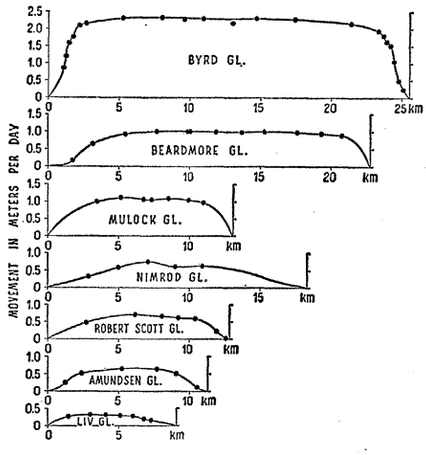

Fig. 2. Observed horizontal displacement expressed as rate of movement.

ice and topped with a red flag. Severe crevassing on Mulock and Byrd glaciers made it necessary to lower a man to the ice by means of the sea rescue winch of a hovering helicopter. Conspicuous crevasses and seracs were sometimes used instead of specially erected survey markers. The position of each marker was established by conventional triangulation, by using a theodolite at each end of a base line measured on mountains overlooking the glacier. A second set of measurements on a subsequent visit to the glacier gave the horizontal component of ice movement during the intervening period. In addition, the movement of Byrd Glacier was determined by aerial triangulation from two sets of vertical air photographs taken 11 months apart (*1*). The thickness of the glaciers is unknown. Ice discharge can therefore be reported only in units of glacier surface area.

The results are shown in Fig. 2. The accuracy varies, depending chiefly on the length of the period available for observing the glacier (Table 1). The most significant discovery is the overwhelming contribution of Byrd Glacier, which adds 19 km^3/year to the ice shelf, equivalent to the combined contribution of the other six glaciers. Byrd Glacier is so little known as a geographical feature that it was named only in 1960, whereas some of the other glaciers were named and mapped 50 years ago. Though a few ice streams (*2*) may move faster (*3*), Byrd Glacier is now the fastest known valley glacier in Antarctica. Several large Greenland glaciers move faster (*4*), but probably only Jakobshavns Isbrae discharges more ice down a single valley (*5*). The most recently published contours of the neighboring ice plateau (*6*) indicate a drainage basin centered on Byrd Glacier and extending several hundred kilometers inland. The drainage basins of the other major glaciers are relatively small. Though the total ice discharge of the seven glaciers studied—38 km^3/year—is greater than earlier observations (*7*) led me to expect, it is probably small compared with the discharge from the unexplored southeastern borderlands of the ice shelf. While flying down the eastern margin on 21 October 1961, I saw what is virtually a single, 200-km-wide ice stream south of latitude 83°S.

Table 1. Observed horizontal displacement and probable error (P.E.) of measurement in middle of glacier.

Glacier	Observing period (days)	Total movement (m)	P.E. (%)
Byrd	348	803.3	± 1.0
Beardmore	332	331.5	± 0.4
Mulock	446	473.9	± 0.8
Nimrod	296	183.5	± 0.8
Robert Scott	7	4.9	± 3.4
Amundsen	5	3.2	± 4.8
Liv	5	1.5	± 8.4

Measurements across Beardmore Glacier over a 14-day period in summer (December) gave the same rates of movement as for the long period. A 2-percent difference would have been measurable. The observations suggest that in high latitudes where there is no surface melting, there may be no seasonal variations in the rate of glacier movement (*8*).

