Epididymis plays a vital role in sperm maturation events. Hence, it has been considered as a most susceptible region for fertility regulation in males (see Bedford, 1975; see Voglmayr, 1975).

Thyroid disorders have been associated with fertility disturbances of varying order, depending upon the severity and duration of disease (see Longcope, 1986a,b; Buchanan et al., 1988; Pringle et al., 1988). Eventhough a number of recent reports indicated the modification in testicular structure and functions due to altered thyroid status (Bruni et al., 1975; Aruldhas et al., 1982a,b; 1983; 1986a,b; Chandrasekhar et al., 1985a,b; Valle et al., 1985), not much information is available on epididymis, baring a few reports from this laboratory and others (Pereira et al., 1982; 1983a,b,c; 1984; Del Rio and Quiros, 1983). Lipids being the important source of energy, structural component of the cells and secretory product of the epididymis (Turner and Johnson, 1971; see Brooks, 1980), the same is investigated in the present study under chronic hypo- and hyperthyroid conditions.

The data on serum thyroid hormones confirm the induction of hypo- and hyperthyroid status in the respective experimental animals. T4 replacement therapy (6 μg/100g body weight/day) to thyroidectomized rats for 30 days could
maintain euthyroid status. Similarly, withdrawal of T4 treatment from hyperthyroid rats for 30 days revealed euthyroid level of T4 and T3, suggesting reversibility of hyperthyroidism.

The data on epididymal weight in rats with modified thyroid status reveal some interesting aspects. Despite diminished serum testosterone, there was not much variation in the wet weights of caput or cauda epididymides. It is well known that androgens are the major regulators of epididymal structure and function (Amann, 1987; Robaire and Hermo, 1988). Epididymal tissue, fluid and sperm constitute the major components of the organ. The epididymal sperm count suggests a marginal decrease in hypothyroid rats but is not sufficient enough to modify the organ weight.

Androgens are transported to the epididymis, through the retetesticular fluid by the androgen binding protein (ABP) (Musto et al., 1980; Feldman et al., 1981). In rats, luminal ABP is needed for transepithelial movement of androgens in the epididymis (Turner et al., 1989). Thyroid hormones, particularly T3 was shown to inhibit ABP synthesis in the Sertoli cells of the testis (Fugassa et al., 1987), through its specific receptors present in the cell (Palmero et al., 1988). Hence, it is logical that the inhibitory effect of T3 on ABP production is removed or curtailed in
hypothyroid rats. A recent study conducted in our laboratory showed only a marginal decrease in intratesticular testosterone concentration in rats subjected to hypothyroidism for 60 days (Aruldas et al., unpublished data). Hence, it may be assumed that there is sufficient quantity of testosterone to be transported into the epididymis, despite low serum testosterone, through the retetesticular fluid by ABP. This may be attributed to the unaltered epididymal weight in hypothyroid rats in the presence of low serum testosterone.

Surprisingly, the epididymal weight in T₄ supplemented thyroidectomized rats showed a declining trend in their wet weights. Probably, the administered T₄ to thyroidectomized rats had an inhibitory effect on Sertoli cell ABP production. It is now well established that T₄ gets converted into T₃ at target cells to bring about its biological effects (see Kohrle et al., 1987; see Mendel, 1989). Since serum thyroid hormone status in these T₄ replaced thyroidectomized rats was at euthyroid level, one may expect only normal influence of thyroid in Sertoli cells. However, the data on epididymal weights reveal an obvious reduction in androgen status, despite a marginal increase in serum testosterone. Presumably, Sertoli cells of hypothyroid rats (30 days duration) might have had modified sensitivity of T₃ receptors and replacement of thyroid hormones has
resulted in augmented inhibitory effect plausibly. By extending the period of T4 replacement, the androgen status in the epididymis may regain normalcy.

Pereira et al., (1983b) have reported reduction in epididymal weight in rats subjected to hypothyroidism for 20 days and have correlated the same to decrease in serum testosterone. However, in the present study involving long term hypothyroidism, no such correlation could be evinced, inspite of diminished testosterone. Probably, during the course of prolonged hypothyroidism, the production of ABP by the Sertoli cells have been enhanced, resulting in a compensatory stimulatory effect of androgens, as discussed earlier. Thus, there appears to be a temporal influence of hypothyroidism on epididymal weight.

