that difference in the quality (linear energy transfer) of the gamma rays and x-rays tested, while it may account for a small part, cannot account for the bulk of the difference between the chronic gamma and acute x-ray mutation rate results. It can be concluded that most of the difference must be due to intensity of radiation.

Intensity and Gametogenic Stage

The results summarized in Table 3 show that radiation intensity effects were found only for spermatogonia and oocytes. In the experiments with postspermatogonial stages, radiation intensity had no appreciable effect on the yield of genetic changes. This conclusion can be drawn with near certainty for dominant lethals. The specific locus data, from experiments still in progress, are not yet extensive, but, as far as they go, they are not in disagreement with the dominant lethal result. In both cases, the stages irradiated were spermatozoa and spermatids, with the bulk of the data from the former. It may thus be concluded that dose rate does not influence the frequency of genetic changes produced by irradiation in mouse spermatozoa, but conclusions regarding spermatids and spermatocytes will have to await further work. The spermatozoa results are in agreement with the findings for *Drosophila* spermatozoa. Thus, the classic finding of intensity independence is supported for spermatozoa (*10*). The explanation for the new phenomenon of intensity dependence resides in gametogenic stage.

Mutation Process versus Cell Selection

The intensity effect in spermatogonia might have been due to secondary causes —that is, selection as a result of cell killing or other interference with the dynamics of the cycle of the seminiferous epithelium, as stated above. This was put forward as one plausible, but not favored, hypothesis in the first detailed publication of the data (*2*). This hypothesis has now been deliberately tested by new experiments on females. Since oogonia are not present in the adult ovary (*11*), and since the completion of the first meiotic division only just precedes ovulation, radiation genetic experiments on adult females deal exclusively with primary oocytes, and the bulk of these are in the uniform dictyate state. Results already reported (*2, 12*) showed that chronic gamma irradiation of oocytes gave mutation rates lower than those from acute x-irradiation of spermatogonia. The new results (Table 3) indicate that acute irradiation of oocytes is at least as effective as acute irradiation of spermatogonia.

In the light of this finding of a dose-rate effect for oocytes as well as for spermatogonia, the hypothesis that the intensity effect on mutation rate is due to cell selection appears to be less tenable. Since oocytes are nonmitotic, since the stages irradiated show no obvious variability, and since, in our chronic irradiation experiment, the continued fertility of the females provides no evidence of extensive killing, selective or otherwise, of the oocytes, it seems highly unlikely that the difference beween the mutation rates following chronic and acute irradiation of oocytes can be attributed to any secondary mechanism similar to that put forward as a possible one for spermatogonia. Of course, this mechanism might still be postulated as playing a role in the spermatogonia results, but it is simpler to assume that the explanation for the results in oocytes—namely, that the intensity effect is on the mutation process itself—also applies to spermatogonia.

It should be noted that, at each dose rate tested, there is at present no evidence of marked difference between oocytes and spermatogonia in sensitivity to mutation induction. Therefore, the interpretation by Carter (*13*), who also found a low mutation rate with chronic gamma irradiation of oocytes, and who thought it most likely that this was attributable to sex, is not upheld. His emphasis on the consequence of his interpretation—namely, that only a small part of the genetic hazard from medical irradiation would come from exposure of females—must now be discounted.

Relation to the Linearity Concept

The various results discussed in the three preceding sections and summarized in Table 3 have determined which among the possible factors are the ones responsible for the lower mutation rate from chronic gamma irradiation. It turns out that these are also the more interesting factors. Two of these are radiation intensity, rather than quality; and the mutation process itself, rather than cell selection. Since the finding of an intensity effect on the mutation process was unexpected, the field is now open for new hypotheses about the nature of this process. Such hypotheses are aided, or at least delimited, by the finding of a third factor—namely, that the intensity effect occurs in spermatogonia and oocytes, but apparently not in spermatozoa. Thus, the mechanism for this effect may be found among the characteristics by which the highly specialized spermatozoa differ from spermatogonia and oocytes.

Speculation concerning the nature of the mutation process has a direct bearing on the fundamental problem of what the mutation rates are now likely to be at other doses and intensities. One specific question is already being debated—namely, whether or not the finding of an intensity effect in spermatogonia and oocytes is strong indication that a threshold dose will be found for mutation induction in these cells. This possibility has obvious and vital importance to the problem of genetic hazards.

