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Appendix VI

CONTROL OF RAT TESTICULAR MONOAMINE OXIDASE (MAO) ACTIVITY: HYPOPHYSECTOMY, ADRENALECTOMY, FSH, LH, PROLACTIN, HCG, AND TESTOSTERONE[1]

Ronald L. Urry,[2] John L. Frehn,[3] and LeGrande C. Ellis

Department of Zoology, Utah State University

Logan, Utah 84321

DISCLAIMER

Suggested Running Head: Control of testicular MAO

Please send proofs to: LeGrande C. Ellis, Ph.D.

Department of Zoology

Utah State University

Logan, Utah 84321

Footnotes

1. We thank the National Institute of Arthritis and Metabolic Diseases for the generous gift of FSH, LH, and Prolactin, and Utah State University Research Project U-300, and U.S. Atomic Energy Commission AT(11-1)-1602 for their support of this research.

2. Predoctoral Candidate, Department of Zoology, Utah State University, Logan, Utah 84321.

3. On sabbatical leave from his present position in the Department of Biological Sciences, Illinois State University, Normal, Illinois 61761.

ABSTRACT

Monoamine oxidase activity (MAO) was measured in male rats using homogenized testicular tissue preparations. Rats were subjected to hypophysectomy, adrenalectomy, and treatment with HCG, FSH, LH, Prolactin, or a combination of FSH, LH, and Prolactin. Hypophysectomy of male rats at three or ten weeks of age significantly reduced testicular MAO activity when these animals were compared with controls. HCG treatment in hypophysectomized animals significantly increased androgen synthesis and prostate and seminal vesicle weights, but had no effect on MAO activity. Treatment with FSH, LH, or a combination of FSH, LH, and Prolactin increased MAO activity in hypophysectomized animals. FSH was the most effective in increasing testicular MAO activity. Adrenalectomy had no effect on MAO activity.

Exogenously administered serotonin (5-HT) in rats (1,2,3,4) and mice (5) was noted to cause testicular regression as evidenced by a reduction in testicular weight and histological abnormalities. Other workers (6) have shown that exogenous 5-HT-^{14}C accumulates in rat testes 120 minutes after sublingual injection. Moreover serotonin is an inhibitor of androgen synthesis in vitro (7-9) and small amounts of endogenous 5-HT (60-100 nanograms/gram of tissue) are present in rat testes (7,10). In addition, 5-HT is thought to be responsible for alterations of spermatogensis observed after postural stress in primates (15).

Monoamine oxidase (MAO, EC 1.4.3.4.), the enzyme responsible for 5-HT inactivation, has been observed in the male gonad (8,11,12) using various techniques. This enzyme was observed histochemically in rat testes during late embryonic and early postnatal periods (8) with the most intense activity present during the late embryonic period. In an aging study (6) in our laboratory MAO activity was assayed in rat testes from 1 day after birth through 410 days of age. In this study, MAO activity was high at birth, low for 41 and 57-day-old rats, elevated for 105-day-old rats and diminished at 365 days of age, and was closely correlated with androgen synthesis. In addition, photoperiod and crowding have been shown to reduce MAO activity in Uinta Ground Squirrel Testes (13).

Although the available data suggest that 5-HT and its metabolism by MAO may play an important functional role in development of the male gonad (6), little information is available as to what regulates the activity of this

enzyme. This study was undertaken to ascertain if hypophysectomy, FSH, LH, Prolactin, HCG, or adrenalectomy would influence MAO activity of rat testes.

MATERIALS AND METHODS

Animals and Treatments

Hypophysectomy

For these investigations, hypophysectomized and control male rats (Hormone Assay Laboratories) were purchased and maintained in a small animal laboratory with a southern exposure under controlled conditions. Artificial lighting was used during the daylight hours only when the caretaker was caring for the animals. Temperature was maintained at 72°F with a relative humidity of 35%. The animals were housed in a professional animal care unit (4-6 animals/cage in cages 13.5" x 16" in size) with feed (laboratory chow) and water given ad libitum.

In the first series of experiments, MAO activity was measured in the testes of hypophysectomized and control animals. These included: 1) animals hypophysectomized at three weeks and sacrificed at seven and eighteen weeks of age (N=7 and 6, respectively). Animals were also sacrificed at seven and eighteen weeks (N=6 and 5, respectively). 2) Additional animals were hypophysectomized at ten weeks and sacrificed at 11, 13, 14, and 16 weeks of age (N=5, 5, 6 and 6, respectively). Control animals were sacrificed at 11 and 14 weeks of age (N=5 and 6, respectively).

In the second experiment, MAO activity and androgen synthesis were measured in the testes of animals hypophysectomized at three weeks of age. Four hypophysectomized animals were injected subcutaneously with 50 I.U. of HCG (Sigma Chemical Co.) for 18 days prior to sacrifice, while four rats were injected with saline. Five unoperated and noninjected rats were used as intact control animals. Injections were administered daily at 4:00 p.m. All animals were sacrificed when 10 weeks of age.

