

LETTERS TO THE EDITORS

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Effect of Testicular Hormone on the Formation of Seminal Fructose

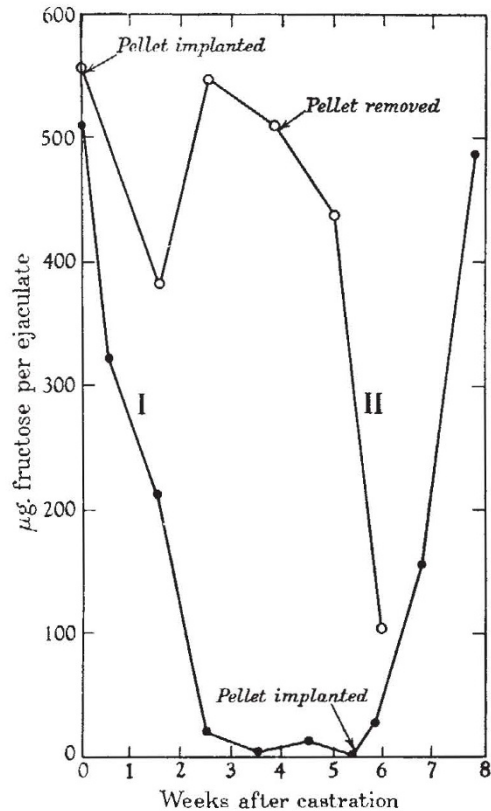
PREVIOUS experiments have established the fact that mammalian semen contains fructose, its concentration varying from a few mgm. per cent in boar to 1,000 mgm. per cent in bull; this sugar serves the spermatozoa as their natural nutrient, and they readily metabolize it^{1,2}. At the site of its origin in the testis, and in the epididymis, the semen contains very little fructose but acquires it during its passage through the male generative tract, from the accessory glands of reproduction, mainly the seminal vesicles³.

In the course of experiments designed to solve the mechanism of fructose formation in the accessory glands, we have now found that the process of fructose generation is linked in a striking manner with the action of the testicular hormone and, furthermore, that characteristic fluctuations in the level of seminal fructose can be evoked in response to testosterone. For the investigation we have used sixteen fully fertile bucks; some of them were castrated, while the others were used as controls. The collection of semen was carried out at weekly intervals by the method of Macirone and Walton⁴. Fructose was determined by the method previously described, using 0.1 ml. samples of semen and 0.05-0.2 gm. samples of the various tissues³.

EFFECT OF TESTOSTERONE ON FRUCTOSE CONTENT IN ACCESSORY GLANDS

	Gl. vesicularis and seminalis		Prostate	
	Weight mgm.	Fructose mgm. %	Weight mgm.	Fructose mgm. %
Non-castrated buck	780	62	860	79
1 week after castration	860	84	590	18
2 weeks after castration	630	5	410	5
5 weeks after castration	420	3	540	3
8 weeks after castration and simultaneous implantation of testosterone pellet (100 mgm.)	1100	29	1170	71

Within the first two or three weeks following castration, there was a remarkable fall in the fructose content of accessory glands, and usually by the end of the second week the organs were almost completely depleted of fructose. This post-castrate fall in fructose, however, could be prevented by the implantation of pellets of pure testosterone under the skin of the castrated animals (see table). An even clearer picture was obtained through the study of the effect of testosterone on the level of fructose in the semen itself, as illustrated in the accompanying graph. Castration caused a sharp decrease in both the volume of ejaculates as well as in the concentration of fructose in semen, so that the actual quantity of fructose per ejaculate, which normally amounts to some 500 μ gm., dropped within three weeks following castration to less than 20 μ gm., remaining low until a pellet of testosterone was implanted. In response to testosterone, there was an increase in the volume of ejaculates together with a rise in fructose which soon reached the pre-castration level (Curve I). In presence of the pellet, a high concentration of fructose could be maintained in the castrated animal for several weeks. However, when the pellet was removed, a significant fall in seminal fructose was registered again (Curve II). From these results we are inclined



to conclude that testosterone is the hormone responsible for the formation and maintenance of fructose in semen.

It has long been known that the weight, size, histological appearance and secretory function of certain accessory organs, notably the seminal vesicles and prostate, are strictly dependent on, and closely regulated by, the internal secretion of the testes; and that the typical retrogressive changes which develop in the accessory glands after removal of the testes can be counteracted by injections of the testicular hormone, thus serving as 'indicator tests' for the male hormone. To these biological tests may now be added the highly sensitive 'fructose test' whereby the testicular hormone can be conveniently assayed in castrated animals, simply by examining the fructose content of the semen or of the accessory glands. It may be added that the post-castration fall in the level of fructose and its restoration by testosterone occurs more rapidly and is much more marked than the rather slow and gradually developing gross morphological changes in the accessory glands of reproduction.

This work has been carried out on behalf of the Agricultural Research Council. We are grateful to Dr. J. Hammond, Mr. J. Hammond, jun., Prof. D. Keilin and Dr. A. Walton for placing the experimental animals at our disposal, for help and advice, and to Dr. W. J. Tindall, Organon Laboratories, for a gift of pure testosterone.

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¹ Mann, T., *Nature*, 157, 79 (1946).

² Mann, T., *Biochem. J.*, 40, xxix (1946).

³ Mann, T., *Biochem. J.*, 40, 481 (1946).

⁴ Macirone, C., and Walton, A., *J. Agric. Sci.*, 28, 122 (1938).