Further, Pereira et al. (1983b) have shown euthyroid level of epididymal weight in thyroidectomized rats given immediate replacement of T4. On the other hand in the present study, there was a marked reduction in the weight of epididymis of thyroidectomized rats given T4 replacement for 30 days, starting from the day 31, post thyroidectomy. Since immediate replacement may not permit any change in the thyroid hormonal influence on epididymis or testis, one can not expect similar results in animals subjected to hypothyroidism for 30 days and then given T4 replacement.
therapy. Therefore, the inhibitory effect of thyroid hormones on ABP production (Fugassa et al., 1987) may be responsible for the reduction in epididymal weight of thyroidectomized rats given T4 therapy, in the present study.

The data on the epididymal weight of hyperthyroid rats suggest an adverse effect and it appears to be irreversible within 30 days. The adverse effect of hyperthyroidism on epididymal weight, despite elevated serum testosterone may also be correlated to the inhibitory effect of excess thyroid hormones on ABP synthesis and secretion by the Sertoli cells (Fugassa et al., 1987). Intratesticular testosterone was also found to be reduced in these hyperthyroid rats (Aruldhas et al. un published data). Thus, the androgenic stimulation of epididymis might have been impaired, resulting in reduced proliferative and secretory activities. This, in addition to the marked reduction in sperm count, (please see table-3) might have contributed for the observed decrease in the wet weights of caput and cauda epididymides of hyperthyroid rats.

30 days of T4 withdrawal period appears to be not sufficient to reverse the adverse effect of hyperthyroidism on epididymal weights. Probably, the Sertoli cells may require more time to recover from the adverse effect of severe hyperthyroidism. This may be the reason that the
epididymis registered reduced weight even after the withdrawal of T₄ treatment which resulted in normal levels of serum and intratesticular testosterone.

Hyperthyroidism for short duration (20 days) was also found to have similar effect on epididymal weight (Pereira et al., 1983a). Nevertheless, these authors were able to get normal weight of the epididymis after 20 days of withdrawal period. Probably, the duration of hyperthyroidism play a significant role than that of the withdrawal period. This may be the reason that the rats of the present study (30 days hypothyroidism) could not maintain normal epididymal weight, even after 30 days of withdrawal period. Further Pereira et al. (1983a) have induced hyperthyroidism at the age of 60 days. On the otherhand, the same was induced in prepubertal rats (at 30 days post partum) in the present study, the period at which testicular testosterone production will be at the raise (see Eik-Nes, 1975), and the epididymis will be at an early phase of active growth. Since the hyperthyroid animals of the present study might have suffered with insufficient androgenic stimulation at the early stages of growth itself, the impact might be more severe than that of pubertal rats employed by Pereira et al. (1983a).

The data on other accessory sex organs weight suggest the non-existence of a correlation between them and serum
testosterone in hypo- and hyperthyroid organs. Unlike epididymis, the structure and function of prostate and seminal vesicles are markedly influenced by serum testosterone (see Cunha et al., 1987). Therefore, the absence of correlation between serum testosterone and the weights of these accessory sex organs may suggest modified androgen expression on these target organs. Submandibular glands, one of the target organs for androgens was shown to depend on thyroid hormones for the expression of androgen dependent activities (Minnetti et al., 1987). A similar mechanism may operate in prostate and seminal vesicles also.

Thus, the data on the weights of epididymis and other accessory sex organs suggest modified androgenic action due to hypo- and hyperthyroidism for a long period, induced at prepubertal age. This may be expected to interfere with the normal structural and functional aspects of these accessory sex organs.