A strong argument that has long been advanced against the threshold concept is the likelihood that a single direct hit (ion or ion cluster) on such a small target as a gene must sometimes be adequate to cause mutation. This hypothesis has not only seemed plausible on physical grounds but has also been supported by the mutation rate data for *Drosophila* spermatozoa and for other material where an intensity independence or a linear relation with dose has been found (*9*). The new data from mouse spermatozoa provide additional support. If the intensity effect reported here for mouse spermatogonia and oocytes is taken as evidence for a threshold effect for all mutations induced in these cells, then this necessarily implies that all mutations in spermatogonia and oocytes are induced by a process different from that which has long been, and still can be, assumed for spermatozoa. This may be true, but it would certainly be incautious to jump to this conclusion. In fact, it seems quite plausible to assume that spermatogonia and oocytes may not be completely different from spermatozoa—in other words, that at least a portion of the mutations in them may be induced by a single-hit process.

To make the consequences of this hypothesis easily understandable, they will be presented in terms of a specific model. Thus, it can be postulated that there are two kinds of mutation which, for simplicity in the following discussion, will be called "reparable" and "irreparable." (They could, alternatively, and perhaps more realistically, be looked upon as "preventable" and "not preventable.") It can be further assumed that in spermatogonia and oocytes there is repair of the reparable mutations at the low radiation-intensity (chronic) level so far tested. Such repair is assumed to be impossible, or less probable, because of radiation damage to the repair process, at high radiation intensities (acute) in spermatogonia and oocytes. Repair is also assumed to be impossible at all intensities in spermatozoa, perhaps because of some property—for example, a metabolic activity—lacking in them that is present in spermatogonia and oocytes.

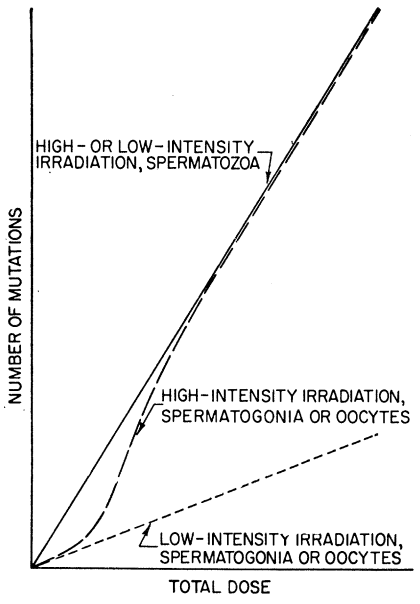

Fig. 2. Theoretical dose curves constructed on the basis of the hypothesis (see text) that "repair" of some mutations is possible in spermatogonia and oocytes but not in spermatozoa.

Such a hypothesis could lead to a set of curves something like that shown in Fig. 2. The straight line for chronic irradiation of spermatogonia and oocytes is assumed to be the single-hit curve for irreparable mutation, all reparable ones having been repaired. A steeper straight line is shown for spermatozoa, where it is assumed that none of the reparable mutations are repaired and that both these and the irreparable ones follow a single-hit relation with dose, regardless of intensity. It follows logically that, as is shown in the third curve, acute irradiation of spermatogonia and oocytes would, at total doses low enough to permit repair, duplicate the curve for chronic irradiation, but that, at higher doses, when repair fails, the curve would shift over to a new position approaching that for spermatozoa. (Actually, the curve for observed mutations in spermatozoa is much steeper than the curve for acute irradiation of spermatogonia. The reasons for this, one of which is probably a large chromosomal aberration component of the mutations in spermatozoa (*14*), are assumed to be irrelevant to the present argument. In Fig. 2, the curve for spermatozoa, as well as the curve for oocytes, may be looked upon as being appropriately adjusted to eliminate the irrelevant factors and to provide an uncomplicated comparison for radiation intensity only.)

No importance is attached to the particular details chosen to make this type of model easily understandable. Thus, "reparable" and "irreparable" need not imply qualitatively different mutational sites. Only one kind of site is necessary if, for example, it is assumed that there is a time lag for the completion of the mutation process and (even with the repair process intact) a probability of less than unity that repair could occur before this completion. Also, the term *repair* is not necessarily restricted to mean the reversal of a damaged gene to normal. In fact, as was mentioned earlier, the term *preventable* might be substituted in place of *reparable*. Prevention could occur at any stage in the mutation process, even at its initiation when there might be diversion, by a "lightning-rod" effect, of ions that might otherwise have caused mutation.