In the third experiment, six control and twenty-nine hypophysectomized animals (three weeks old at the time of surgery) were sacrificed three weeks after surgery, and testicular MAO activity and androgen synthesis were determined for each animal. The hypophysectomized animals were randomly subdivided into five groups. The first group contained five animals that were injected sub. Q. with 20 μg FSH (in 0.1 ml of 0.9%NaCl) twice daily (9:00 a.m. and 4:00 p.m.) for four days prior to sacrifice. The second group of six rats was injected with 2.5 μg of LH. The third group of six animals was injected with 100 μg Prolactin, while the fourth group of six rats was injected with a combination injection of 20 ug FSH, 2.5 ug LH, and 100 ug Prolactin. The fifth group of six animals was injected with 0.9% NaCl.

Adrenalectomy

Seven control and eight male rats, adrenalectomized at 62 days of age, (Hormone Assay Laboratories) were purchased and maintained in the animal

laboratory with feed, tap water, and saline (0.9%) given ad libitum to all animals. The animals were sacrificed after ten days and testicular MAO activity was assayed for each rat.

Tissue preparation

The animals used in the above investigations were weighed and sacrificed by decapitation. The testes, adrenals, seminal vesicles, and prostates were rapidly removed, chilled, trimmed, and weighed. The testes were decapsulated and aliquots were weighed, minced or homogenized, and androgen synthesis and MAO activity were assayed as described below.

MAO assays

Aliquots of the decapsulated testicular preparations described above were incubated with either 5-HT-2-^{14}C binoxolate (18 mCi/mmole New England Nuclear Corp.) or 5-HT Creatine sulfate (1-^{14}C 45 mM Schwarz/Mann). MAO activity was expressed as DPM of recovered 5-HIAA-2-^{14}C obtained using specific extraction and purification procedures combined with thin-layer chromatography (Eastman Chromatofilm) as described in detail elsewhere (14). All MAO and androgen samples were counted in a liquid scintillation spectometer (Packard Instr. Co.).

Androgen synthesis

From one to three-tenths aliquots of minced preparations were assayed

for androgen synthesis as described elsewhere (9, 20-23).

Statistics

Statistical comparison of sample means was made with a standard t-test.

RESULTS

Hypophysectomy

In all investigations utilizing hypophysectomized animals, body, testicular, seminal vesicle, and prostatic weights were significantly reduced ($P < 0.001$ unless otherwise specified) by hypophysectomy when compared to control animals.

In the first series of experiments, hypophysectomy of the rats at three weeks of age and sacrificing them at seven (Fig. 1) and eighteen weeks of age (Fig. 2-B) resulted in a significant decrease in testicular MAO activity compared with the controls when expressed on either a per animal or on a per 100 grams of body weight basis. When the data were expressed on a per mg tissue basis (Fig. 1), however, the MAO activity of the hypophysectomized animals was significantly higher than that of the control group.

Animals hypophysectomized at ten weeks of age and sacrificed either one-week or three-weeks after surgery (Fig. 2-C) also showed a decrease in testicular MAO activity when the data were expressed on a per animal basis when compared with the control group. The difference between the three-week

hypophysectomized rats and the controls approached significance ($P < 0.10$). A significant difference was noted between the one- and three-week treated animals. There were also significant differences in testicular, prostatic, and seminal vesicle weights when the one-week hypophysectomized animals were compared with the three-week hypophysectomized animals (data not shown). In addition, animals hypophysectomized at ten weeks of age and sacrificed at either four weeks or six weeks after surgery (Fig. 3) also exhibited significantly lower MAO activity than control animals when expressed on a per animal basis. MAO activity differed significantly between the four- and six-week hypophysectomized animals. When the data were expressed per 100 grams of body weight basis, a significant difference was observed between the control and four-week hypophysectomized animals. MAO activity, when expressed on a per mg tissue basis, for the four-week hypophysectomized animals was significantly higher than that observed for the control group, and MAO activity of the six-week hypophysectomized rats was significantly higher than that observed for the four-week hypophysectomized animals. Significant differences were noted in testicular, prostatic, and seminal vesicle weights when the four-week hypophysectomized animals were compared with the six-week hypophysectomized animals (unpublished data).

In the second investigation (Fig. 2-A), MAO activity was significantly lower for the HCG-treated and the hypophysectomized control animals. MAO activity and testicular weight did not differ significantly when the HCG-treated group was compared with the untreated hypophysectomized

animals, but prostatic and seminal vesicle weights were significantly increased by the HCG treatment. Testosterone and androstendione synthesis were significantly increased ($P<0.05$) by HCG treatment when these animals were compared with the control hypophysectomized animals (Table 2). Testosterone and androstendione biosynthesis was significantly lower for the control hypophysectomized and HCG-treated hypophysectomized animals when compared to the unoperated animals.

