SUMMARY
The influence of hypo and hyperthyroidism as well as hypo and hyperadrenalism on the uterine metabolism in intact, ovarioctomized, ovarioctomized plus estradiol, plus progesterone, plus estradiol and progesterone treated rats, have been investigated. Uterine glycogen and the associated enzymes, PH₅, aldolase, LDH, MDH, G6PDH and alkaline phosphatase as well as protein have been taken into consideration, as parameters to assess the influence of thyroid and adrenals. The LDH isoenzymes have also been studied.

In intact rats, thiouracil-induced hypothyroidism caused a marked decrease in proteins and the enzymes studied. Glycogen was not much altered. Hypothyroidism may favour the influence of progesterone but affects estrogen biosynthesis. The altered uterine metabolism may be due to the above factors.

Hyperthyroidism in intact rats, resulted in an increase in uterine glycogen, LDH and G6PDH. However, a decrease in uterine PH₁, aldolase, alkaline phosphatase and proteins were observed. Hyperthyroidism has been shown to facilitate estrogen influence and discourages progesterone effect on the uterus.

Hypothyroidism in ovarioctomized rats as well as ovarioctomized plus estradiol treated rats, induced marked increases in all the parameters studied. The facilitatory influence of hypothyroidism in retaining estradiol, supplemented by the adrenals, in the absence of ovaries as well as estradiol administered exogenously might have caused the alterations in the uterus.
Hypothyroidism in ovariectomized plus progesterone treated rats, decreased markedly all the parameters except PH1, which exhibited an increase. This may be due to the facilitation of progesterone action on the uterus in hypothyroidic state. Progesterone appears to initiate Embden-Meyerhof pathway for glycogen breakdown.

Hypothyroidism, in ovariectomized rats treated with estradiol plus progesterone induced a decrease in uterine glycogen, proteins and alkaline phosphatase activity. A marked increase in MDH activity was also observed. It appears that the influence was essentially due to the mixed effects of estradiol and progesterone which are antagonistic to each other. Glycogen breakdown may be through the citric acid cycle since MDH activity was selectively increased.

In ovariectomized as well as ovariectomized plus estradiol-treated rats, hypothyroidism induced a marked decrease in glycogen, the associated enzymes, alkaline phosphatase and proteins. This effect appears to be due to the increased catabolism of estrogen resulting in low synthesis of glycogen.

Hypothyroidism in ovariectomized rats treated with progesterone, induced a decrease in uterine glycogen. The decrease in glycogen was due to its utilization through pentose phosphate pathway, since a selective increase in G6PDH was evident.
The decrease in glycogen was less in ovariectomized hyperthyroidic rats treated with estradiol plus progesterone. In these groups, the altered parameters were suggestive of the mixed influence of estradiol and progesterone.

Hypo and hyperadrenalism invariably affected the uterine glycogen synthesis by depleting the same, quite markedly. The depletion may be due to decreased synthesis rather than utilization, as the enzymes of glycogen breakdown were also found to decline along with glycogen. Hypoadrenalism may encourage protein synthesis whereas hyperadrenalism may inhibit the same.

The isoenzyme pattern of LDH, suggested certain extra bands in ovariectomized but estradiol treated rats, irrespective of the altered conditions of thyroid or adrenals, which appears to be specific to estrogen influence. However, in intact rats, rendered hypothyroidic or hypoadrenalic, more extra bands were observed than in ovariectomized plus estradiol treated rats. It appears that these bands are specifically associated with the altered conditions of thyroid or adrenal.