Thyroid disorders, either hypo- or hyperthyroidism have been associated with varied degrees of infertility. Normospermia, oligospermia and azoospermia have been reported to occur in men and experimental animals with modified thyroid functions (Smeleser, 1939; Chandrasekhar et al., 1985a, b; see Ingbar, 1985; see Longcope, 1986a, b; Monson et al., 1988). The data on epididymal sperm count suggest
oligospermia in hypothyroid condition and severe oligosperma in hyperthyroidism. This may be due to impaired spermatogenesis in these animals. A number of early reports on rats and rams suggest disruption of spermatogenesis in hypo- and hyper thyroid rats and rams (Amin and El-Sheikh, 1977; Aruldhas, 1981; Chowdhury and Arora, 1984; Chandrasekhar et al., 1985 a,b).

The data also suggest that 30 days of recovery (T4 replacement to hypothyroid rats and withdrawal of T4 treatment from hyperthyroid rats) may not be sufficient to bring back normospermia. This may be mainly because of the adverse effect of hypo- and hyperthyroidism induced at prepubertal stage on testicular germ cells. Chowdhury and Arora (1984) reported that thyroidectomy of immature rats lead to degeneration of resting spermatocytes. In Wistar rats, the duration of spermatogenesis (time between the division of a A1 spermatogonia and release of spermatozoa) was calculated to be nearly 53 days (see Steinberger and Steinberger, 1974). Therefore, it is obvious that the signs of recovery from damages caused by hypothyroidism or hyperthyroidism on spermatogenesis can be visualised only after a prolonged period of convalescence. This may be the reason that the low count of epididymal sperm persisted event after 30 days of replacement therapy or withdrawal period in
hypo- and hyper thyroid rats, respectively. Priyadarsini (1989) from this laboratory also found low epididymal sperm count in the caput, corpus and cauda epididymides of rats subjected to short term (30 days) and long term (60 days) hypothyroidism.

Altered hypothalamo-hypophyseal-testicular hormone axis in hypo- and hyperthyroid rats and rams have been reported (Bruni et al., 1975; Aruldhas et al., 1982 a,b; Pereira et al., 1983 a,b; Chandrasekhar et al., 1985 a,b; Valle et al., 1985). This may be correlated to the observed reduction in the sperm count of hypo- and hyperthyroid rats. Further ABP is the carrier for testosterone from the interstitium into the seminiferous tubules to act upon the germ cells (Fawcett, 1975; see Means et al., 1976). Since thyroid hormones have an inhibitory effect on ABP synthesis in the Sertoli cells (Fugassa et al., 1987), diminished testosterone effect may also be expected on spermatogenic process, resulting in the production of less number of sperm.

The data on sperm motility also suggest adverse effects of both hypo-and hyperthyroidism. Epididymis occupies a pivotal role in sperm maturation process (see Bedford, 1975; see Orgebin-Crist et al., 1975). The binding of specific epididymal glycoproteins on sperm head provides fertilizing capacity and forward motility (see Orgebin-Crist et al.,
1975; Acott and Hoskins, et al., 1978; Brand et al., 1978) to sperm which otherwise will have only rotatory movement (Gaddum, 1968). The synthesis and secretion of this forward motility protein and other specific epididymal glycoproteins coating sperm are androgen dependent (see Orgebin-Crist et al., 1975,1987; Brooks, 1983; Brooks and Higgins, 1980; Hall and Killian, 1987) The testosterone transported by ABP is converted into DHT in the epididymis (Robaire et al., 1977; Scheer and Robaire, 1980; see Robaire et al., 1981) DHT is closely associated with forward motility protein synthesis (Orgebin-Crist et al., 1976; Orgebin-Crist and Jahad, 1978).

As discussed earlier, the androgenic stimulation of the epididymis may be impaired in hypo-and hyperthyroidism due to reduced synthesis and transport of testosterone. Therefore, the availability of testosterone to be converted into DHT in the epididymis may be low. Further, the binding of DHT to its specific receptors in the epididymis may also vary under altered thyroid status. This could be deduced from the finding of reduced concentration of androgen receptors in submandibular glands of hypothyroid mice (Minetti et al., 1986; 1987). The findings of these authors have clearly shown that dependency of androgen expression on submandibular glands, a target organ for these steroids (Minetti et al., 1987). Hence, the reduced synthesis of epididymal specific
proteins associated with sperm motility may be expected in rats with modified thyroid status.

