$^{35}S/^{3}H$ ratios of 0.87 and 1.37 for untreated and treated cultures respectively and a similar ratio of 1.48 obtained from cultures containing foetal calf serum and without any added IGF I, tend to suggest that glycosaminoglycans synthesized without serum or IGF I appear to be less sulphated in addition to being synthesized at a lower rate. The remarkably similar effects of foetal calf serum and IGF I on chondrocytes, causing them to synthesize increased amounts of glycosaminoglycans with similar $^{35}S/^{3}H$ ratios, demonstrates not only that IGF I, which is contained in foetal calf serum, supports the synthesis of collagen and glycosaminoglycans; the data also indicate that IGF I may play an important role in the sulphation of cartilage glycosaminoglycans.

Interestingly, in the case of the chick embryo cartilage, incorporation of $^{35}SO_4$ is not as markedly stimulated by IGF I as are other processes[12]. These species differences of the response of cartilage to growth factors are remarkable and yet unexplained.

Finally, when IGF I was added for 24 h to already confluent chondrocytes, a modest but statistically significant rise in cell proliferation was noted (table 3). These data re-emphasize the potency of IGF I on cell growth, shown previously with fibroblasts[12].

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A histometric analysis of male accessory sex glands after administration of prolactin[1]

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Summary. Histometric analysis of accessory sex glands in male albino mice after prolactin treatment revealed stimulatory responses in the epithelium of the dorsolateral lobe of the prostate. This lobe was more sensitive to prolactin than the ventral lobe. This stimulatory effect may be mediated through testosterone rather than being a direct action of prolactin.

Prolactin (PRL) is involved in the mammary function[2] and luteotrophic activity in female mice[3]. It stimulates testicular growth and induces fertility in PRL deficient male mice[4]. Although there is considerable evidence to suggest that PRL can stimulate the growth and development of accessory sex glands in male rats[5,6], its mode and site of action on these glands are not fully understood. Moreover, histometric details of accessory sex glands in male mice under the influence of PRL are not well documented. The present study is aimed at evaluating the site and mode of action of PRL in accessory sex glands of male mice.

Materials and methods. 2 groups (10 in each) of colony-bred adult male albino mice (72 days old; b.wt 25–27 g) were used in the present study. Animals were maintained at

Table 1. Effect of prolactin on histometry of dorsolateral prostate in male albino mice

	Volume/unit volume of tissue			Surface area of sec. epi.	Height of epithelium
	Stroma	Sec. epi.	Sec. alveoli	(mm^2/mm^3)	(μm)
Control	0.446 ± 0.013	0.214 ± 0.007	0.553 ± 0.013	16.86 ± 0.18	8.25 ± 0.07
Experiment	0.363 ± 0.012**	0.322 ± 0.003*	0.636 ± 0.012**	20.52 ± 0.23*	10.6 ± 0.16*

Results are expressed as mean ± SEM. * $p < 0.001$; ** $p < 0.05$. Sec. epi., secretory epithelium; sec. alveoli, secretory alveoli.

Table 2. Weights of accessory sex glands in male albino mice after prolactin treatment

	Seminal vesicle (1 lobe with secretion; mg)	Ventral prostate (mg)	Dorsolateral prostate (both lobes; mg)
Control	56.6 ± 5.47	12.5 ± 1.02	8.6 ± 0.73
Experiment	72.0 ± 7.2	10.4 ± 1.64	11.7 ± 1.78

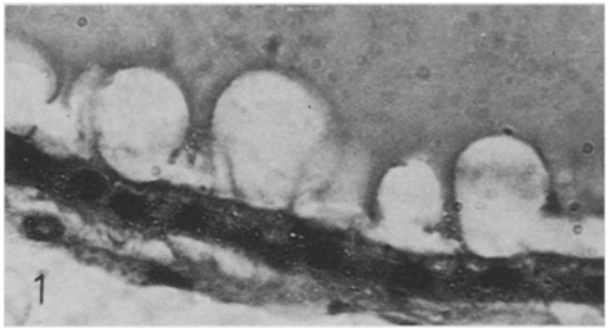

Figure 1. Photomicrograph of section through an alveolus of dorsolateral prostate of a control male albino mouse illustrating the height of the epithelium. H and E stain × 1000.

