Cystic Prostatic Hypertrophy in Two Inbred Lines of Syrian Hamsters[1] (34777)

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Benign, spontaneous, prostatic hypertrophy is rare in laboratory rodents. In fact, the only reference in the literature relates to a proliferative hyperplasia of the prostate gland in female mastomys (1). Among experimental animals, only the age-dependent, cystic, spontaneous prostatic hypertrophy of the dog has been studied (2).

This report describes an age-dependent, cystic, spontaneous, prostatic hypertrophy of Syrian hamsters which appears to be genetically controlled, since it was found in only two of six inbred lines studied.[2]

Materials and Methods. The animals studied were BIO® hamsters[3] (*Mesocricetus auratus*) of six pedigreed inbred lines developed at this Institute and characterized as follows:

Line 2.4—originated from London School of Hygiene, agouti color, inbred for 30 generations; longevity (expressed as the number of days during which half a population survives under standard conditions) is 532 days.

Line 87.20—originated from Ingham-Toolan-Gulf, rust color, inbred for 20 generations; longevity is 670 days.

Line 12.14—originated from Warren, cream color, inbred for 22 generations; longevity is 473 days. All males are affected by late-occurring, sex-linked, progressive hind-leg paralysis (3).

Line 40.54—originated from Schwentker-Toolan-LaCasse-Gulf, agouti and acromelanic white color, inbred for seven generations; longevity is unknown. All animals are affected by a hereditary (autosomal recessive) myopathy and cardiopathy.

Line 1.5—originated from the National Institutes of Health, acromelanic white color, inbred for 19 generations; longevity is 590 days; dental caries-susceptible (4).

Line 4.24—originated from Schwentker, agouti color, inbred for 36 generations; longevity is 589 days; obesity, high adrenal tumor incidence (5).

Male animals at about sexual maturity (60 days old) and males older than 150 days, were taken from a breeding colony which was maintained under standard conditions (temperature controlled at 65–72°F.; light exposure from 7:00 AM to 9:00 PM; Guilford breeder chow and Cambridge City water *ad libitum* with Ab-Sorb-Dri bedding). They were killed by intraperitoneal pentobarbital, pelvic organs were dissected, and the prostate was weighed, fixed in formaldehyde, and prepared for histological examination in hematoxylin–eosin-stained sections.

Results. The results are summarized in Table I in Figs. 1–4. In from four to seven

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[3] Available from the Trenton Experimental Laboratory Animal Company (TELACO), Bar Harbor, Maine.

FIG. 1. Section of a normal prostate (hematoxylin–eosin, ×100) of hamster of BIO 4.24 line, aged 298 days. Note size of acini and papillae in glandular mucosa.

FIG. 2. Section of a moderately hypertrophic prostate (hematoxylin–eosin, ×100) of hamster of BIO 87.20 line, aged 148 days. Note the marked distention of most acini and the flattened appearance of the epithelium in the distended acini.

FIG. 3. Section of a markedly hypertrophic prostate (hematoxylin–eosin ×100) of hamster of

BIO 87.20 line, aged 206 days. Note the extreme dilation and cystic nature of acini in absence of any stromal reaction.

FIG. 4. Gross specimen of reproductive tract of hamster of BIO 2.4 line, aged 195 days; arrow indicates hypertrophied prostate.