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1. The photogrammetric computation was made by A. J. Brandenberger, R. B. Forrest, S. K. Ghosh, M. W. Hindman, and R. K. H. Adler, of Ohio State University.
2. Part of an ice sheet in which the ice flows more rapidly and not necessarily in the same direction as the surrounding ice. A valley glacier, in contrast, flows between rock walls.
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8. A paper is in preparation in which these measurements are discussed in detail, together with observations of ice surface strain rate, ice regime, ice temperature, and glacier morphology. I thank T. E. Taylor, A. S. Rundle, D. G. Darby, J. Tuck, and O. Liestøl for their untiring help in the field; and E. Dorrer for help in office computation.
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Reduced Incidence of Persistent Chromosome Aberrations in Mice Irradiated at Low Dose Rates

Abstract. *A marked difference was observed between the effectiveness of high and low dose rates of ionizing radiation in producing persistent chromosome aberrations in the marrow cells of mice. Clones of cells with chromosome abnormalities were present in the marrow of all the mice previously exposed to single or fractionated doses of x-rays given at a rate of 30 rad/min. The frequency of chromosome aberrations in these mice varied from 14 to 72 percent of the cells examined. By contrast, none of the mice exposed to continuous gamma radiation at a low dose rate (1.45 rad/hour) showed definite clones of abnormal marrow cells, and the frequency of persistent chromosome aberrations varied from zero to 8 percent in this group.*

The importance of dose rate in the delayed and late effects of ionizing radiation has been demonstrated, both with respect to genetic effects (mutations) in mice (*1*, *2*), and in studies of life span (*3*). However, since there are no results available of observations on persistent radiation-induced chromosome abnormalities relative to dose rate, we were interested in obtaining such data, and in correlating them with late pathological changes after irradiation at high and low dose rates.

We studied the chromosomes of bone marrow cells from three groups of female LAf$_1$ mice irradiated at 2 to 3 months of age. Group I received a single dose of 500 rad of x-rays, at a rate of 30 rad/min as measured in air. Group II received fractionated doses of x-rays, 100 rad given on 9 of 11 successive days, for a total dose of 900 rad, at a rate of 30 rad/min. Group III received Co^{60} γ-radiation, given continuously at a rate of 1.45 rad/hr, the dose totaling either 935 rad (*a*) or 926 rad (*b*) (see Table 1).

The radiation factors for the x-radiation were: 250 kv; 15 ma; half-value layer, 1.5 mm Cu; filter, 0.5 mm Cu

plus 1 mm Al; target to skin distance, 100 cm. For the Co^{60} irradiation, mice were placed in Lucite cages, which were placed around a 10 curie source (described previously, *4*). The dose rate was measured at the midpoint of the cage, using 5-minute readings with a 250-mr chamber. The mean value for dose rate was 1.45 rad/hr as of 4 August 1961. Appropriate corrections were made for decay of the source at the time of exposure. Since the duration of exposure was approximately one month, it was necessary to remove the mice from the source periodically for brief periods, in order to feed and water them, and to clean the cages. The total "off-time" during the entire exposure was 7 hours.

At various times from 4 to 76 weeks after irradiation, air-dried chromosome preparations were made from the bone marrow of each mouse according to techniques described previously (*5*). Fifty good metaphases from each animal were examined. At the time of the cytological examinations, the examiner did not know which slides were from high dose rate animals and which were from low dose rate animals.

Chromosome changes observed in the irradiated mice are given in Table 1. Because of the uniform morphology of mouse chromosomes, all having terminal centromeres, the only aberrations recognizable are those producing aneuploidy, those with a marker chromosome obviously longer or shorter than the normal complement, or those with a centromere which is not terminal (*6*). The most commonly observed change was an apparent reciprocal translocation resulting in two markers, an abnormally long chromosome and a "minute" (Fig. 1). It was, of course, impossible to determine if morphologically similar changes in different animals, or even in different cells, involved identical chromosomes.

In mice with a high frequency of chromosome aberrations, only one or two types of aberrations were generally present in any given animal. Thus, the abnormal cells appeared to be largely in the form of "clones" or "stemlines" derived from only one or two radiation-damaged precursors (*7*). For the purpose of tabulation, any group of five or more cells having the same morphological chromosome change was considered to represent a clone (that is, 10 percent or more of the cells examined).