Whether or not the proposed hypothesis is favored, it demonstrates clearly that the discovery of an intensity effect does not necessarily imply that all induced mutations in spermatogonia and oocytes must follow a threshold response. Of course the hypothesis does involve a threshold concept, but it applies to only a portion of the mutations. As demonstrated, the theoretical consequence for chronic irradiation of spermatogonia and oocytes, in this particular model, is a linear relation between mutation rate and dose, even down to the lowest doses, in spite of a lower mutation rate than with acute irradiation.

Other plausible models can, of course, be constructed. Experiments now under way with various intensities of radiation and with fractionated doses will undoubtedly narrow down the possibilities. It should be noted, however, that the range of intensities already tested is tremendous—namely, 10,000-fold (100,000-fold at one point). The fact that this has yielded only a fourfold difference in mutation rate certainly raises the question of whether a further decrease in intensity would be likely to give a further drop in mutation rate. The mutation rate at the lowest intensity tested—10 roentgens per week—and the rate reported by Carter *et al.* (*15*) for a similar intensity still have such wide confidence intervals that they are not particularly informative in a comparison with the results from the 90-roentgen-per-week intensity.

Human Hazards

Caution must be exercised against reaching dangerous conclusions from the present results. Thus, as has been emphasized, it is not safe to conclude that the data imply a threshold dose for all mutations in spermatogonia and oocytes. There might not even be any further reduction in mutation rate with further decrease in intensity. Furthermore, it should not be forgotten that even the lower mutation rates obtained with the present intensity levels are still appreciable and at least as high as *Drosophila* rates for acute irradiation. However, from the results as they stand—results that apply to the germ-cell stages (spermatogonia and oocytes) that are important in appraising human hazards—it does seem safe to conclude that, with at least some intensities of radiation, the genetic damage would not be as great as that estimated from the mutation rates obtained with acute irradiation.

Summary

New data have clearly confirmed the earlier finding that specific locus mutation rates obtained with chronic gamma irradiation of spermatogonia are lower than those obtained with acute x-rays. Since this result is in contrast to classical findings for *Drosophila* spermatozoa, and apparently contradicts one of the basic tenets of radiation genetics, it was important to determine what factors were responsible for it.

Experiments undertaken for this purpose reveal the following: (i) the lower mutation frequency is due mainly to difference in dose rate of radiation, rather than quality; (ii) a dose-rate effect is not obtained in experiments with mouse spermatozoa, confirming classical findings for spermatozoa, and indicating that the explanation for intensity dependence in spermatogonia resides in some characteristic of gametogenic stage; and (iii) a dose-rate effect is found not only in spermatogonia but also in oocytes, where cell selection is improbable, indicating that the radiation intensity effect is on the mutation process itself.

A threshold response for all mutations in spermatogonia and oocytes is not a necessary consequence of the findings. Plausible hypotheses consistent with the present results can lead to other predictions.

From a practical point of view, the results indicate that the genetic hazards, at least under some radiation conditions, may not be as great as those estimated from the mutation rates obtained with acute irradiation. However, it should not be forgotten that even the lower mutation rates obtained with the present intensity levels are still appreciable (*16*).

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Groningen Radiocarbon Dates III

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The present series of radiocarbon dates obtained at the University of Groningen covers the period from March 1956 to August 1957. The first two lists (*1, 2*) will be referred to as I and II. Characteristics of the counters and descriptions of the technical procedures, statement of errors, and so forth, were given in list II.

Samples numbered between 600 and 900 were measured in the small counter; samples numbered between 500 and 600 and between 1200 and 1500 were measured in the large counter; and samples between 900 and 1200 and above 1500 were measured in the medium-sized counter.

Measurements on the radioactivity of shells and snails from different environments during the last 4 years have been published separately (*3*), since they are not given "dates." One of the conclusions drawn from these measurements is that the amount of carbon-14 in the atmosphere increased by about 5 percent between the end of 1953 and the spring of 1957. This increase is due to the explosions of atomic bombs. A group of Würm interstadial samples has been published separately (*4*), since they require a more detailed discussion. The results can be summarized briefly as follows: About 26,000 years ago a fairly short interstadial (or warmer oscillation) occurred, which produced the Paudorf fossil soil. The first Würm interstadial occurred at about 50,000 years ago, no indication of a warmer period between 50,000 and 26,000 years ago being found up to now.

The remaining dates are given here in four groups (Tables 1–4). The first group consists of a series of geological samples from northwestern Europe; it

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