et al.[12] found the inulin from the Jerusalem artichoke produced additional glycogen-storage in the white rat. The average glycogen content found by these workers was 0.69%. It will be observed that the percentage of liver-glycogen in the experiments with rats fed the inulin from Allium are less than half the values obtained when the Camas and Burdock inulins were fed. Owing to the tremendous variations inherent to the experiment the authors do not interpret this as meaning that Allium is less capable of forming liver-glycogen than the other 2 inulins.

In all cases, however, the high percentage of absorption from the alimentary tract, accompanied with a high percentage of utilization is significant. With feeding experiments on dogs the authors found 86% absorption when Burdock inulin was fed.

Summary. In the white rat, the absorption and utilization of the insoluble inulin from Burdock and the soluble inulins from Camas and Allium, show no significant difference.

7273 P

Further Studies Concerning Testicular Function.

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Fifty-four albino rats were divided into 4 groups: (1) normal controls, (2) castrated animals, (3) castrated animals injected with androtin (male sex hormone prepared from urine) and (4) normal animals injected with androtin. The androtin was prepared by extracting male urine with fat solvents. The injected rats each received from 8 to 10 bird units of androtin daily.

The results are shown in Table I. The weights of these animals are expressed in grams and the weights of the formalin-fixed organs as percentages of the total body weight. In the glands other than the sex glands, the difference between the weights of the glands in the control animals and of those in the injected normal animals is not sufficiently great to be of any significance. The seminal vesicles, the ventral and dorsal prostates all show an increase in weight of approximately 100% after the injection of androtin into normal rats. Other workers have stated that hormone prepared from the testes causes

[12] Bodey, M. G., *et al.*, *J. Biol. Chem.*, 1927, **75**, 715.

testicular degeneration.[1] If there was any testicular degeneration in the animals injected with androtin, it was not discernible by gross examination. The weights of the epididymises of the injected rats are slightly increased above those of the normal.

TABLE I.

Effect of Injection of Hormone into Normal and Castrated Rats.

	Normal	Normal injected with male sex hormone for 20 days	Castrated	Castrated and injected with male sex hormone for 21 days	Castrated	Castrated and injected with male sex hormone for 21 days
Duration of Exp.		20	22 days	21 days	85 days	85 days
No. of animals	9	7	6	12	10	10
Original wt. of animals	211	223	207	204	195	174
Final " " "		226	200	208	195	194
Testes	1.050	.986				
Epididymis	.319	.353				
Seminal vesicles	.231	.366	.043	.306	.032	.165
Ventral prostate	.142	.256	.015	.189	.017	.085
Dorsal "	.044	.076	.010	.061	.010	.035
Thyroid	.014	.013	.013	.015	.014	.014
Adrenals	.012	.016	.013	.015	.025	.016
Pituitary	.003	.004	.004	.004	.006	.006
Thymus	.090	.080	.098	.072	.097	.086

In mature rats, the effects of castration have been prevented for 3 weeks by the injection of androtin. In that length of time, the secondary sex glands become almost completely atrophic in untreated, castrated animals. However, during the same period, there are no significant changes in the thyroid, adrenals, pituitary or thymus glands, with the possible exception of the pituitary, which may be slightly hypertrophic.

Because the atrophy of the secondary sex glands commences within 5 days after castration and progresses very little, if any, after 3 weeks, much work previously done on the castrated rats has been limited to a 3-week period. We have studied the organs of rats which have been castrated for 76 to 107 days, averaging 85 days, and have also studied a similar group which were castrated for 85 days and treated for the last 21 days with testicular hormone. Following castration, atrophy of the secondary sex glands occurred. Grossly, the atrophy is not much more marked than that which was observed in animals examined at the end of 3 weeks. At the end of 85 days, the weight of the thyroid was not changed. The adrenals and the pituitary showed very definite and unquestionable hyper-

[1] Moore, C. R., *Proc. Second Int. Cong. Sex Res.* (London), 1931, 293.

trophy at the end of this longer period, both being increased in weight approximately 100%. On injecting these animals with androtin, the secondary sex glands commence to regenerate. No hypertrophy of the adrenals was observed in 4 of the animals which were injected with androtin for the last 21 days of an 85-day period, and in the other 6, the hypertrophy was so slight as to be questionable. In this group of castrated animals which had an adequate amount of hormone to maintain the prostate and to reduce the hypertrophy of the adrenals, the pituitary hypertrophy was not altered during a period of 3 weeks.

7274 C

Spread of Poliomyelitis Virus from the Gastrointestinal Tract.*

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Flexner and Lewis[1] first showed that poliomyelitis could be transmitted by intranasal instillation, the spread of the virus being along the olfactory nerve to the brain. The olfactory nerve, however, is not the only unmedullated one situated in the nasal area, for it is precisely here under the mucosa that the 1500 cells of nervus terminalis are placed,[2] composing a network of cells and fibers as might be found in the myenteric or submucous intestinal sympathetic.[2, 3] The position of the plexus of the nervus terminal near the embryological juncture of the stomodeum and the possible upper tip of the mesenteron, *i. e.*, the upper end of the foregut of the endodermal tube is significant, since the other portion of the alimentary tract, the hind gut, also possesses the same kind of unmedullated nerve fiber plexuses. If the virus has a facultative or almost an obligate affinity for grey fibers, this area should also provide an easy portal of entry. Perhaps failure to produce the disease after the virus of poliomyelitis had been introduced into the gastrointestinal tract was because the virus never approximated the grey fibers.

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[1] Flexner, S., and Lewis, P. A., *J. Am. Med. Assn.*, 1910, **54**, 1140.

[2] Brookover, C., *J. Comp. Neur.*, 1917, **28**, 2.

[3] Huber, C. G., and Guild, S. R., *Anat. Rec.*, 1913, **7**, 253.