The data indicate that clones of cells with chromosome abnormalities were present in the bone marrow of all the mice in group I as well as in group II. The frequency of cells with aberrations varied from 14 percent to 72 percent, without any obvious effect of the length of time between irradiation and killing (12 to 76 weeks). It may be significant that the two mice in group I which were examined 72 weeks after exposure (the longest period in this group) showed the most profound chromosome changes. Abnormal cells constituted 46 percent and 72 percent, respectively, of their marrows, and those included the only two aneuploid clones observed, one with 41 chromosomes and one with 39 chromosomes. The possibility of incipient leukemia in these two mice might be suggested by the chromosome findings (*6, 8*), but neither these nor any other mice in the present study showed clinical or hematological evidence of the disease.

Definite clones of abnormal marrow cells were not observed in any of the mice in group III. Frequency of persistent chromosome aberrations varied from zero to 8 percent in this group, and in no animal did more than 6 percent of the cells show the same chromosome change. The only unstable changes observed in any of the groups were occasional chromatid breaks, present in less than 2 percent of the cells from all groups. Other unstable aberrations, such as acentric fragments, dicentrics, and rings, were not found, undoubtedly because they were eliminated within one or two cell divisions after irradiation (*6*).

Our data correlate well with the observations of Russell *et al.* (*1, 2*) who found that, following exposure to x-radiation at low dose rates (90 r/wk), the incidence of specific locus mutations was one-fourth that in mice exposed to x-rays at high dose rates (80 to 90 r/min). The present findings would appear to be, on the chromosome level, the counterpart of these observations. Russell *et al.* have also provided evidence to show that the effect of dose rate on mutation frequency "is an intracellular one and not a consequence of cell selection" (*2*). The rarity of persistent chromosome abnormalities after radiation at a low

Table 1. Chromosome changes in marrow cells of mice irradiated at different dose rates.

Animal No.	Interval after irradiation (wk)	Chromosome aberrations (No./50 cells)				Total aberrations (%)	Clonal aberrations (%)
		Minute and abnormally long	Minute	Abnormally long	Other		
Group I. Single dose of x-rays totaling 500 rad at a rate of 30 rad/min							
1	12	22			1	46	44
2	12	9				18	18
3	52	7	4	2		26	14
4	52		3	15		36	30
5	52	7				14	14
6	52	9	8			34	18, 16
7	52	5	3	1		18	10
8	72		5		18*	46	10, 36
9	72	10	1	2	23†	72	20, 46
Group II. Fractionated doses of x-rays totaling 900 rad (100 rad × 9 daily) at a rate of 30 rad/min							
10	76			21		42	42
11	76		6	1		14	12
12	76	5	6	2		26	10, 12
Group III(a). Single dose of Co^{60} γ-radiation totaling 935 rad at a rate of 1.45 rad/hr							
13	1					0	0
14	1		3			6	0
15	1			1		2	0
16	1		1			2	0
17	1			1		2	0
18	1		1			2	0
19	4					0	0
20	4	2	1			6	0
21	5	3		1		8	0
22	5			1		2	0
Group III(b). Single dose of Co^{60} γ-radiation totaling 926 rad at a rate of 1.45 rad/hr							
23	48					0	0
24	48					0	0
25	48		1			2	0
26	48				1	2	0
27	48		1			2	0
28	48					0	0
29	48		1			2	0

* 41 chromosomes (one abnormally long). † 39 chromosomes (one metacentric).

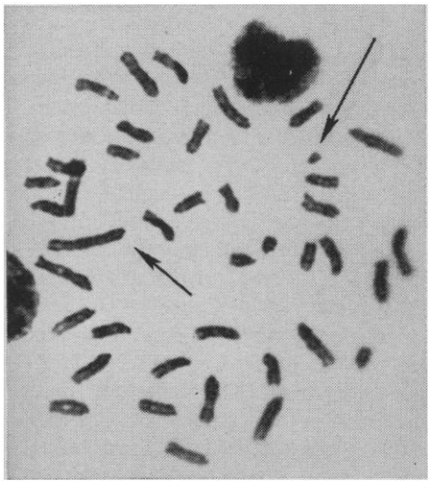

Fig. 1. Metaphase from bone marrow of LAf_1 mouse x-irradiated (500 rad, dose rate 30 rad/min) 72 weeks previously. Note the abnormally large and minute chromosomes indicated by arrows. Twenty percent of the marrow cells examined were of this type.

