with many strains, we came to the conviction that a reliable species-distinction is only possible if definitely determinable and stable characters are considered. In the genus *Streptomyces*, such characters are (1) morphology of the spores; (2) colour of the aerial mycelium; (3) morphology of the aerial mycelium; (4) formation of a melanoid pigment.

Influence of Hormones and X-Rays upon the Tissue Mast Cell[1]

Treatment with x-radiation[2] cortisone and ACTH[3] elicit widespread damage and disruption of mast cells in rats and hamsters. While numerous reports indicate increased function of the adrenal cortex in animals exposed to x-radiation[4], damage and disruption of mast cells has been found after x-irradiation of adrenalectomized or hypophysectomized animals[5]. The present experiments test whether hormones other than ACTH and cortisone affect mast cells, and whether damage and disruption of mast cells elicited by x-irradiation is referable to some general systemic mechanism or to local tissue injury.

Male, 200-g Sprague-Dawley rats and 100 g-Syrian hamsters were used in this study. Samples of mesentery, skin, and cheek pouch were removed from treated and control animals, prepared as whole mounts, fixed in alcohol, and stained with toluidine blue[6]. Microscopic examinations were carried out at magnifications up to 1000. To evaluate the treatments, separate counts were made of typical mast cells and of abnormal mast cells together with phagocytes (fibroblasts and macrophages) containing metachromatic material. The numbers of phagocytes with metachromatic inclusions indicate the degree of mast cell damage and disruption[7]. Such cells occur in small numbers in normal animals and with great frequency in animals treated with x-radiation[8], cortisone[9], and ACTH[9].

To determine whether hormones other than ACTH and cortisone affect the tissue mast cells, groups of male rats were subjected to the following treatments; testosterone propionate, 25 mg and 230 mg/kg/day; estradiol benzoate, 0·99 and 9·99 mg/kg/day; progesterone, 5 mg/kg/day; chorionic gonadotropin, 2500 I.U. and 5000 I.U./kg/day; and growth hormone[10], 1000 mg/kg/day. All drugs were given daily for 3–4 days by intramuscular injection. Groups of 3 animals for each dosage of the hormones were sacrificed on each day of injection, and noninjected controls were studied simultaneously. None of the hormonal treatments induced significant changes in the number of typical mast cells in the mesentery and skin, or brought about changes in the number of abnormal mast cells and phagocytes containing metachromatic material.

To establish whether the damage to tissue mast cells elicited by irradiation is caused by some systemic effect or is a consequence of local tissue injury, rats and hamsters were subjected to partial-body irradiation. The animals were anesthetized and exposed to single dosage of 600 r or 1200 r of x-rays (250 kv; 15 ma; 0·5 mm Cu and 3·0 mm Bakelite filters; 26·7 cm target distance; 1·5 mm Cu half-value layer; 215–225 r per min). Hamsters were shielded by placing $^1/_4$ inch thick lead forms over the anterior half of the animal; in the rat a lead form was placed over the entire length of either the right or the left side of the body during irradiation. Tissues from irradiated and non-irradiated portions of the animals were examined. The Table shows that damage and disruption of mast cells was confined to those portions of the body exposed to x-rays. The data were analyzed by the H-test and by WILCOXON's two sample, non-parametric test[11]. No significant differences ($p = > 0{\cdot}10$) exist between the phagocyte content of tissues from nonirradiated animals and that of non-irradiated tissues from partially-irradiated animals. In partially-irradiated rats the differences in phagocyte number between irradiated and nonirradiated skin are significant ($p = 0{\cdot}002$) on the second day but not significant at 4 h after exposure to x-rays. In partially-irradiated hamsters such significant differences occur in the 600 r group in the skin on the third day and in the mesentery on the seventh day. In the 1200 r group of hamsters the numbers of phagocytes in both skin and mesentery are significantly different from those in non-irradiated cheek pouch.

Discussion.— Our data show that testosterone, estrogen, progesterone, chorionic gonadotropin, and growth hormone do not affect the mast cells of the male rat. Of these hormones only estrogen has been tested previously; it has been reported to cause a generalized increase in number of mast cells in mice of both sexes[12], and to cause expulsion of cytoplasmic granules from mast cells of the sexual skin of the monkey[13]. It is probable that there are sex and species differences with respect to the effect of estrogen on the tissue mast cell. At present it seems certain that the adrenal-pituitary system can bring about damage and disruption of mast cells[14] and that the thyroid-pituitary system can stimulate production of mast cells[15]. It has been suggested that the estrogen effect in mice is based upon a thyroid-pituitary mechanism[16].

The results of the partial-body irradiations show clearly that damage to mast cells is the result of local injury

[1] This work was performed under auspices of U.S. Atomic Energy Commission.

[2] M. CAMPANI, Boll. Soc. med. chir. Modena *48*, 279 (1948). – D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *82*, 208 (1953); *85*, 306 (1954).

[3] C. CAVALLERO and C. BRACCINI, Proc. Soc. exp. Biol. Med. *78*, 141 (1951). – G. ASBOE-HANSEN, Proc. Soc. exp. Biol. Med. *80*, 617 (1952). – F. BLOOM, Proc. Soc. exp. Biol. Med. *79*, 651 (1952). – D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *87*, 515 (1954); *88*, 631 (1955).

[4] E. P. CRONKITE and V. P. BOND, Ann. Rev. Physiol. *18*, 483 (1956).

[5] D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *87*, 515 (1954); *88*, 631 (1955).

[6] D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *82*, 208 (1953).

