INFLUENCE OF CASTRATION AND TESTOSTERONE PROPIONATE ON PROSTATIC AND SEMINAL VESICULAR LIPIDS IN MATURE MONKEYS

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Summary : The effect of castration and administration of testosterone propionate (TP) has been studied in mature monkeys in relation to prostatic and seminal vesicular lipids to castrates on prostatic and seminal vesicular lipids were studied in mature monkeys. Castration decreased prostatic total lipid, total phospholipids and total glyceride glycerols. Among neutral lipid classes only diacyl glycerol was decreased due to castration. Similarly among phospholipid classes, phosphatidyl choline and ethanolamine were decreased. A marked decrease in the seminal vesicular total lipid, total phospholipids, total cholesterol and total glyceride glycerol were observed due to castration. Mono and diacyl glycerols and phosphatidyl choline and ethanolamine also registered a significant decrease after castration. Administration of TP to castrates brought back all the lipid classes to normal level which showed a decrease due to castration. However, TP treated to castrates on both seminal vesicles and prostates had no effect on other classes of lipids which were unaltered by castration.

The present study reveals that lipid metabolism in the male accessory sex organs is under androgenic control. Nevertheless, the influence of testicular androgen is specific to different classes of lipids as a number of phospholipids and neutral lipid classes were resistant to castration or administration of TP.

Key words : Prostate, seminal vesicles, lipids, glyceride glycerols, phospholipids, cholesterol

INTRODUCTION

A major portion of seminal lipids is contributed by seminal vesicle and prostate (20). The prostatic and seminal vesicular lipids have been reported to be under androgenic control in the rat (9) but not much information is available for primates in this regard. Further, monkey is an ideal model for the study of many human diseases and pathological conditions due to its phylogenetic proximity to man (13, 17). In this paper, we report the influence of androgens on prostatic and seminal vesicular lipids in monkeys.

MATERIAL AND METHODS

Healthy mature male bonnet monkeys (9-10 *kg* body weight) of *Macaca radiata* species were used in the present study. The age of the animal was computed on the basis of dentition (12). The animals were divided into the following 3 groups of 10 animals each.

1. Control : Sham operated and administrated vehicle only, every day for 21 days, intraperitoneally.
2. Castrated : Administered vehicle only ip every day for 21 days after castration.
3. Castrated and treated with testosterone propionate (TP) : 1 *mg/kg* body weight (ip) every day for 21 days.

Bilateral castration was performed through scrotal route following the guide lines of Resko and Phoenix (16). Hormone administration was begun 21 days after castration. TP was dissolved in propane-1-2-diol. Twenty four hours after the last injection, all the animals were sedated by sodium pentobarbitone (30 *mg/kg* body weight, intraperitoneally) and transcardially perfused with physiological saline till the tissues were completely bleeched. The seminal vesicles and prostate was dissected out quickly and trimmed of adhering connective tissue. They were cut into small pieces and rinsed several times in cold saline, blotted, and weighed accurately on a torsion balance. The tissues were processed as quickly as possible.

Analytical methods : The lipids were extracted in Chloroform : Methanol (2 : 1 v/v) containing 0.01% butylated hydroxytoluene as an antioxidant (10). Total lipid was quantified by the classical gravimetric method (10). Standard colorimetric methods were used for determining cholesterol (11) and glyceride glycerol (6). Phospholipid phosphorous was estimated by the method of Marinetti (15). The phosphorous values so obtained were multiplied by 25 to obtain phospholipid phosphorous values (4).

Separation of neutral lipids was achieved by two dimensional thin layer chromatography on glass plates coated with Silica Gel G (BDH, England) using the following systems (14). Solvent system I : n-hexane : diethyl ether : glacial acetic acid (60:40:1 v/v/v); Solvent system II : n-hexane : diethyl ether : glacial acetic acid (90:10:1 v/v/v). The spots were identified in an iodine chamber, scrapped out into glass tubes and individual fractions were eluted with 5 *ml* of chloroform. Individual neutral lipids were quantified by the methods as described earlier (6, 11).

Individual phosphatides were also separated by thin layer chromatography on silica gel G, using the solvent system of : chloroform : methanol : ammonia (7N) (115:45:7.5 v/v/v) as described by Abramson and Bleecher (1). Phosphatides were identified as blue spots by spraying molybdenum blue spray as proposed by Dittmer and Wells (6). The identified spots were scrapped quantitatively into test tubes and were eluted with a mixture of chloroform : methanol : formic acid : water (97:97:4:2 v/v/v/v). The phosphorous was quantified as described earlier (15). Data were statistically analysed using Students 't' test.

RESULTS

Table I depicts the effect of castration and testosterone propionate treatment on prostatic lipids in adult monkeys. Castration decreased total lipids ($P<0.05$), total phospholipids ($P<0.05$), total glyceride glycerols ($P<0.05$) and diacyl glycerol ($P<0.05$). In addition, among the phospholipids fractions, phosphatidyl choline ($P<0.05$) and phosphatidyl ethanolamine ($P<0.05$) were also decreased in the prostate of castrated monkeys. The other fractions were maintained without any change. Administration of testosterone propionate to castrates for 21 days brought back all the altered lipid classes to normal.

TABLE I : Influence of castration and testosterone propionate on prostatic lipid classes in mature monkeys, *Macaca radiata*.