There were significant reductions in adrenal, seminal vesicle, prostatic, body, and testicular weights when the untreated hypophysectomized and various hormone-treated hypophysectomized animals were compared to control animals (Table 1). Untreated hypophysectomized adrenal weights were significantly lower than adrenal weights for the combination-treated hypophysectomized animals. No significant changes in seminal vesicle weights were noted among the hypophysectomized groups. Prostatic weights of the Prolactin-treated hypophysectomized animals were significantly lower than those observed for the untreated hypophysectomized FSH, LH, or combination-treated hypophysectomized animals. Combination injected rats had significantly increased prostatic weights when compared with untreated hypophysectomized animals. Testicular weights of the LH and Prolactin-treated animals were significantly lower than the FSH or combination-treated animals. Testicular weights of the FSH, LH, and combination-treated animals were higher than those of the untreated hypophysectomized animals.

MAO activity (Fig. 4), calculated on a per animal basis, was significantly lower for the untreated hypophysectomized and all groups of treated hypophysectomized animals when compared to controls. MAO activity of the untreated hypophysectomized animals was significantly lower than the FSH, LH, or combination ($P<0.05$) treated animals. FSH and combination treatment significantly increased ($P<0.05$) MAO activity when these groups were compared to the Prolactin-treated animals. The difference between the MAO activity of the FSH-treated animals with the combination-treated animals approached significance ($P<0.20$). There was no significant difference in MAO activity between the FSH- and LH-treated animals.

Testosterone and androstenedione synthesis were measured for these animals (Table 2). Untreated hypophysectomized and all groups of treated-hypophysectomized animals had significantly lower testosterone and androstenedione synthesis when compared to control animals. Treatment of the animals with FSH, LH, or a combination of FSH, LH, and Prolactin significantly increased (see Table 2 for P values) testosterone and androstenedione synthesis when compared to untreated hypophysectomized animals. The testosterone and total androgen fractions were significantly higher in the combination-treated animals when compared to untreated hypophysectomized or all other groups of treated-hypophysectomized animals. Treatment with FSH or LH significantly increased testosterone and the combined testosterone plus androstenedione fractions when compared to Prolactin-treated hypophysectomized or untreated hypophysectomized animals.

Adrenalectomy

Adrenalectomy significantly reduced body weights compared to control animals, but testicular weights were not significantly changed (Table 3). MAO activity did not differ significantly from control animals when calculated on a per mg tissue or per animal basis, but it was significantly different from the control group when expressed on a per 100 grams of body weight basis.

DISCUSSION

Our data show that a factor from the hypophysis is important in regulating testicular MAO activity. This conclusion is based on the finding that hypophysectomy reduced testicular MAO activity and that LH or a combination of FSH, LH, and Prolactin can increase testicular MAO activity, although neither of these treatments was as effective as FSH. Thus, FSH may be the important factor from the hypophysis which can regulate testicular MAO activity.

It is highly unlikely that the increase in MAO activity by LH and Prolactin was due to contamination with FSH since LH was judged to contain less than 0.050 NIH-FSH-S1 units/mg while Prolactin had less than 0.018 NIH-FSH-S1 units/mg. Similarly, it is unlikely that the increase in MAO activity by FSH would have been due to LH activity since it contained less than 0.010 NIH-LH-S1 units/mg and had negligible effects

on testosterone production.

The fact that those hypophysectomized animals that were sacrificed at sixteen weeks of age had a higher MAO activity than those sacrificed at fourteen weeks of age suggests that there is another mechanism independent of the hypophysis that can regulate testicular MAO activity.

Our data indicate that endogenous androgens do not play an important role in the regulation of testicular MAO activity since HCG treatment did not alter MAO activity, but did increase both androgen synthesis (testosterone and androstenedione) and accessory sexual organ weights. This conclusion is corroborated by the observation that a combination of FSH, LH, and Prolactin was the most effective treatment for increasing androgen synthesis and testicular and accessory sexual organ weights, but it was not as effective as FSH in increasing testicular MAO activity.

Since adrenalectomy did not alter testicular MAO activity we conclude that the adrenocorticosteroids do not play an important role in the regulation of this enzyme. This finding differs from that reported by other workers (24,25) who used different substrates and found that adrenalectomy increased MAO activity in the rat heart and hypothalamus. This has led us to conclude that there may be a different pattern of response in testicular MAO when compared to MAO in other tissues, especially when using different substrates.