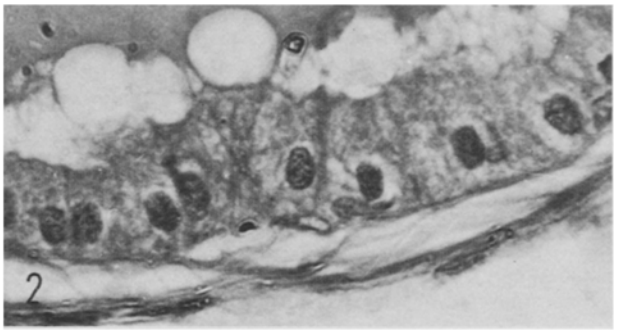

Figure 2. Photomicrograph of section through an alveolus of dorsolateral prostate of a PRL-treated male albino mouse. Note the increased height of the epithelium as compared with the same in controls. H and E stain × 1000.

room temperature (25–30 °C). JIPMER mouse food and water were given ad libitum. 20 µg of ovine PRL in saline was administered s.c. daily for 14 days to group A animals. Group B animals (controls) were similarly injected with 0.05 ml saline only. All the animals were sacrificed by cervical dislocation and decapitation 24 h after the last injection. Seminal vesicles and prostates were removed from each animal and fixed in Bouin's fluid for 24 h and processed for light microscopy. Paraffin blocks of seminal vesicles and prostates were sectioned at 5 µm thickness. Step sections at regular intervals through the entire tissue block, representing different areas of the same organ, were mounted on microscope slides. Sections on alternate microscope slides were stained with H and E and Mallory's triple stain. Volumes of various tissue components and surface areas of the secretory epithelium of accessory sex glands were determined histometrically by point and intercept counts using an eyepiece reticule[7] (American optical, 21.90 mm diameter disc; 10 mm^2 division; into 1 mm^2). The magnification used was × 100. The formula used for estimation of volume:

$$Vi = Pi/PT$$

where Vi = Volume of the particular tissue component in question per unit volume of tissue.
Pi = Number of points touching the particular tissue component.
PT = Total number of points in the reticule.

The formula used for estimation of surface area:

$$Sv = 2P/L$$

where Sv = Surface area of the particular tissue component in question in square units per cubic units.
P = Number of intercepts in relation to the particular component in question.
L = Sum of length of all lines in the line grid.

The heights of the epithelia were measured using an ocular micrometer (magnification × 1000).

Data were analyzed statistically including the determination of means and SD, and subjecting these to Student's t-test.

Results. Alveoli of the dorsolateral prostate (and seminal vesicle) of controls were lined uniformly by cuboidal epithelium and they contained thick eosin stained colloidal secretion. Histometric data for the dorsolateral prostate are summarized in table 1. It is evident from this table that the volume of secretory epithelium was significantly increased in the experimental group. Moreover, the epithelium was very much folded, so that the surface area was significantly increased. In the PRL-treated animals there was an increase in the volume of secretory alveoli at the expense of the stroma, which was significantly reduced. The height of the epithelium was also increased significantly in the same animals (fig. 2). No significant change was observed in the ventral prostate, but the seminal vesicles showed a slight increase in epithelial height in experimental animals. Though there was a slight increase in the weight of seminal vesicle and dorsolateral prostate in PRL-treated animals, no statistical significance was observed when compared with controls.

Discussion. The present histometric study on the prostate clearly shows that PRL administration causes a significant stimulation of the secretory epithelium in the dorsolateral lobe of the prostate, whereas the ventral lobe remains unaffected. This fits in with the findings of Grayhack et al. who demonstrated that PRL administration could augment the weight of the dorsolateral lobes more than that of other lobes of the rat prostate[8,9]. Further studies revealed that citric acid levels in the dorsolateral prostate were augmented by PRL[9]. The possible mechanism for the stimulation of the dorsolateral prostate may be mediated through testosterone rather than being due to a direct action of PRL, resulting from the fact that Leydig cells are stimulated and their volume increased after PRL treatment[10]. Now the question is why the ventral lobe is not stimulated. It has been shown that higher amounts of exogenous PRL are necessary to cause an enlargement of the ventral lobe of the prostate in rats[11]. It appears that the ventral lobe of the prostate is PRL dose-dependent, and PRL may have a direct action on this lobe (experiments in progress).

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