beetles are consistently associated with specific fungi and feed on them regularly but not exclusively. Thus, young adults of *Ips avulsus, I. calligraphus, I. grandicollis,* and *I. pini* were observed feeding on conidia and perithecia of *Ceratocystis ips* in North America; their intestinal tracts contained perithecial fragments and viable ascospores and conidia. *Ips grandicollis, I. pini,* and *I. oregonensis* were also observed regularly feeding in nature on sporodochia of *Tuberculariella ips,* which is invariably found in pupal cells and is morphologically very closely related to—perhaps congeneric with—*T. ambrosiae,* the primary ambrosia fungus of *Platypus wilsoni* (*7*) and *Trypodendron* spp. *Ips pini* and *I. oregonensis* are often smeared outside with the ascospores of *C. ips,* which bud in the intersegmental folds of the beetles, the resulting yeast-like growth resembling the ambrosia propagules in mycangia of ambrosia beetles.

Trichosporium tingens and *T. tingens* var. *macrosporum,* symbiotically associated respectively with the bark beetles *Tomicus minor* and *Ips acuminatus* in Sweden, are quite similar to some ambrosia fungi, particularly *Monilia ferruginea,* the primary ambrosia fungus of *Trypodendron lineatum* and other *Trypodendron* spp. in Canada, the United States, and Sweden. *Trichosporium* spp., as well as bark, serve as foods for these beetles, and hibernating *Tomicus minor* carries inoculum of *T. tingens, Ceratocystis cana,* and some yeasts "in a primitive manner in its median suture and in the lateral folds of the elytra" (*3*). Inoculum of *T. tingens* var. *macrosporum,* on the other hand, is contained in specialized pockets in the oral cavity of *Ips acuminatus;* such structures corespond to oral mycangia of some *Xyleborus* spp., which are true ambrosia beetles (*3*).

I have isolated 51 fungi from inoculum surviving as yeast cells or as ambrosia propagules on various parts of many Platypodidae and Scolytidae from India (unpublished). Such propagules are usually indistinguishable from each other, yet when plated they yield diverse fungus flora and many do not sporulate.

I therefore postulate that fungi that may have been fortuitously carried at one time into the tunnels of bark- and wood-inhabiting beetles today live symbiotically with ambrosia beetles in a truly mutualistic relation. Initially the fungi established themselves beneath the bark, ramifying into the frass-packed tunnels. The fungi were occasionally consumed along with bark, as with *Ips, Tomicus,* and *Dendroctonus* spp. During the course of evolution some fungi perhaps invaded wood and made it possible for the beetles to penetrate weakened xylem while feeding on fungi. Eventually some beetles abandoned the eating of bark and wood, became wholely adapted to mycetophagy, and developed mutualism with ambrosia fungi.

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5. Only 6 of 15 beetles in tunnels with *Endomycopsis fasciculata* and 4 of 15 with *Ceratocystis minor* maintained brood for a period long enough to yield adults in 1962; these were individuals that had not yet laid eggs in tunnels from which they were initially removed. Most of the mothers that did not initiate or maintain a brood to maturity had left eggs and larvae in the tunnels from which they were originally transferred; presumably they were already considerably worn out. In 1963, when only young beetles were used, mortality in their new tunnels was much lower than in 1962.
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Chromosome Aberrations: Increased Incidence in Bone Marrow of Continuously Irradiated Rats

Abstract. *The number of chromosomal aberrations in the bone marrow of continuously irradiated rats temporarily decreases after exposure, and then increases as the total dose and time after irradiation increase. This increase in aberrations is greater than that expected as a result of age alone.*

Chromosomal abnormalities which may be a rough index of mutations (*1–3*) and, in accordance with the somatic mutation theory, can be related to aging (*4*), appear to be a suitable criterion for determining late radiation injury. While radiation-induced aberrations slowly decrease in tissues whose cells rarely divide (*2–5*), most aberrations in tissues with continually dividing cells, such as the bone marrow, are eliminated soon after irradiation (*6*). Nevertheless, one type of aberration, the polyploid forms, persists in bone marrow or in peripheral leucocytes long after irradition (*7*).

To determine the hematologic changes in rats during continuous irradiation, male albino rats (Wistar, 7 to 12 months) were irradiated with various dose rates of Co^{60} γ-rays for 23 to 23.5 hours per day. Abnormal and normal cells in anaphase or telophase were scored at 5 days, 6 months, and 18 months after cessation of the irradiation. Unfortunately, there were not enough rats to form groups irradiated under the same conditions of dose and time (Table 1, groups II, III, and IV). Later, the incidence of aberrations during continuous irradiation (Table 1, group I) and the frequency of spontaneous aberrations, in relation to age, in nonirradiated rats were studied. Bone marrow was taken from the femur which had been fixed in acetic alcohol, and squash preparations were stained by the Feulgen method. Bridges and acentric fragments (*4*) in anaphase and telophase mitotic figures were scored and expressed as the percentage of total anaphase and telophase figures found in 10,000 marrow elements.