Testicular MAO activity has been shown to be influenced by photoperiod, and crowding (13), and irradiation (14). Thus, environmental

factors, including social stress and population density may influence MAO activity in the testis, and may be important in regulating testicular function in those animals subjected to conditions of crowding and changes in photoperiod. These effects could be mediated in part through changes in FSH. Further studies are being undertaken to clarify this point and to further ascertain the specific effects of irradiation, stress, crowding, and photoperiod on testicular MAO activity.

It is possible that factors which change the metabolism of serotonin may play an important role in normal testicular function. This conclusion is based on the observations that serotonin alters spermatogenesis and androgen biosynthesis and appears to be the factor responsible for determining the androstenedione/testosterone ratios in the blood of animals (2-5, 7- 9). Moreover, serotonin metabolism in the testis has been shown to be related to the aging process and directly related to androgen synthesis (6) and serotonin may also be involved in mediating the effects of stress in the testes (7). Moreover, immobilization has been shown to cause severe generalized testicular degeneration in Macaca nemestrina with only severe ischemia and serotonin administration known as possibilities to explain the disappearance rate of the spermatids and spermatocytes (15). In addition, blood levels of serotonin have been observed to increase after irradiation (26,27) and MAO levels change in the testes after this treatment (14). Changes in some testicular enzymatic processes and spermatogenesis have been noted in

similar studies (16,17) where serotonin has been implicated. Atrophy of the testicular germinal epithelium with disappearance of spermatocytes and sperm motility has been noted in dogs during the first few weeks of cage life (18), and testicular damage in bulls has been noted after they have been transported some distance by truck (19). The mechanisms causing the above testicular degenerations are not fully known, but serotonin and its metabolism could be involved in these changes.

References

1. Salgado, E.D. and D.M. Green, Amer. J. Physiol. 183:657, 1955.
2. Boccabella, A.V., E.D. Salgado, and E.A. Alger, Endocrinology 71:837, 1962.
3. Kormano, M., P. Karhunen, and K. Kahanpaa, Ann. Med. Exp. Fenn. 46:474, 1968.
4. Liberman, S.S., and N. G. Shakhnazarova, Arkh, Patol. 32:29, 1970.
5. O'Steen, W.K., Proc. Soc. Exptl. Biol. Med. 113:161, 1963.
6. Ellis, L.C., A.W. Jaussi, M.H. Baptista, and R.L. Urry, Endocrinology--submitted for publication 1971.
7. Ellis, L.C., in Johnson, A.D., W. R. Gomes, and N.L. Vandemark (eds.), The Testis, Academic Press, Inc., New York, Vol. III, 1970, p. 333.
8. Penttila, A. and M. Kormano, Ann. Med. Exp. Biol. Fenn. 46:557, 1968.
9. Ellis, L.C., Endocrinology 90:17, 1972.
10. Kormano, M. and A. Penttila, Ann. Med. Exp. Fenn. 46:468, 1968.
11. Bhagvat, K., H. Blaschko, and D. Richter, Biochem. Jour. 33:1338, 1939.
12. Zeller, E.A., and C.A. Joel, Helv. Chim. Acta. 24:968, 1941.
13. Frehn, J.L., R.L. Urry, D.F. Balph, and L.C. Ellis, J. Exp. Zol.--submitted for publication 1972.
14. Urry, R.L., A.W. Jaussi, and L.C. Ellis, Anal. Biochem.--submitted for publication 1972.

15. Zemjanis, R., B. Gondos, W.R. Adey, and A.T.K. Cockett, Fertil. Steril. 21:335, 1970

16. Cockett, A.T.K., A. Elbadawi, R. Zemjanis, and W.R. Adey, Fertil. Steril. 21:610, 1970.

17. Cockett, A.T.K., R. Zemjanis, A. Elbadawi, and W.R. Adey, Fertil. Steril. 22:565, 1971.

18. Huggins, C. and M.K. Masina, J. Exp. Med. 70:543, 1939.

19. Meschaks, P. in Wolstenholme, E.W., M.P. Cameron, and J.S. Freeman (eds.), Ciba Foundation Symposium of Mammalian Germ Cells, Little Brown and Co., Boston, 1953, pp. 37-45.

20. Ellis, L.C., and K.R. Van Kampen, Radiation Res.--in press 1971.

21. Ellis, L.C. and D.L. Berliner, Radiation Res. 20:549, 1963.

22. Ellis, L.C., and D.L. Berliner, Endocrinology 76:591, 1965.

23. Berliner, D.L., in Rothchild, S. (ed.), Advances in Tracer Methodology, Plenum Press, New York, Vol. II, 1964, p. 135.

