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NON-GENETIC CONGENITAL LUXATE

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The recent report by Strong and Hardy[8], with its extensive discussion of other recorded inherited "luxoid" conditions (by Carter[1], Green[3], and Rabaud[7]), reminds the author of the importance of reporting congenital limb anomalies. Data for luxate mouse #2213 will be presented to clearly inlicate the phenotypic normality of limb structure for both the parents and the progeny. Thus, the appearance of a mouse with a luxate leg does not necessarily indicate a genetically transmitted condition, even though it is accompanied by polydactyly.

Origin of luxate male #2213

While studying the Crooked mutation[6], it was deemed desirable to establish a parallel normal-tailed tester stock. Both stocks were derived from strain C, which had initially been selected from Bagg albino mice, and were routinely maintained by brother X sister matings. Several hundred genetically normal-tailed mice were produced in the new sublime (2159). Polydactyly was not observed in several thousand mice produced in these two parallel lines. Therefore, when a mouse with a luxate right leg and polydactylous foot was observed in the third generation following establishment of the new line, special efforts were made to determine if the condition was genetic.

Breeding tests

At weaning, the mother and three sisters were left with the luxate male. Data for this and subsequent matings of near relatives are given in table I. The backcross mating of son X mother (item 4) produced four normal mice. Full sibship matings resulted in 32 normal and no abnormal mice (item 5). Inasmuch as litter size was not depleted in matings 4, 5 and 6, the evidence strongly indicated that the luxate condition of #2213 was not transmitted by a dominant gene.

The values in items 7 and 9 constitute F_2 data resulting in 41 normal and no abnormal phenotypes. With the exception of the litter in which the luxate male was produced, there are 255 normal mice accounted for in table I. They were produced from the closest possible matings of relatives. This negative finding excluded the possibility that the anomalous leg shape was transmitted by a recessive gene. Indeed, all evidence, which includes the aforementioned several thousand normal mice from the two

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inbred lines, pointed to the fact that this luxate condition was not transmissable.

*Morphological description**

When the breeding tests were concluded, skeletal studies were made from alizarin-red stained specimens. The right rear leg of male #2213 was the only affected limb. The hallux on the right hind foot was duplicated. The femur appeared normal, so that all of the anomalous features were evident distal to the femur. Each femur measured approximately 15 mm. long. The right patella also appeared normal. Instead of the normal tibia and fibula on the right leg there was one curved bone connecting the femur with the foot. The thickness of this bone was intermediate between that of the tibia and fibula of the left rear leg. Its shape was such that it formed aproximately 200° of a circle from the femur to the foot. The net result was that even though the bone was approximately 10 mm. long, as opposed to 16 mm. for the left tibia, the foot was only 3 mm. from the femur. This, of course, caused the male to hobble about on the stump at the distal end of the femur. Fortunately, this structural defect did not prevent mating.

TABLE I. Progeny of Luxate #2213 and His Relatives.

Mating	*Sire*	*Dam*	*normal*	*luxate*
(1)	Father	Mother	6	1
(2)	Father	Grandmother	2	0
(3)	Father	Other related females	41	0
(4)	#2213	Mother	4	0
(5)	#2213	Full sisters	32	0
(6)	#2213	Other related females	24	0
(7)	Son #2492	Full sisters of son	23	0
(8)	Son #2492	Aunts of son	12	0
(9)	Son #2493	Full sisters of son	18	0
(10)	Son #2493	Aunts of son	11	0
(11)	Sons	Other related females	88	0

Inasmuch as vertebral counts were routinely recorded for the Crooked-tail study, a number of specimens were prepared. Table II contains counts for the caudal, sacral, lumbar, thoracic, and cephalic vertebrae for #2213 and 12 of his near relatives. All were within the expected normal range.[6] Although some of this family did carry the gene for Brachycephaly,[5] no leg or foot abnormalities were observed in any mice except #2213.