The percentage of chromosomal aberrations decreases within 5 days after irradiation (group II) and is about one-fourth that found in rats during exposure to an approximately equal total dose of irradiation (group I). Within 6 months after exposure (group III) the percentage of aberrations is more than twice that 5 days after exposure

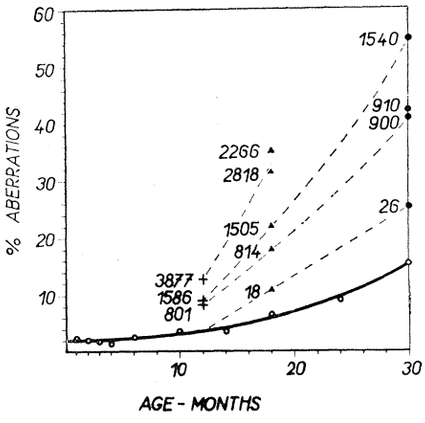

Fig. 1. Solid Line: Spontaneous chromosome aberrations in normal nonirradiated rats as a function of age. Each point represents mean values from eight animals. On the broken lines are values from Table 1 showing the increase in the number of aberrations in irradiated rats as a function of total dose and of time after irradiation. Crosses, group II; Solid triangles, group III; and Solid circles, group IV.

(group II). Further considerable increase is evident until 18 months after exposure (group IV). The increase in spontaneous aberrations of nonirradiated rats is a function of age (Fig. 1). The increase in the percentage of aberrations in irradiated rats over the background of spontaneous aberrations is proportional to the total dose and time after irradiation.

It was difficult to distinguish time after irradiation and age of the rats as separate factors because of the relatively short life span of these animals. This difficulty complicates the interpretation of the increase in aberrations. Two possible mechanisms might account for the progressive increase in aberrations. (i) The degree of damage to stem cells induced by radiation varies. Within 5 days after exposure most of the chromosomal aberrations are eliminated, perhaps because only the less seriously damaged cells divide. At this stage, the number of aberrations is relatively constant or decreases. Later, part of the seriously damaged cells may sufficiently recover to undergo one or more divisions thereby increasing the incidence of aberrations. (ii) Simultaneously the number of abnormal chromosomes may be augmented by spontaneous aberrations in the aging rats. It may be possible to distinguish the effect of the time after irradiation from that of age in organisms with a life span longer than that of the rat. In human bone marrow and leucocytes, the nuclear abnormalities that persist after irradiation slowly decrease (*7*).

Although rats which receive even the lowest doses of irradiation (15 r, 50 r) develop a higher percentage of aberrations than the 30-month-old nonirradiated rats, the increase in spontaneous aberrations in the bone marrow from 2 to 4 percent in young rats to 15 percent in 30-month-old animals cannot be considered negligible. We agree with Curtis (*3, 4*) that spontaneously occurring chromosomal aberrations accumulate in organs whose cells rarely divide, but our results do not completely support his statement that spontaneous aberrations are extremely rare in tissues like bone marrow in which the cell continually divide.

On the basis of our results, we cannot draw definite conclusions concerning the occurrence of threshold doses of irradiation below which increases in aberrations cannot be observed, nor can we determine whether the relation between the increase in aberrations and the total dose of irradiation is linear or exponential.

Table 1. Chromosomal aberrations in bone marrow of continuously irradiated rats. Results are given as mean percentage values of abnormal anaphase and teleophase chromosomes. Group I, rats, 10 to 12 months old, during irradiation. Group II, rats, 10 to 12 months old, 5 days after exposure. Group III, rats, 16 to 18 months old, 6 months after exposure. Group IV, rats, 28 to 30 months old, 18 months after exposure.

Rats (No.)	Dose rate (r/day)	Total dose (r)	Abnormal chromosomes (%)
	Group I		
8	0	0	3.6
8	0.5	15	18.6
8	0.5	50	21.0
8	2.5	250	23.4
8	5	500	27.9
8	10	800	30.3
8	10	1200	37.9
8	20	2000	46.9
8	36	3600	56.6
	Group II		
8	0	0	3.3
8	0.2	27	3.2
7	5.85	801	8.5
8	20.6	1586	9.0
7	82.5	3877	12.9
	Group III		
8	0	0	6.5
10	0.13	18	10.8
10	3.2	445	9.1
10	4.08	567	17.3
10	5.85	814	18.1
10	10.9	1505	21.8
10	27.0	1971	28.8
10	16.3	2266	35.1
10	36.6	2818	31.6
	Group IV		
8	0	0	15.3
10	0.16	16	15.4
14	0.26	26	25.6
14	0.46	46	30.4
10	1.8	234	33.9
8	30.0	900	41.0
10	14.0	910	42.7
10	22.0	1540	54.9

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