24. Avakian, V.M., and B.A. Callingham, Br. J. Pharmac. Chemother. 33:211, 1968.

25. Holzbauer, M., and M.B.H. Youdim, Br. J. Pharmac.

26. Momma, H., Nakamura, W., and Eto, H., Compt. Rend. Seances Soc. Biol. 159:2094, 1965.

27. Nakamura, W., Momma, H., and Eto. H., Compt. Rend. Seances Soc. Biol. 159:1629, 1965.

TABLE 1

Changes in Organ and Body Weights from Rats Sujected to Hypophysectomy, FSH, LH, Prolactin, and Combination Injection Treatments *

Treatment	Adrenals (mg)	Seminal Vesicles (mg)	Prostate (mg)	Body Weights (grams)	Testes (grams)
Control	37.6 ± 2.01[5]	32.2 ± 4.47	36.9 ± 4.88	117.9 ± 16.3	1.30 ± .046
Control Hypophy.	11.8 ± 0.59	10.5 ± 1.82	9.6 ± 0.45	74.1 ± 2.30	.14 ± .007
FSH	11.7 ± 1.6	10.8 ± 1.03	10.6 ± 1.92	72.6 ± 1.31	.24 ± .009[3]
LH	12.6 ± 0.66	11.9 ± 0.94	10.3 ± 2.18	70.5 ± 2.42	.17 ± .009[1,4]
Prolactin	13.3 ± 1.11	10.1 ± 1.53	6.4 ± 0.77[2]	75.9 ± 2.69	.15 ± .011[4]
Combination	14.2 ± 1.28[1]	12.0 ± 0.54	13.3 ± 0.60[3]	69.2 ± 2.44	.26 ± .008[3]

Adrenals, seminal vesicles, prostate, body, and testicular weights of the hypophysectomized, control, and treated hypophysectomized animals were significantly lower ($P < 0.001$) when compared with the control animals

$P < 0.05$ when compared with hypophysectomized controls

$P < 0.001$ when compared with hypophysectomized controls or all groups of treated hypophysectomized animals

$P < 0.001$ when compared with hypophysectomized controls

$P < 0.001$ when compared with FSH or Combination-treated hypophysectomized animals

Standard error of mean

TABLE 2

Testosterone and Androstenedione Synthesis in Rats Subjected to Hypophysectomy, and Treatments with HCG, FSH, LH, Prolactin, or a Combination of FSH, LH, and Prolactin. (Calculated on a per animal basis).

Treatment	Testosterone (Nanomoles)	Androstenedione (Nanomoles)	Total Androgens (Testosterone plus Androstenedione Nanomoles)
Experiment Two			
Control	2.110 ± 0.359	1.700 ± 0.239	3.810 ± 0.590
Hypophysectomized	0.013 ± 0.002[1]	0.065 ± 0.011[1]	0.078 ± 0.012[1]
HCG-Treated Hypophysectomized	0.114 ± 0.043[2,3]	0.579 ± 0.202[3,4]	0.653 ± 0.219[2,3]
Experiment Three *			
Control	2.085 ± 0.257	1.242 ± 0.127	3.327 ± 0.439
Hypophysectomized	0.127 ± 0.040	0.040 ± 0.012[1]	0.167 ± 0.044[1]
FSH-Treated Hypophysectomized	0.485 ± 0.127[3]	0.132 ± 0.035[3]	0.622 ± 0.161[3]
LH-Treated Hypophysectomized	0.610 ± 0.055[5]	0.493 ± 0.075[5,6]	1.104 ± 0.060[5,7]
Prolactin-Treated Hypophysectomized	0.266 ± 0.065[8,9]	0.108 ± 0.032[8,10]	0.373 ± 0.010[8,10]
Combination-Treated Hypophysectomized	1.016 ± 0.060[5,7,12]	0.200 ± 0.023[5,9,12]	1.216 ± 0.074[5,7,11,12]

$P < 0.001$ when the untreated and all groups of treated hypophysectomized animals were compared to control animals for testosterone, androstenedione, and total androgen fractions.

1. $P < 0.001$ when compared to control animals
2. $P < 0.01$ when compared to control animals
3. $P < 0.05$ when compared with untreated hypophysectomized animals
4. $P < 0.05$ when compared to control animals
5. $P < 0.001$ when compared to untreated hypophysectomized animals
6. $P < 0.01$ when compared to FSH-treated hypophysectomized animals
7. $P < 0.05$ when compared to FSH-treated hypophysectomized animals
8. $P < 0.10$ when compared to untreated hypophysectomized animals
9. $P < 0.01$ when compared to LH-treated hypophysectomized animals
10. $P < 0.001$ when compared to LH-treated hypophysectomized animals
11. $P < 0.05$ when compared to LH-treated hypophysectomized animals
12. $P < 0.001$ when compared to prolactin-treated hypophysectomized animals

TABLE 3

Testicular MAO Activity from Rats Subjected to Adrenalectomy

Statistical Basis	TREATMENT Control	Adrenalectomized
Counts/mg tissue	21,703 ± 960[1]	21,765 ± 540
Counts/animal	64,304,691 ± 2,154,739	65,207,830 ± 1,942,477
Counts/100gm Body Weight	27,167,965 ± 1,110,492	34,482,381 ± 1,043,588[2]

[1] Standard Error of Mean

[2] $P < 0.001$ when compared to control animals

Legends for Figures

Fig. 1. Rat testicular monoamine oxidase activity of control animals and animals hypophysectomized at three weeks of age. Both groups of rats were sacrificed at seven weeks of age.