Another factor associated with sperm motility and maturation is epididymal sialic acid (Prasad et al., 1970; Yanagimachi et al., 1972; Nicolson et al., 1977). A recent study conducted in our laboratory showed accumulation of sialic acid in the epididymal sperm of rats subjected to hypothyroidism for 30 and 60 days duration (Priyadarsini, 1989). Pereira et al., (1982) reported that hypothyroidism for 20 days is associated with decreased sialic acid content in the epididymal tissue and an opposite trend in hyperthyroid (20 days duration) rats. Therefore, the observed decrease in cauda epididymal sperm motility of hypo- and hyper thyroid rats may also be correlated to modified sialic acid levels in the epididymal environment. Epididymal sialic acid is also androgen dependent (Prasad et al., 1970; Gupta et al., 1974).

Carnitine and acetyl carnitine have also been implicated in sperm motility (Tanphaichitr, 1977; White et al., 1987). Epididymis derives carnitine from circulation and not from de novo synthesis (Casillas and Erickson, 1975). Thyroid hormones modulate the vascularization and thus blood supply to the epididymis. Hypothyroidism was reported to be associated with insufficient vascularization in the
epididymis of rats (Del Rio, 1979). Therefore, altered supply of carnitine to the epididymis can be expected in hypo- and hyperthyroid rats and this may also contribute for the reduced sperm motility observed in the present study.

The study of Priyadarsini (1989) revealed altered glycoprotein metabolism in the epididymal tissue and sperm of hypothyroid rats. She found that thyroid hormones have an inhibitory effect on epididymal glucosaminidase and galactosaminidase, and a stimulatory effect on glucosidase. Since the epididymal specific proteins are glycoprotein in nature (Lea et al., 1978; Moore, 1981; Olson and Danzo, 1981; Young et al., 1985) altered thyroid status may be suggested to inhibit sperm motility by modifying the composition of these epididymal glycoproteins.

Epididymal lipids also play an important role in sperm maturational events (Scott et al., 1967; Poulos et al., 1973; Evans and Setchell, 1978, 1979). Glycerides serve as the oxidizable energy for sperm (White et al., 1976; Evans and Setchell, 1979). In addition to their role in preserving the structure and characteristics of sperm plasma membranes, along with cholesterol, phospholipids also provide fatty acid side chains to be utilized by the sperm as energy source (Scott et al., 1967; Davis and Byrne, 1980a,b). Therefore, any alteration in epididymal lipids may also be reflected in
sperm motility. The data obtained in the present study suggest tremendous modifications in epididymal neutral and phospholipids (discussed in detail below) in hypo- and hyperthyroid rats. In view of the importance of lipids in sperm maturational activity, it is suggested that hypo- and hyperthyroidism might have diminished sperm motility by interfering in epididymal lipid metabolism, as well.

Thus, altered thyroid functions may impair sperm motility by modifying the composition and levels of proteins, glycoproteins, sialic acid, carnitine and lipids in the epididymal luminal fluid.

The data on caput and cauda epididymal lipids suggest marked influence of thyroid hormones on the same. The effect of thyroid hormones on epididymal lipids appears to be specific to various classes of lipids and epididymal region.

Accumulation of epididymal lipids has been attributed to non-utilization of the same (Turner and Johnson, 1971). Therefore, the accumulation of total lipid content in caput and cauda epididymides of hyperthyroid rats may be correlated to the diminished sperm count in these animals. Nevertheless, such a correlation could not be evinced between the decreased lipid content of hypothyroid rats and their sperm concentration. A careful analysis of the data may suggest
only a marginal decrease in sperm count (about 20%) in hypothyroid rats which persisted even after replacement of T4 to hypothyroid rats. However, the epididymal lipid content depicted a decrease due to hypothyroidism which was reversible by T4 replacement. Probably, the change in epididymal lipid of hypothyroid rats is not due to changes in their utilization, it may be due to a dip in the synthesis itself.

The decrease in total lipid content of caput epididymidis in hypothyroid rats is mainly due to decrease in glyceride glycerols and major phospholipid classes, i.e. phosphatidyl choline, phosphatidyl ethanolamine, sphingomyelin, phosphatidyl serine and phosphatidic acid. On the other hand, in cauda epididymidis, the decrease in total lipid was a contribution from neutral lipids only. Thus, there appears to be a regional difference in the influence of hypothyroidism on epididymal lipid metabolism.