[7] D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *82*, 208, (1953); *85*, 306 (1954). – D. E. SMITH, Proc. Soc. exp. Biol. Med. *97*, 872 (1958).

[8] D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *82*, 208 (1953); *85*, 306 (1954).

[9] D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *87*, 515 (1954); *88*, 631 (1955).

[10] Obtained through kindness of Dr. S. W. HIER, Wilson Laboratories, Chicago. The authors thank Mr. SYLVANUS TYLER for statistical analysis.

[11] W. H. KRUSKAL and W. A. WALLIS, J. Amer. statistical Assoc. *47*, 583 (1952).

[12] L. ARVY, Nature *175*, 506 (1955).

[13] O. E. AYKROYD and S. ZUKERMAN, J. Physiol. *94*, 13 (1938).

[14] D. E. SMITH and Y. S. LEWIS, Proc. Soc. exp. Biol. Med. *87*, 515 (1954); *88*, 631 (1955).

[15] G. ASBOE-HANSEN, *Transactions of the Fifth Conference on Connective Tissues* (Josiah Macy Foundation, New York 1954). – L. ARVY and M. GABE, Exper. *6*, 23 (1950).

[16] L. ARVY, Nature *175*, 506 (1955).

Influence of Partial-body X-irradiation upon the Tissue Mast Cell

Treatment	Days after exposure	Irradiated Area				Shielded Area			
		Mesentery		Skin		Cheek Pouch		Skin	
		No. typical cells*	No. phago-cytes**	No. typical cells	No. phago-cytes	No. typical cells	No. phago-cytes	No. typical cells	No. phago-cytes
I. Rat									
600 r	1/6			565	19			507	6
	2			265	155			384	9
1200 r	1/6			396	23			346	4
	2			324	114			349	10
Control				410	4				
II. Hamster									
600 r	3	236	20	318	53	825	14		
	7	246	41	401	16	1205	20		
1200 r	3	298	131	272	64	612	22		
Control		329	9	404	6	569	15		

* No. of cells per 2·028 mm². Average of 4–6 animals/group.
** Fibroblasts and macrophages containing metachromatic material. Also includes a small number of abnormal mast cells.

rather than to some general systemic effect. This finding does not obviate the possibility that the adrenal-pituitary system may add to the irradiation effect.

D. E. SMITH and YEVETTE S. LEWIS[17]

Division of Biological and Medical Research, Argonne National Laboratory, Lemont (Ill.), June 20, 1958.

Résumé

Diverses hormones sexuelles et pituitaires étaient sans effet sur les labrocytes du mésentère et de la peau du rat. L'irradiation partielle du rat et du hamster provoquait des altérations signifiques dans les tissus irradiés mais était sans effet dans les tissus non irradiés.

[17] With assistance of SALLY T. EGAN.

Haploidy Induced by Radiations in Wheat

Devising suitable techniques for the production of haploids in crop plants has long been an important ambition of plant breeders. For this purpose, delayed pollination, distant hybridization, use of irradiated pollen, and use of different hormones to stimulate parthenogenetic development of seeds have all been tried with varying degrees of success in different crop plants. The frequency of occurrence of haploids was found to be increased by X-ray treatment of pollen in *Triticum monococcum* ($2n=14$) by KATAYAMA[1], and in *T. dicoccum* and *T. persicum* ($2n = 28$) by YEFEIKIN and VASILJEV[2]. KIHARA[3] and SMITH[4] raised the frequency of haploid formation from about 1% to 20% by delaying pollination until 6 or more days after emasculation. During the course of our study on the induction of mutations in bread wheat (*Triticum aestivum* L.; $2n = 42$) by the use of different radiations, we found several haploid plants in some of the irradiated progenies and our observations are summarized in this report.

Treatments found effective in inducing haploidy in bread wheat

Radiation	Dose	Stage of treatment
X-rays	5500 r	Irradiation of inflorescences 2 to 3 days prior to anthesis
S^{35}	10 μc/seed	Soaking dry seeds or germinated seedlings for 48 h
P^{32}	10 μc/seed	Soaking dry seeds or germinated seedlings for 48 h
P^{32}	25 and 50 μc/8 lb of soil	Application of P^{32} to soil in pots prior to the initiation of microsporogenesis in the main tiller

Seeds and seedlings of the bread wheat variety N.P. 809 were treated with different doses of X-rays, P^{32} and S^{35} and the second generation progenies were screened for the occurrence of haploids. The X-ray treatments given were (a) irradiation of dry seeds with 11 000, 16 000, or 22 000 r; (b) irradiation of inflorescences of pot grown plants with 5200 r two to three days prior to anthesis, and (c) irradiation of inflorescences with 5200 r 3–4 days after anthesis. Among these, only the treatment of inflorescences prior to anthesis was effective in inducing haploidy. When the earheads were irradiated 3–4 days after anthesis, a large proportion of the seedlings from the X-rayed earheads had twin and triple embryos. No haploid was found among them and cleavage embryony is apparently responsible for the occurrence of the twin and triple seedlings. Haploids occurred more frequently in the progenies of P^{32} and S^{35} treated plants. The different treatments which gave rise to haploid plants in the second generation are described in the table.

It seems likely that owing to the chromosome structural changes induced by the radiation treatments, a certain

[1] Y. KATAYAMA, Cytologia *5*, 235 (1934).
[2] A. K. YEFEIKIN and B. I. VASILJEV, Bull. appl. Bot. Genet. Pl. Breed *2*, 39 (1936).
[3] H. KIHARA, Bot. Mag. (Tokyo) *54*, 174 (1940).
[4] L. SMITH, J. agric. Sci. *73*, 291 (1946).