Fig. 2. Rat testicular monoamine oxidase activity. A, control animals and animals hypophysectomized at three weeks of age and sacrificed at ten weeks of age. One group was treated with 50 I.U. of HCG for 18 days. B, control animals and animals hypophysectomized at three weeks of age and sacrificed at eighteen weeks of age. C, control animals and animals hypophysectomized at ten weeks of age and sacrificed 1 and 3 weeks after surgery.

Fig. 3. Rat testicular monoamine oxidase activity of control animals and animals hypophysectomized at ten weeks of age and sacrificed at four or six weeks after surgery.

Fig. 4. Testicular monoamine oxidase activity of control animals and animals hypophysectomized at three weeks of age and sacrificed at six weeks of age. Groups of hypophysectomized animals were injected with either FSH, LH, Prolactin, or a combination of FSH, LH and Prolactin.

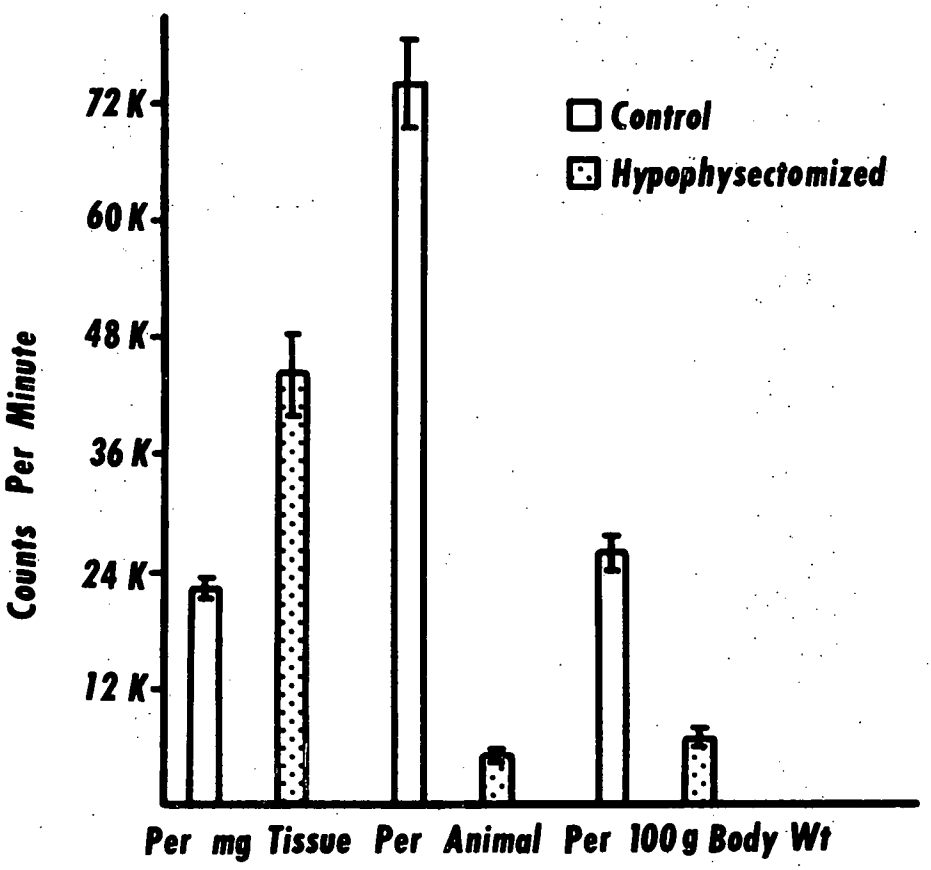

Fig. 1.
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Fig. 2.
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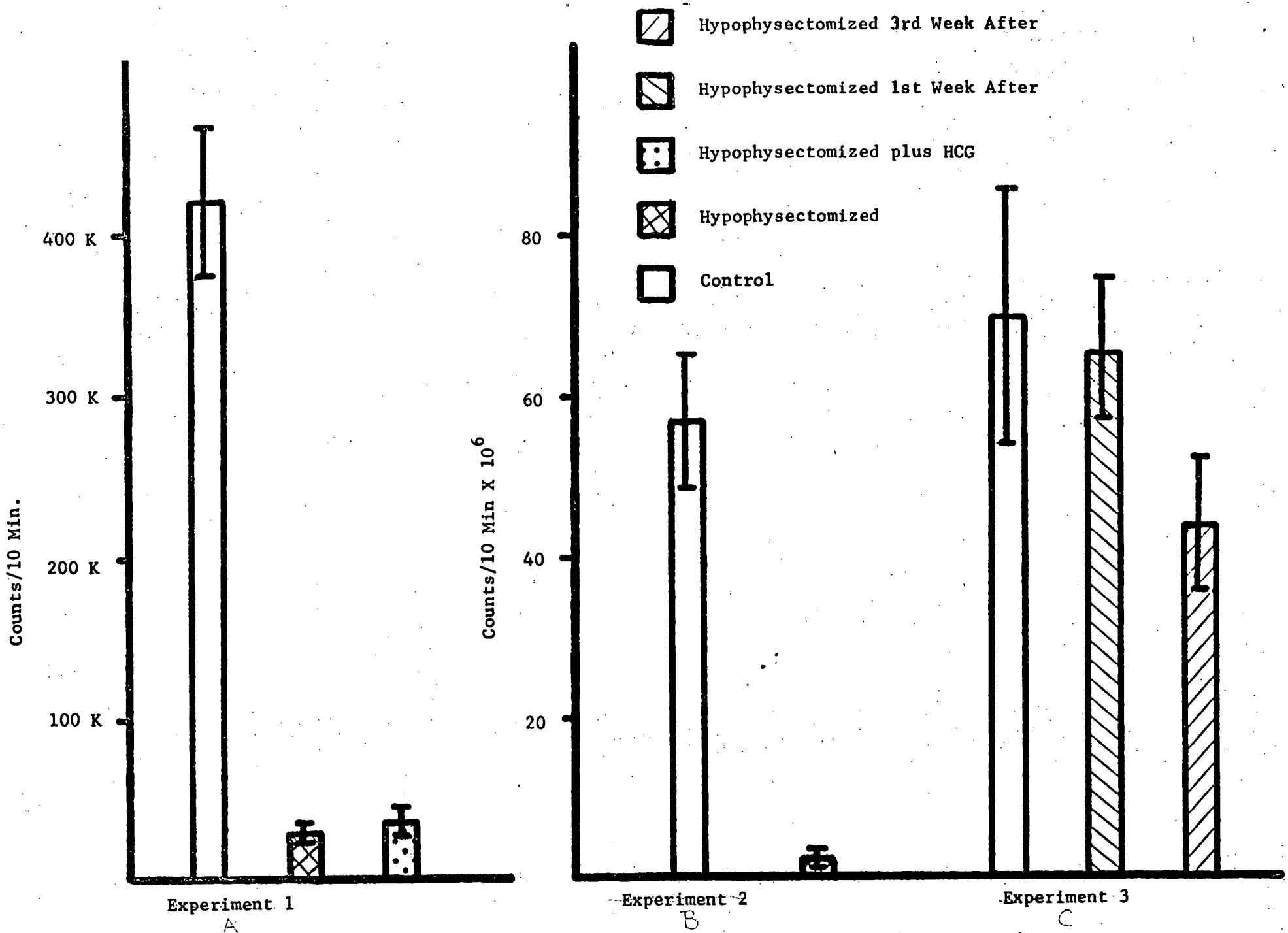

Figure 1 Testicular MAO Activity in Control, Hypophysectomized Control, and HCG-Treated Animals