Discussion

Three other instances of luxate mice have been reported without evidence for transmissability. One of these[4] is easily disregarded, despite Gruneberg's attempt to expand upon the

*An examination by Dr. T. C. Carter, when he visited the mouse laboratory, indicated that the morphological alterations were indistinguishable from those produced by the "luxate" gene.

possible genetic implications, because the affected female was a runt which died at 23 days of age. However, direct breeding data, which indicated that the anomalous limb condition was not inherited, were presented for one of the other abnormal mice.[2]

Notwithstanding this non-genetic similarity, there are several descriptive differences which make the present report of #2213 distinctive. Perhaps the most obvious difference is that an asymmetrical expression was found for #2213 whereas the other three involved bilateral anomalies. In this respect, the skeletal changes present in congenital luxate #2213 may be more nearly compared to those of Carter's, where the right limb was most severely affected. However, morphologically #2213 could easily be mistaken for one of the mice described as "Luxoid" by Strong, as "Luxate" by Carter, or as "Luxoid" by Green.

TABLE II. VERTEBRAL COUNTS FOR LUXATE AND 12 RELATIVES

Relationship	*Number*	*Cephalic*	*Thoracic*	*Lumbar*	*Sacral*	*Caudal*
mother	1437	7	13	6	4	31
luxate	2213	7	13	6	4	30
sister	2216	7	13	6	4	29
son	2492	7	12	6	4	30
daughter	2494	7	13	6	4	30
daughter	2560	7	13	5	4	31
son	2586A	7	13	6	4	31
son	2586B	7	13	5	4	30
son	2586C	7	13	5	4	30
son	2586D	7	13	5	4	30
son	2586E	7	13	5	4	31
daughter	2588	7	13	6	4	29
daughter	2597	7	13	5	4	31

Inasmuch as a dominant mutation could, conceivably, occur in the somatic tissues of the developing embryo, one possible explanation would be that one small locus which controlled distal differentiation of the right hind limb may have been affected. Although this type of phenomenon has been anticipated, actual morphological proof of its existence has eluded mouse geneticists except for case reports of abnormalities such as color mosaics. As Falconer has stated, when referring to genetic explorations, "The mutation of genes in the somatic tissues of the embryo has been several times observed in mice. But in these cases the mutated gene has always been present also in parts of the germinal tissue, and has therefore been inherited by the offspring." The restriction of the effect on #2213 to one extremity, with the exclusion of the axial skeleton and three-fourths of the appendicular skeleton, suggests that the change was initiated relatively late during embryogeny.

Summary

An abnormal limb development (luxate) observed in an albino mouse is described. The anomalous condition could not be demonstrated as being hereditary. It has been compared to previously reported limb deformities.

LITERATURE CITED

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News of Tennessee Science

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in the tundra of northern Alaska for the Arctic Institute of North America. The project is being directed by Dr. Royal E. Shanks who has spent the previous 3 summers in Alaska. Other botanists participating in this project are John Koranda, Harry Sherman, and Dr. A. J. Sharp, head of the Botany Department at U-T.

Marshall V. Otis, Senior Research Chemist, Tennessee Eastman Company, was recently appointed to the Advisory Board of the Office of Critical Tables, National Academy of Sciences, to represent the American Society for Testing Materials.

Biology Division—Oak Ridge National Laboratory

Dr. William A. Arnold is spending the summer months working with Dr. Albert Szent-Gyorgi at the Institute for Muscle Research, Woods Hole, Massachusetts. Dr. Arnold's studies concern energy transfer in biological systems, particularly in muscles.

Dr. Madeline Moutschen-Dahmen, a citizen of Belgium, has reported to work in the Cytology and Genetics Section for a period of one year under an International Cooperation Administration fellowship. Dr. Moutschen-Dahmen is a member of the staff of the University of Liege, Belgium.

Dr. John S. Kirby-Smith has returned to the Biology Division from a one yearp's leave of absence spent at St. Bartholomew's Hospital, London, England. Dr. Kirby-Smith is head of the Biophysics Group.

Karl Bruce Jacobson has reported to work in the Nucleic Acid Chemistry Group. Dr. Jacobson received the Ph.D. degree from the McCollum-Pratt Institute of the John Hopkins University and for the past two years has been associated with Dr. Linus Pauling at California Institute of Technology, Pasadena.

Dr. N. E. Tolbert, who has been head of the Plant Biochemistry Group, ha saccepted a position as Professor of Agricultural Chemistry at Michigan State University, East Lansing.