dose rate could similarly be due to an intracellular recovery process, perhaps involving restitution and repair of radiation-induced chromosome breaks comparable to that observed by Wolff (*9*) in plant cells. This recovery process would presumably be analogous to that described by Elkind and Sutton (*10*) in studies of the viability of hamster cells in tissue culture after fractionated radiation. In their work, however, irradiation was completed within one mitotic cycle, while in our experiments with low dose rates, numerous generations of cells were involved. Hence, it is possible that additional recovery mechanisms, both intracellular and extracellular, might operate in our system.

It is also possible that both the high and low dose rates may have produced the same amount of non-recoverable chromosome damage, and that cell selection rather than intracellular recovery mechanisms are responsible for the observed difference in persistent chromosome changes. The extensive destruction of marrow cells which occurs at the high dose rate, producing an aplastic marrow for a brief period, could provide the opportunity and "space" for a few stem cells, with radiation-induced chromosome changes conferring a slight growth advantage, to repopulate the marrow with recognizable clones. Cells with similar changes produced by low dose rate radiation, which does not deplete the marrow, might not have a sufficient selective advantage to permit them to overgrow an already populated marrow and, hence, they would continue to survive as only a small proportion of the marrow cells. The continued persistence of large clones of abnormal cells, without concomitant hematological disorders, in animals given a high dose rate indicates that radiation-induced chromosome abnormalities do not necessarily confer either a marked selective advantage or disadvantage on cells bearing them (*6*).

Although it might be expected that Co^{60} γ-radiation would produce fewer breaks per rad than x-radiation on the basis of the well-known difference in the relative biological effectiveness between these radiations, this would not, by any means, explain the marked difference between the effects of low and high dose rates observed in the present study.

The present study permits further speculation on the relationship of radiation-induced chromosome changes to leukemogenesis. Data from both mice and humans indicate that there is no uniform correlation between demonstrable chromosome changes produced by radiation at high dose rates and the subsequent development of leukemia (*6*). Whether the incidence of radiation-induced leukemia in mice exposed to γ-radiation at a low dose rate would parallel the present chromosome findings remains to be determined. Single acute exposure at a high dose rate, however, may be lethal for a larger number of marrow stem cells than is radiation given at a low dose rate, thus possibly eliminating many potentially leukemic cells from the "pool"—the so-called "therapeutic" effect of high radiation dose on leukemia incidence (*11*). If leukemogenesis is related to the total number of point mutations produced, it is conceivable that the frequency of point mutations could be the same in all three groups in the present study and that the incidence of leukemia might actually be highest in the group exposed to radiation at the low dose rate, as a result of accumulation of such radiation-induced point mutations with a minimum of cell killing. Observations on leukemia incidence in mice exposed to low dose rate γ-radiation are now being made.

The relationship between chromosome aberrations and the occurrence of other late pathological effects of irradiation, such as solid-tissue tumors, nephrosclerosis, and other lesions resulting in shortened life span, is even less clear. Data are not available on chromosomes from the organs involved, and extrapolation to these tissues from the effects of dose rate on bone marrow cells might well be erroneous, because of the great differences in mitotic rates and cell turnover. It would be of interest to assess the importance of chromosome damage in the production of late radiation effects in organs such as the liver and kidney (*12*).

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Reserpine: Its Effect on Silver-Stained Structures of the Heart

Abstract. *The administration of reserpine to dogs in doses sufficient to deplete myocardial catecholamines resulted in alterations in the affinity of the heart for silver stain. Most noticeably affected was the "perimysial plexus."*

Silver-stained preparations of heart tissue from many species reveal a plentiful mesh of fine and course tortuous structures which appear to envelope cardiac muscle cells. Some evidence indicates that these structures constitute