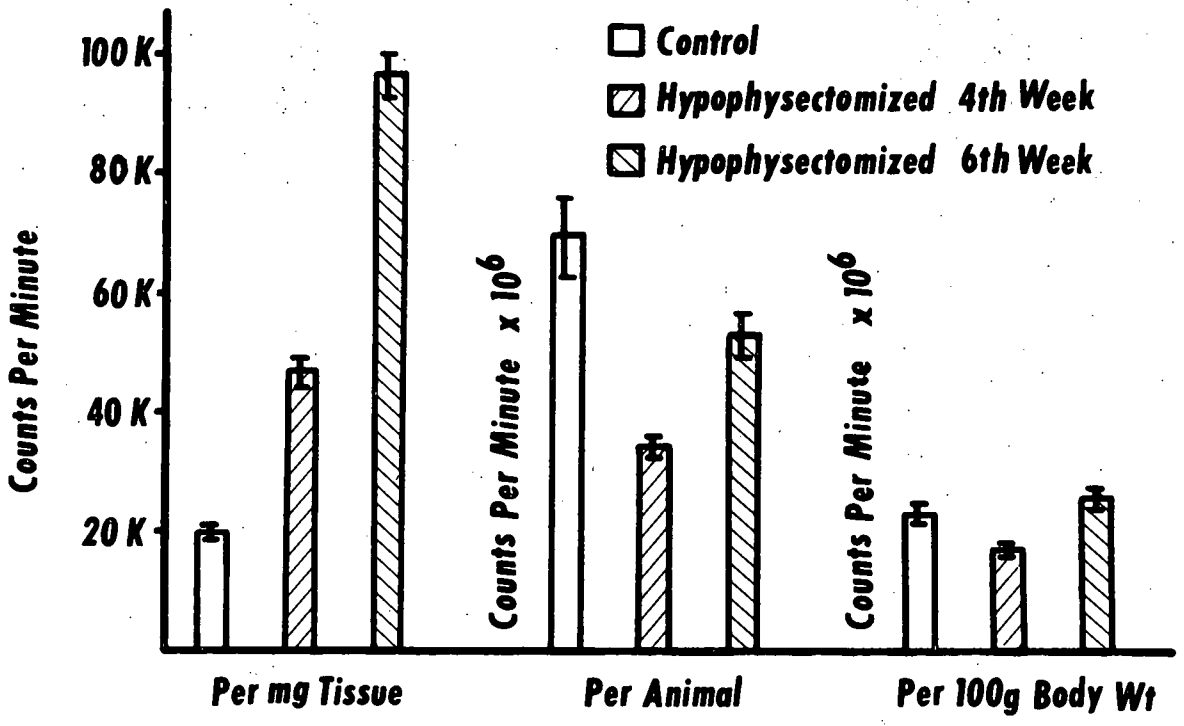

Fig. 3.
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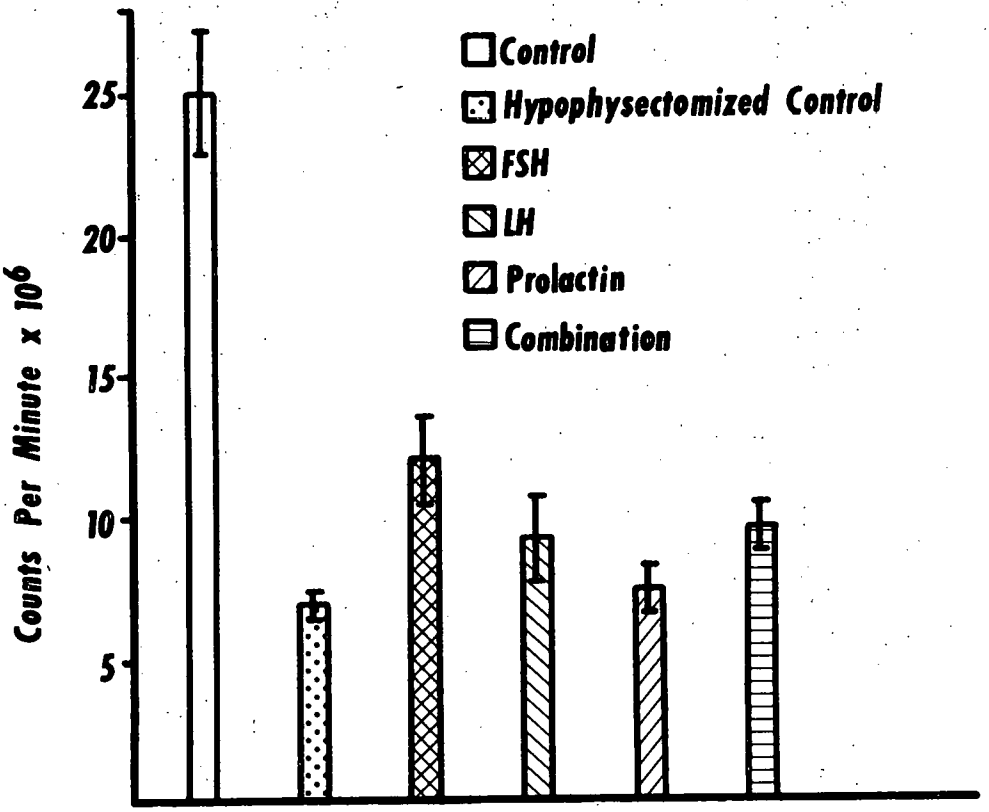

Fig. 4.
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ABSTRACT

Monoamine oxidase (activity (MAO)) was measured in male rats using homogenized testicular tissue preparations. Rats were subjected to hypophysectomy, adrenalectomy, and treatment with HCG, FSH, LH, Prolactin, or a combination of FSH, LH, and Prolactin. Hypophysectomy of male rats at three or ten weeks of age significantly reduced testicular MAO activity when these animals were compared with controls. HCG treatment in hypophysectomized animals significantly increased androgen synthesis and prostate and seminal vesicle weights, but had no effect on MAO activity. Treatment with FSH, LH, or a combination of FSH, LH, and Prolactin increased MAO activity in hypophysectomized animals. FSH was the most effective in increasing testicular MAO activity. Adrenalectomy had no effect on MAO activity.