of the nasal cavity and is extruded through the opposite nostril as a red bladder. The extrusion of this bladder appears to be confined to the breeding and mating season in the spring.

Nothing in the work of Brönsted[2] is thought to be contrary to the view expressed here.

It would be of interest to know if the male elephant seal, *Mirounga leonina*, the proboscis of which is similar to that of the hooded seal, can extrude a bladder through the nose in the same way.

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Genetic Hazard of Ionizing Radiations

IN current estimates of the genetic hazard of ionizing radiations to man[1,2] four important assumptions are made: (1) that the mutagenic effect of a given radiation dose to the gonads is independent of dose-rate; (2) that the relationship of mutation-rate to accumulated dose is linear; (3) that the spectrum of radiation-induced mutation is similar to the spectrum of spontaneous mutation, and (4) that a dose of about 30–80 rads induces as much mutation as occurs spontaneously per generation ('doubling dose'). Assumptions (1), (2) and (3) derive largely from experiments with *Drosophila*; assumption (4) derives in part from the fact that a spermatogonial doubling dose of this order was estimated for seven specific genes in the mouse from observations of the spontaneous mutation-rate[3,4] and from experiments[4] in which adult males were given a high X-ray dose at a high dose-rate (600 rads, 90 rads/min.), the validity of assumptions (1), (2) and (3) being presupposed.

However, Russell and Kelly[5] have recently stated that they have found a much lower induced spermatogonial mutation-rate at these loci when the dose of 600 rads was given as cæsium-137 γ-rays at a low dose-rate than when it was given as 250 kV. X-rays at a high dose-rate. This means that for mouse spermatogonia one can no longer make assumption (1) and that estimates of the doubling dose will vary with the conditions of irradiation. Since man is likely to resemble the mouse more than *Drosophila* in this respect one can no longer make assumption (1) for man. Most genetically effective radiation exposure of man is due to low doses accumulated over an appreciable fraction of the life-span; it therefore becomes important to have some estimate, however rough, of the mutagenic effect of radiation delivered to the mouse under conditions more nearly comparable with those of most human exposure.

During the past ten years we have been carrying out two such experiments. In the present experiment young adult male mice accumulate 37·5 rads as cobalt-60 γ-radiation over 35 16-hr. nights. They are placed with females immediately after exposure and are bred for up to two years, so the great majority of gametes tested were spermatogonia at the time of exposure. Progeny of exposed and control males are scanned for recessive mutation at the *a*, *b*, *c*, *d*, *se*, *p* and *s* loci and for dominant visible mutation at any locus. The stocks and techniques used have been described elsewhere[6]. The results are summarized in Table 1, together with those from our earlier experiment[6] in which radium γ-rays were used. They yield an estimate of 95 rads for the doubling dose for the seven specific loci, which is higher than previous estimates[1,3] by a factor of 2 or 3. However, the lower 95 per cent confidence limit[7] is only 10 rads and therefore the data do not warrant any change in assumption (4). More than anything else they show the enormous numbers of animals that must be raised in low-dose mutagenesis studies: clearly at least a million more mice must be scanned before we shall have an estimate of the spermatogonial doubling dose that approaches precision.

Table 1

Dose (rads)	Progeny scanned	Specific locus mutations *a*	*b*	*c*	*d*	*se*	*p*	*s*	Total	Dominant visibles
0	18,355*	0	0	0	1	0	0	1	2	0
0	99,372†	0	1	0	3	‡	1	1	6	2
37·5	10,024*	0	0	0	0	0	0	1	1	0
37·5	53,298†	0	2	0	3	0	0	0	5	1

* Data from ref. 6; radium γ-rays used.
† Includes data from ref. 3: cobalt-60 γ-rays used.
‡ Two of the *d*-mutants were also mutant at the closely linked locus *se*. These have been counted as single mutational events in the analysis.

These results have a bearing also on assumption (3). In our experiments *d* has been the most mutable locus, there having been as many mutations at this locus alone as at the other six loci together; but in his high-dose, high-dose-rate experiments Russell[4] found only six *d*-locus mutations among a total of 54, the most mutable locus being *s*, with 25 mutations. Thus in this small sample of loci the spectrum of mutation induced by a high acute dose, on one hand, differed from that of spontaneous mutation and mutation induced by a low chronic dose, on the other. Clearly the validity of assumption (3), as applied to larger samples of loci, requires further test.

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Possible Autosomal Linkage in Man

FAMILIAL intestinal polyposis is known to be an inherited dominant disease[1]. Most families die out after a few generations as the condition frequently causes death before reproduction has occurred, and affected individuals often refrain from marriage because of the danger of passing the condition on to their offspring.

In New Zealand a pedigree is being studied which consists of nearly two hundred individuals covering six generations. This pedigree contains eight families segregating for polyposis. A preliminary investigation, designed to test for possible linkage relations, has suggested that the polyposis gene may be closely linked to the *MN* locus. The method used is that described by