An Elastin Gel

The present report describes the conversion of insoluble aorta elastin to a gel. Wood[1] recently described the preparation of an elastin gel from a soluble protein derived from cattle ligamentum nuchæ. These observations may have some bearing on the relationship between collagen and elastin[2–5].

Elastin was prepared from cattle aorta by a method described previously[6], in which 1 *N* sodium hydroxide is used for removing collagen. (Some cattle aorta do not yield insoluble elastin under these severe conditions, perhaps owing to their age.) For the preparation of the gel, the elastin was suspended at room temperature in a 0·1 per cent solution of elastase, in acetate buffer at *p*H 4·7 and ionic strength 0·1. (Two different elastase preparations were used : a lyophilized water extract of porcine pancreatin and porcine elastase *PI*[6].) After at least 10 min., the suspension was centrifuged and the residue washed with the buffer. The washed residue was then suspended in 10 vol. of 0·2 *N* acetic acid, from which, on standing, the gel formed.

Gel formation does not occur when (*a*) cattle ligamentum nuchæ is the elastin source ; (*b*) cattle aorta elastin is boiled in 0·1 *N* sodium hydroxide ; or (*c*) the enzyme treatment is omitted.

The gel melts upon either warming or adding sodium chloride, with subsequent formation of an elastin precipitate. An approximation of the melting point can be made by suspending 1 vol. of packed gel in 5 vol. of aqueous solvent and noting the temperature at which flocculation begins. As the concentration of sodium chloride of the suspension medium increases from 0·01 *M* to 2·0 *M*, the melting and precipitation point first rises and then falls, the maximum occurring between 0·05 and 0·5 *M* sodium chloride. Syneresis has never been observed, even after prolonged storage at 4°C., in the absence of salt.

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PHYSIOLOGY

Experimentally Induced Proliferation of the Rudimentary Gonad of an Intact Domestic Fowl

It is well known that the right female gonad of the domestic fowl ceases to grow about the ninth day of incubation and that it always hypertrophies following sinistral ovariectomy. I have shown[1] that œstrogen could act *in vivo* as the inhibitor of rudiment growth. In view of the anti-œstrogenic effect of 17 α-ethyl-19-nortestosterone ('Nilevar', Searle) upon the chicken's oviduct[2], the effect of this substance upon the rudimentary chicken gonad was investigated. Starting at 60 days of age intact White Leghorn females received 40 subcutaneous injections of 'Nilevar'. The substance was dissolved in a carrier of 5 per cent benzyl alcohol in corn oil and 0·1 c.c. of solution was injected daily. Experimental procedures were analogous to those outlined in the earlier paper[1] and the weights of blotted, fixed (Bouin's fixative) tissue were recorded (Table 1).

Table 1

No. of birds	Treatment	Rudiment weight (mgm.) at 100 days ± standard error of the mean
9	Carrier	5 ± 1
10	1·5 mgm. 'Nilevar'/day	13 ± 1
8	3·0 mgm. 'Nilevar'/day	20 ± 2

Statistical analysis showed the differences due to treatment to be highly significant.

This is the first report of experimentally induced proliferation of the rudimentary intact fowl gonad. Detailed results, including evidence that the proliferation is due to the anti-œstrogenic action of 'Nilevar', will be published elsewhere.

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Effect of Testosterone Propionate and Insulin on Muscle Cholinesterase Activity

Various hormones influence the cholinesterase activity of tissues[1,2]. The purpose of the present work was to investigate the influence of some anabolic hormones such as testosterone and insulin on muscle cholinesterase activity.

Muscle cholinesterase was studied on male albino rats weighing 250 gm. and kept on a standard stock food. The animals were divided into three groups. One group received 1 mgm. testosterone propionate in 0·2 c.c. sesame oil per animal for ten days, the second group 2 I.U. of crystalline insulin per kgm. body-weight for the same period. The third group was used as control. The animals were autopsied 24 hr. after the last injection, and the cholinesterase activity was determined in diaphragm and gastrocnemius in tissue homogenates by Michel's method[3]. The results (Table 1) are expressed in Δ *p*H/hour per gm. of wet tissue weight.

Table 1. Effect of Testosterone Propionate and Insulin on Rat's Gastrocnemius and Diaphragm Cholinesterase

	No. of animals	Gastrocnemius	Diaphragm
Control	24	0·783 ± 0·068	0·910 ± 0·070
Testosterone propionate (1 mgm. per day)	12	1·410 ± 0·110 ($P < 0·01$)	1·065 ± 0·050 ($P < 0·05$)
Insulin (2 I.U. per kgm. body-weight per day).	12	1·157 ± 0·048 ($P < 0·01$)	1·204 ± 0·064 ($P < 0·05$)

The cholinesterase activity is expressed in Δ *p*H/hour per gm. of wet tissue. The determinations were carried out on 0·2 gm. wet tissue.

As is seen in Table 1, testosterone propionate produces a significant increase in cholinesterase activity of gastrocnemius ($P < 0·01$) and a rather moderate increase in diaphragm cholinesterase activity