Glycerides are the major source of oxidizable energy for the epididymal sperm (Evans and Setchell, 1979), apart from phospholipids (Scott et al., 1967). Therefore, the marginal but significant decrease observed in the motility of caudal sperm from hypothyroid rats may be attributed to the decreased source of oxidizable energy from glycerides. On the other hand, in hyperthyroid rats perceptible decrease in sperm motility was evident despite increased lipid content.
Probably, other factors associated with sperm motility are affected than lipids, in hypothyroid rats, as discussed earlier. Since the sperm count also was declined, it is assumed that the accumulation of lipids in hyperthyroidism is due to non-utilization. However, a look at the data on various classes of glyceride glycerols and phospholipids may suggest enhanced synthesis also as a factor responsible for the accumulation of lipids in the epididymis of hyperthyroid rats. In view of the accumulation of neutral and phospholipids in the epididymal tissue, there is a possibility of diminished secretion into the epididymal luminal fluid and thus decreased availability of lipids for sperm in the luminal fluid, resulting in reduced motility/fertility.

A proper ratio of cholesterol : phospholipid is essential for the optimal secretory activities of epithelial cells (see Benedetti and Emmelot, 1968; Grunze and Deuticke, 1974; Phillips and Finer, 1974). Since there is accumulation of cholesterol and phospholipids in the epididymis of hyperthyroid rats, altered membrane characteristics may be expected. The cholesterol : phospholipid ratio also suggests a trend of decrease in hyperthyroid rats. Cholesterol reduces the fluidity of phospholipid acyl chains and inhibit membrane transport activities as indicated by inhibition of Na\(^+\)-K\(^+\)-ATPase (Kimelberg and Papahadjopoulos, 1974). A recent study
conducted in the author's laboratory revealed modified activities of caput and cauda epididymal Na\(^+\)-K\(^+\)-ATPase, Ca\(^{2+}\)-ATPase and Mg\(^{2+}\) ATPase due to hypo- and hyperthyroid conditions for 60 days (Rose, 1989). This may provide further support for the suggestion of altered secretory activities in the epididymis of rats with modified thyroid status.

The pattern of cholesterol in hypo- and hyperthyroid rats also indicates regional difference in the caput and cauda epididymides. In caput epididymidis, hypothyroidism-induced increase in total cholesterol was mainly due to elevated free cholesterol suggesting enhanced mobilization of cholesterol. On the other hand, free cholesterol alone was diminished in the cauda, suggesting an adverse effect of hypothyroidism on cholesterol synthesis and mobilization. There is also a possibility of increased secretion of cholesterol in the cauda epididymidis.

Eventhough the T\(_4\) replacement to thyroidectomized rats, total cholesterol was maintained at normal level in caput and cauda epididymides of T\(_4\) supplemented hypothyroid rats, the mechanism varies. While both free and esterified cholesterol were at normal level in the caput, increased esterification of free cholesterol was evident in the cauda epididymidis. This may sugest euthyroid status of cholesterol metabolism in the caput epididymidis of T\(_4\) supplemented hypothyroid rats but illproportionate
mobilization and utilization of cholesterol in the cauda region.

Similarly, the increase of caput epididymal cholesterol in hyperthyroid rats was consistently evident in free and esterified fractions. The same pattern was also persisting in $T_4$ withdrawn hyperthyroid rats. On the other hand, predominance of esterification was evident in cauda epididymidis, leading to accumulation of cholesterol in hyperthyroidism. In hyperthyroid rats withdrawn of $T_4$ treatment also, the predominance of esterification was evident but was accompanied with a decrease in the free form. Thus, regional differences in the ratio of free and esterified cholesterol is evident in the epididymis of rats subjected to modified thyroid status.