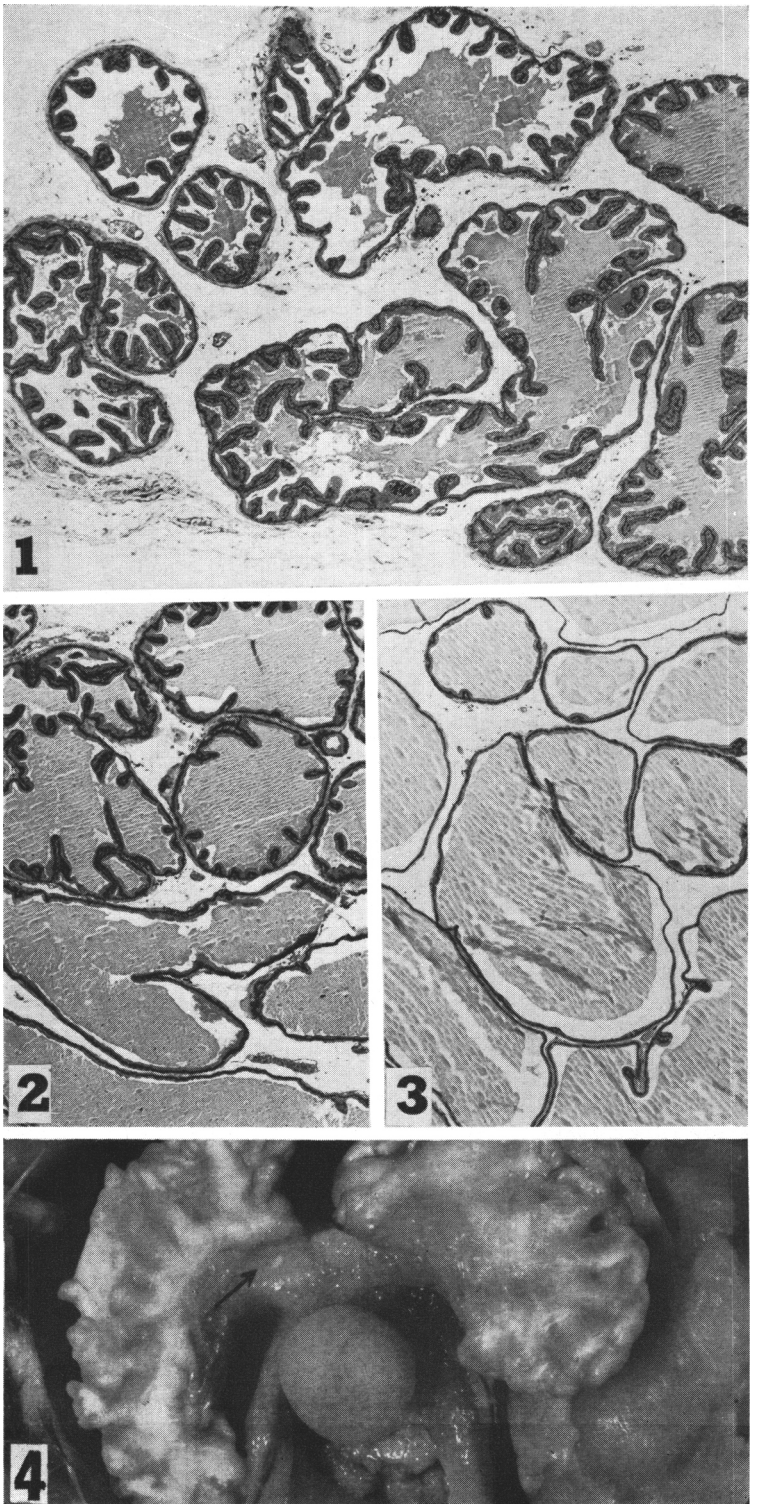

TABLE I. Age and Prostate Weight in Six Inbred Strains of Hamsters.

Strain	No. animals	Age in days average (range)	Prostate weight average (range)
2.4 (LSH)	13	191 (117–186)	198 (102–419)
	4	61 (all 4, 61)	75.5 (73– 89)
87.20	11	184 (122–347)	124 (89–168)
	7	61 (60– 64)	70 (52– 89)
12.14	9	210 (134–450)	50 (40– 63)
	4	62 (all 4, 62)	45 (36– 50)
40.54	9	164 (117–181)	55 (35– 84)
	4	58 (54– 61)	47 (44– 48)
1.5	9	177 (182–390)	70 (50– 90)
	4	63 (all 63)	50 (42– 56)
4.24	7	175 (118–229)	80 (47–114)

animals of each of five inbred lines, aged approximately 60 days, the prostate weight averaged just under 50 mg in three lines, and 70 and 75.5 mg in the 87.20 and 2.4 lines, respectively. These apparent differences are not statistically significant.

Animals aged, on the average, from 164 to 210 days were studied in each of six lines. In four of these lines, there were no significant differences between prostate weights (50–80 mg average). The prostate weights of animals of lines 87.20 and 2.4, on the other hand, differed markedly from all others, ranging from 89 to 419 mg, and averaging 124 mg and 198 mg, respectively.

Histological examination revealed that the marked weight changes were due to a cystic dilatation of the prostatic acini which are filled with an eosinophilic, amorphous material. There was no evidence of inflammation, except in a few instances in which there were large deposits of calcium in the enlarged glands. There were minimal stromal hyperplasia and minimal changes in the epithelial cells.

Discussion. In two inbred lines of Syrian hamsters, there occurs in all aging animals a cystic hypertrophy of the prostate. In some respects, this resembles the cystic prostatic hypertrophy of dogs. No evidence was found suggesting that this caused obstruction of the urinary tract. Since four inbred lines of hamsters were free of this change, prostatic hypertrophy in this species appears to be governed by genetic factors which remain to be studied.

1. Snell, K. C., and Stewart, H. L., J. Nat. Cancer Inst. **35**, 7 (1965).

2. Berg, O. A., Acta Endocrinol. **27**, 140 (1958).

3. Nixon, C. W., and Connelly, M., J. Heredity **59**, 276 (1968).

4. Keyes, P. H., *in* "The Golden Hamster, Its Biology and Use in Medical Research," (R. A. Hoffman *et al.*, eds.), p. 253. Iowa State Univ. Press (1968).

5. Homburger, F., and Russfield, A. B., Cancer Res. Vol. 30, May, 1970.

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