As a result of these observations, the possibility presented itself of studying the structure when in the uniaxial condition, using a high-temperature single-crystal camera[2]. The first results, however, indicated that the crystals may be too unstable, but it is hoped to continue the study.

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[1] Data contributed by Armour Research Foundation: *Anal. Chem.*, **20**, 491 (1948).
[2] Steward, E. G., *J. Sci. Instr.*, **26**, 371 (1949).

Polydactyly in Mice

THE genetics of polydactyly have attracted attention in a good many organisms, and in mice the pioneer researches of Fortuyn[1], Murray[2] and others have sufficed to reveal a situation of more than ordinary complexity. The first substantial step towards bringing the problem within reach of experimental elucidation was taken by Holt[3] at the Galton Laboratory at University College, London, who demonstrated that by systematic selection a strain could be constructed manifesting polydactyly in nearly 100 per cent of its members.

Last year, using a strain derived from Holt's, but containing in addition a number of recessive genes, a systematic system of matings was set in train which has now gone so far as to clear up the situation to a very large extent.

It appears from recent tests in this Department that the main factor postulated by Holt, *py*, is linked with leaden (*ln*) with a recombination fraction of probably 20-30 per cent. Since no other linkage with leaden has so far appeared, this establishes a new linkage group, to which the number XIII should be assigned, unless indeed some other contemporaneous discovery is more properly assigned to this number.

Homozygotes for *py* are not unconditionally polydactylous, but may have normal feet if one or more of a number of suppressive factors are present. One of these suppressors has been located in the Vth chromosome, between the loci of agouti and pallid. Since this short region already contains the two useful markers, undulated, *un*, and wellhaarig, *we*, we are in a very favourable position for ascertaining the position of its locus with accuracy. It is a powerful suppressor for which a single gene substitution seems to reduce the frequency of manifestation about ten-fold; but more accurate estimates will be possible when it is more accurately located.

At least two other suppressors of unequal intensity have been found in my own stocks, and, of course, elsewhere there may be others. So far, however, I have no indications of linkage for these, though they have been sufficiently isolated to make systematic tests possible. The results to date are, however, enough fully to substantiate the general theory put forward by Holt.

A point of evolutionary interest is that a morphologically similar mutation is known in several species of birds, and is there found to be partially dominant, or, to put it more accurately, not completely recessive. In mice the mutant is not only completely recessive, but appears to have advanced very far in the succession of changes needed to suppress its action even in the homozygote. It is not irrelevant to this comparison that in the last hundred million years or so mouse populations and their ancestors have probably experienced three or four times as many generations as have the birds.

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[1] Fortuyn, A. B. D., *Genetica*, **21**, 97 (1939).
[2] Murray, J. M., Abstr. in *Anat. Rec.*, **57**, 63 (1933).
[3] Holt, S. B., *Ann. Eugen.*, **12**, 4, 220 (1945).

Experimental Enamel Hypoplasia

IN a previous paper[1], a method was described of producing enamel hypoplasia in the rat's incisor tooth by stopping matrix formation by the tall ameloblasts. This method consisted in essentials of first causing healing of low-phosphorus rickets and then suddenly stopping healing by re-imposing the rachitic condition. This procedure stops calcification of bone[2] and dentin, but does not interfere with matrix formation in these tissues. In the case of enamel, however, matrix formation by the tall ameloblasts is stopped permanently, and these cells become temporarily seriously disoriented. The matrix and ameloblasts affected have been called the 'experimental' matrix and ameloblasts.

The chief disadvantage of this technique was that the ameloblasts younger than the experimental ones were often also greatly disoriented. When this happened, they laid down amorphous globular material before starting amelogenesis proper, and sometimes recovery was never seen, and very little new enamel was formed.

Further experiments were therefore undertaken to see if this post-experimental irregular amelogenesis could be stopped. Subsequent work has shown that it was due to the re-imposition of a continuous rachitic condition, probably alternating with periods of healing, since the animals usually gained very little more weight and generally went rapidly downhill after a fortnight or more. To overcome this difficulty, animals were given the full rachitogenic diet for only a short period and were then put on to the complete diet used for stock rats. The procedure may be summarized as follows:

	Treatment	Effect on amelogenesis
1	Rachitogenic diet, 28 days	Nil
2	Dietary restriction to cause healing, 6 days	Nil
3	Full feeding on the rachitogenic diet, 3 days	Cessation of matrix formation
4	Stock diet	Normal formation of new enamel matrix. No more experimental matrix formed

The second period of rickets (3 in the table) lasted long enough for amelogenesis to be stopped, but the post-experimental ameloblasts did not become disorganised. As a result, amelogenesis was invariably resumed by these cells (see photograph). About the first hundred ameloblasts, however, also contributed to the hypoplasia, as they produced matrix normal in structure but abnormally narrow. As one moved towards the formative end of the tooth, the enamel became wider and was finally of proper width. It would appear that the extent of amelogenesis by a particular ameloblast is conditioned by that of the cell ahead of it.

The experimental ameloblasts, which were very disoriented by the procedure, recovered after about eight days, but were now much shorter than before. The changes were similar in many ways to those caused by strontium injections[3]. As found by Chase[4], these ameloblasts could not completely mature or calcify the experimental matrix. This became largely