CONCLUDING REMARKS
HYPOTHYROIDISM

Hypothyroidism was found to exert varied effects on the uterine metabolism concerned with glycogen and its associated enzymes, depending upon the presence or absence of sex steroids. In rats with ovaries intact, hypothyroidism appeared to deplete all the enzymes markedly but glycogen was unaltered. This may especially be due to the influence of hypothyroidic condition on the ovarian steroidogenesis (Leather, 1972). Hypothyroidism in ovariectomized rats resulted in an increase in glycogen and all the enzymes studied. The reason for such an increase may be due to the augmented secretion of estrogens from the adrenals, which is normally the case in the absence of ovaries, as well as due to the retention of available estrogens. More or less, similar results have been observed in ovariectomized but estradiol-treated rats, rendered hypothyroidic. However, no increase in glycogen, as observed earlier, was evident. Probably the glycogen utilization due to estrogenic stimulation might have resulted in glycogen depletion. The discrepancy in the glycogen level between ovariectomized and estradiol-treated rats may be due to the differences in the estrogen titres. The estrogen titres from the adrenal source may not be as much as that of exogenously administered.

Progesterone was found to cause an opposite effect compared to estradiol, with reference to glycogen and its enzymes,
This may be due to the facilitation of progesterone action on the uterus, in hypothyroidic state (Freeman et al., 1959). In estradiol plus progesterone treated rats, a mixed effect has been found. Here, the glycogen breakdown may be involved through the pentose phosphate pathway, as the glycogen depletion was found to be associated with an enhancement of G6PDH.

**HYPERTHYROIDISM**

Hyperthyroidic condition, resulted in the glycogen breakdown in all the groups except the intact rats. It appears that the glycogen synthesis is favoured in intact rats due to thyroxine administration. Since thyroxine is a hypermetabolic hormone in general, the uterus may result in glycogen breakdown. Since there was no increase in the glycolytic enzymes in the present studies, it appears that there may be a latent period for the full reflection of activities connected with glycogen utilization. More or less, similar results were obtained in ovariecotomized as well as estradiol-treated rats. In spite of the availability of estradiol, their effect on the uterus appears to be much less and this may be due to the increased catabolism of estrogens, by the thyroxine (Leatham, 1972). Progesterone in hyperthyroidic rats may favour glycogen breakdown through pentose phosphate pathway. Hypothyroidism, in the presence of estradiol and progesterone, appears to bring about rather a mixed effect of estradiol and progesterone. Since these two
hormones are antagonistic when given together, neither of their specific effects have been shown. However, in general, they have a common effect on glycogen depletion which may be due to reduced synthesis of glycogen.

HYPOADRENALISM

Irrespective of the status of the animal and the availability of sex steroids, hypo adrenalism resulted in the depletion of glycogen due to reduced synthesis, since almost all the enzymes concerned with glycogen breakdown were found to decline markedly. However, the presence of progesterone appears to favour Embden-Meyerhof pathway for glycogen breakdown which was quite marked compared to all the experimental groups. Even in the presence of oestradiol, the progesterone influence on Embden-Meyerhof pathway was evident, to a certain extent, since an increase in PHI was observed. In all the experimental groups of hypoadrenalic rats, protein synthesis appears to be encouraged. Such an encouragement may be due to increased amino acid transport thereby, resulting in increased protein synthesis (Kostyo and Redmond, 1966).

HYPERADRENALISM

The same picture, as obtained in hypoadrenalism was evident in hyperadrenalism also, except proteins and alkaline phosphatase, which were found to decrease. This may be due to the interruption of hydrocortisone in the amino acid transport
and protein synthesis. Apart from these, protein catabolism was reported to be more in hyperadrenalism (Jewell and Hunter, 1972).

**LDH ISOENZYMES**

Thiouracil administration, caused an increase in the M-subunits (LDH-5) by the enhanced estrogenic effects on the uterus in ovariectomized and estradiol-treated rats. The H-subunits (LDH-1) were also found influenced by thiouracil administration, which may be due to the influence of progesterone as its action was facilitated in hypothyroidism. The reduced levels of M-subunits, in intact rats may be due to the decrease in their synthesis.

The decreased levels of H-subunits in hypoadrenal, intact rats may be due to the reduced progesterone secretion by the ovary. The profound reduction in the M-subunits, observed in rats treated with hydrocortisone may be due to less estrogenic influence.

Certain extra bands associated with LDH-2 and 4 were exclusively seen only in intact rats rendered hypothyroidic or hypoadrenal. These bands appear to be specific only to the altered conditions of thyroid and adrenal. The isolation and characterization of such bands are being attempted. These bands have never been associated with estrogen or progesterone administration.
The shift towards the cationic isoenzymes in the various experimental conditions suggests an increase in the anaerobic isoenzymes which is in consonance with the increased glycolytic activity in the tissues.

Therefore, from the above studies, it could be seen that altered status of thyroid as well as adrenal interfere with the uterine metabolic activities. Such an influence depends upon the availability of the sex steroids as well as the status of the ovary. The direct effect of hypo and hyperthyroidism, or hypo and hyperadrenalism on the uterine metabolic activities may be much less. Any effect of thyroid or adrenal might be by altering either the metabolism of sex steroids or altering the uterine sensitivity to the available sex steroids.

In reproductive disorders associated with the altered conditions of thyroid or adrenal, an investigation into the uterine metabolic activities may be helpful, in correcting certain pathophysiological conditions.