Lipid class	*Control*	*Castrated*	*Castrated & TP treated (1 mg/kg, body weight)*
Total lipid	86.6±6.0	71.1±4.5*	99.2±5.0
Total phospholipid	19.6±1.0	15.2±1.2*	18.9±1.3
Total cholesterol	17.0±1.7	15.9±1.5	22.0±1.2
Total glyceride glycerol	50 0±3.3	40.9±3.0*	56.9±3.5
Monoacyl glycerol	18.0±1.4	16.3±1.3	18.1±1.6
Diacyl glycerol	22.0±2.0	16.9±1.4*	24.2H-1.9
Triacyl glycerol	10.0±0.5	8.0±1.0	14.0±1.0
Phosphatidyl inositol	2.0±0.1	2.0±0.1	2.0±0.1
Phosphatidyl serine	1.0±0.1	1.6±0.1	1.9±0.1
Sphingomyelin	1.2±0.1	1.0±0.1	1.6±0.1
Phosphatidyl choline	7.6±1.0	5.0±0.6*	8.2±1.7
Phosphatidyl ethanolamine	6.0±0.9	4.0±0.3*	6.1±0.6
Phosphatidic acid	1.2±0.1	1.8±0.4	0.99±0.1

Each value is mean ± S.E.M. of 10 estimations.
*$P<0.05$. Control Vs others.
All the values are expressed as *mg/gm* wet tissue.

Table II illustrates the effect of castration and TP treatment on the seminal vesicular lipids in mature monkeys. Seminal vesicular total lipids, total phospholipids ($P<0.05$), total cholesterol and total glyceride glycerol ($P<0.01$) were decreased in castrated monkeys. In addition, mono and diacyl glycerols ($P<0.001$) were also diminished after castration. TP to the castrates significantly increased total phospholipids ($P<0.001$) and total cholesterol ($P<0.01$) concentration above intact controls. The other lipid classes were brought back to normal after TP treatment in castrates.

TABLE II : Influence of castration and testosterone propionate on seminal vesicular lipid classes in mature monkeys, *Macaca radiata.*

Lipid class	*Control*	*Castrated*	*Castrated & TP treated (1 mg/kg, body weight)*
Total lipid	114.20±8.2	86.2±6.5*	130.12±7.9
Total phospholipid	26.26±1.2	20.0±2.0*	38.92±2.7***
Total cholesterol	22.0±1.8	16.0±0.9**	30.90±2.5**
Total glyceride glycerol	64.8±3.7	50.0±3.4**	62.04±3.8
Monoacyl glycerol	24.09±1.4	15.5±1.0***	23.08±1.8
Diacyl glycerol	28.09±1.6	18.0±1.0***	28.12±2.0
Triacyl glycerol	12.87±0.9	14.8±1.9	16.40±1.9
Phosphatidyl inositol	3.30±0.3	3.5±0.3	3.00±0.1
Phosphatidyl serine	2.30±0.2	2.5±0.2	2.90±0.2
Sphingomyelin	1.70±0.1	1.0±0.1	1.80±0.1
Phosphatidyl choline	9.60±0.7	6.0±0.4***	9.90±0.9
Phosphatidyl ethanolamine	7.50±0.4	5.9±0.3***	7.00±0.5
Phosphatidic acid	1.90±0.1	0.9±0.1	2.20±1.0

Each value is mean ± S.E.M. of 10 estimations.
*P< 0.05 **P< 0.01 ***P< 0.001 Control Vs others.
All the values are expressed as *mg/gm* wet tissue.

DISCUSSION

The data obtained in the present study show that there is considerable variation in the lipid composition of the prostate and seminal vesicles. The androgen dependency of the seminal vesicular and prostatic lipids in monkeys is clearly proved by the decreased lipids after castration and the testosterone induced increase of lipids in castrates. Beall (3) showed that stimulation of metabolic activity increases the esterification of acyl groups leading to an accumulation of glycerides in adult rat male accessory sex organs

The present study reveals that in monkeys also the phospholipid synthesis in prostate and seminal vesicles is androgen dependent as reported earlier in rats. Phospholipid synthesis may be associated with the enhanced membrane permeability, protein synthesis and increased secretory activity (2, 19). Phosphatidyl choline and phosphatidyl ethanolamine, the major structural component of the cell, have been implicated in membrane permeability (2). Hence the observed decrease in the phospholipid classes in castrates may result in reduced membrane transport in these organs.

The decreased concentration of phosphatidyl choline and phosphatidyl ethanolamine in castrates may be due to low availability of testosterone. This was confirmed by the normal levels of the phospholipid classes in TP treated castrates. Regarding cholesterol, the results are well in accordance with the earlier reports on rats (18) that testosterone has a stimulatory effect and leads to increased levels of cholesterol in the seminal vesicles and prostate.

The absence of any significant change in the level of triacyl glycerols with the obvious reduction in diacyl and monoacyl glycerol in the prostate and seminal vesicles of castrated monkeys suggest that the decrease in total glyceride glycerols is mainly due to the diminution in the levels of mono and diacyl glycerols. The data suggest that the synthesis of triacyl glycerol is not favoured by testosterone. The changes observed in total phospholipids of seminal vesicles and prostate were mainly due to phosphatidyl choline and phosphatidyl ethanolamine.

An overall perusal of the data on phospholipid fractions reveal that phosphatidyl choline and phosphatidyl ethanolamine may be more sensitive to androgens. Thus, the present study shows that testosterone in optimal amounts is essential for the maintenance of prostatic and seminal vesicular lipids. The exact mechanism by which testosterone induces such changes in the seminal vesicle and prostate is not clear at present and needs further study.

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