In normal animals, esterified cholesterol was found to be the major fraction in the caput and the opposite was evident in the cauda. This pattern was reversed in the cauda epididymidis alone in hypo- and hyperthyroid rats. Thus, increased esterification appears to be the major change in cauda epididymidal cholesterol under altered thyroid status. On the other hand, increased mobilization of both free and esterified cholesterol was the feature seen in the caput epididymidis, particularly under hyperthyroid condition. In caput epididymidis, only hypothyroidism produced an increase
in the ratio of free : esterified cholesterol. Due to the uniform increase in both free and esterified cholesterol in T₄ treated groups, there was no change in their ratio. 30 days of recovery period appears to be not sufficient to maintain normal ratio of free : esterified cholesterol, in hypo- and hyperthyroid rats.

In contrary to the present finding, short term hypo- or hyperthyroidism (20 days) did not bring about any significant change in epididymal cholesterol of pubertal and adult rats (Pereira, 1983a,b). This may suggest that epididymal cholesterol undergoes changes only in chronic hypo- or hyperthyroid conditions.

The data on glyceride glycerol suggest inhibition of synthesis in caput epididymidis of hypothyroid rats. This could be evinced from the finding of significant decreases in mono-, di- and total glyceride glycerols in the caput epididymidis of these animals. On the otherhand, in cauda epididymidis, the synthesis of triacyl glycerol from mono- and diacyl glycerol seems to be favoured during hypothyroid condition.

Diacyl glycerol is the precursor for both, triacyl glycerol and phospholipids, particularly phosphatidyl choline, phosphatidyl ethanolamine and phosphatidyl serine (see Mayes, 1988). Therefore, any modification in glyceride
glycerol synthesis can be expected to be reflected in phospholipids also. In hypothyroid rats, there was a concomitant decrease in phosphatidyl choline and phosphatidyl ethanolamine along with mono- and diacyl glycerols. Hence, it becomes obvious that the decrease in glyceride glycerols is mainly due to decreased synthesis, rather than a shift in their utilization. The available diacyl glycerol might have been utilized for the synthesis of triacyl glycerol, to maintain it at normal level.

On the other hand, in cauda epididymidis the triacyl glycerol pathway is favoured with normal pattern of diacyl glycerol phospholipid pathway. There appears to be no appreciable increase in mono- and diacyl glycerol synthesis, in association with enhanced triacyl glycerol formation. This may be the reason for the diminution in mono- and diacyl glycerols in this epididymal region of hypothyroid rats.

T₄ supplementation to hypothyroid rats was able to maintain normal total glyceride glycerol in the caput region. However, the ratio of different fractions of glyceride glycerols was modified. There appears to be diminished formation of phospholipids and a marginal decrease in triacyl glycerol formation, resulting in the accumulation of monoacyl glycerol in the caput epididymidis.
On the otherhand, in cauda epididymidis of T4 supplemented hypothyroid rats, the synthesis of triacyl glycerols and phospholipids seem to be enhanced. This may be deduced from the observed increase in triacyl glycerol, phosphatidyl choline and phosphatidyl inositol with a decrease in diacyl glycerol.

There appears to be increased turnover of glyceride glycerols in caput and cauda epididymides of hyperthyroid rats. Both, diacyl glycerol-triacyl glycerol pathway and diacyl glycerol - phospholipid pathway might be active in caput and cauda epididymides of hyperthyroid rats. This could be deduced from the finding of increased triacyl glycerol along with elevated phosphatidyl ethanolamine (only in caput) phosphatidyl choline and phosphatidyl serine with diminished or normal diacyl glycerol levels.

Withdrawal of T4 treatment from hyperthyroid rats did not bring about any discernible change, except for the specific reduction in diacyl glycerol - phosphatidyl ethanolamine pathway, which remained normal in hypothyroid rats. Thus, the accumulation of epididymal triacyl glycerol in hyperthyroid rats may be attributed to increased synthesis from diacyl glycerol, apart from non-utilization of the same by epididymal sperm, as discussed earlier. Along with triacylglycerol, phospholipid synthesis also seems to be favoured in hyperthyroid condition.
Short term hypothyroidism (20 days) in pubertal rats was shown to be associated with augmentation of triacyl glycerol, phosphatidyl choline and phosphatidyl ethanolamine formation from diacyl glycerol, in the caput epididymidis (Pereira et al., 1983c). On the other hand in the present study, glyceride glycerol synthesis, itself was adversely affected with concomitant diminution in phospholipid fractions. Further, Pereira et al., (1983c) could not find any change in cauda epididymidal glyceride glycerols or phospholipids. The same authors have also reported diminution of triacyl glycerol synthesis from diacyl glycerol in the caput epididymidis of pubertal rats subjected to short term (20 days) hyperthyroidism, without any change in phospholipids (Pereira et al., 1984). In short term hyperthyroid rats also cauda epididymidal neutral and phospholipids remained static. Thus, the influence of modified thyroid status on epididymal lipid metabolism appears to be temporal and age-dependent.

Apart from phosphatidyl choline, phosphatidyl serine and phosphatidyl ethanolamine, other phospholipid fractions also undergo changes in the epididymidis of rats subjected to chronic hypo- and hyper thyroidism. Comparatively, the caput epididymidal lipids appear to be vulnerable to changes in thyroid status than that of cauda epididymidis. This is more true in hypothyroid rats than that of other groups.
While caput epididymidal phospholipids, with the exception of phosphatidyl inositol and cardiolipin, were adversely affected by hypothyroidism, cauda epididymidal phosphatidyl inositol, phosphatidyl ethanolamine and phosphatidic acids were specifically favoured in hypothyroid rats. This may suggest that the influence of hypo- and hypothyroidism is specific to the classes epididymal of phospholipids with regional difference.

In general, the data on phospholipid fractions suggest a specific stimulatory effect of thyroid hormones on all phospholipid classes coupled with diacyl glycerol pathway (phosphatidyl choline, ethanolamine and inositol) in caput epididymidis. Invariably, all phospholipid fractions are favoured in hyperthyroidism. As discussed earlier, non-utilization may also be the reason for such a trend, apart from enhanced synthesis.

Androgens are the major regulators of epididymal lipid metabolism (Umapathy et al., 1980). This study also revealed the varying specific influence of testicular androgens on different classes of epididymal neutral and phospholipids. Therefore, modified androgen status may also be attributed to the varying changes in different fractions of epididymal lipids in the present study.
The many fold increase in phosphatidyl choline observed in hyperthyroid rats may facilitate 5-α reductase activity and thus increased availability of DHT in the epididymis (Cooke and Robaire, 1985). Since DHT is the potent androgen associated with the growth and functions of epididymis (see Orgebin-Crist et al., 1975), it may be expected to enhance cell proliferation. Therefore, the synthesis of phospholipids and cholesterol which are associated with membrane structure (see Hamilton 1975; Orgebin Crist et al., 1976) may also be enhanced.

Apart from androgens, prolactin may also modify epididymal lipids (Jayakumar, 1989) through its specific receptors (Aragona and Friesen 1975). Further, hypothyroidism was found to be associated with increased prolactin receptors in the ventral prostate (Kharroubi and Slaunwhite, 1984). Therefore, it is logical that altered thyroid status may modify prolactin action on its target organs. Hyperprolactinemia was shown to have an inhibitory effect on caput epididymal mono- and triacyl glycerols, and phosphatidyl choline and ethanolamine in monkeys (Jayakumar, 1989). In the present study also, hypothyroidism was associated with similar trend in caput epididymal phosphatidyl choline, phosphatidyl ethanolamine and monacyl glycerol. Hypothyroidism may be accompanied with hyperprolactinemia (Buchanan et al., 1988) and this might
have also contributed for the observed decrease in these lipid classes in the caput epididymidis of hypothyroid rats, in the present study. Nevertheless, it needs further confirmative studies to arrive at a definite conclusion on these lines.

Thus, the present study suggests that epididymal lipid metabolism undergo significant modification due to chronic hypo- and hyperthyroidism. The secretory activity of epididymis may also undergo changes due to modified lipid status, particularly cholesterol: phospholipid ratio. This may lead to impaired sperm motility, due to reduction in the availability of forward motility protein and metabolic energy from lipids. Hence, thyroid disorders may be suggested to alter male fertility through modified epididymal lipid metabolism and sperm motility.