S U M M A R Y

SUMMARY

CHAPTER II

Gravimetric and histoarchitectural studies were carried out under induced chronic hypocorticalism [by two doses of dexamethasone: low dose - DXM(L) and high dose - DXM(H)] and hypercorticalism (by corticosterone - CORT) in male White Leghorn breed of chicks during the first 30 days of development. All the treatments significantly retarded body weight gain. Both DXM(L) and DXM(H) reduced the absolute as well as relative weight of the adrenals while CORT tended to increase the relative weight. CORT treatment also decreased the relative weight of liver and kidney. All treatments induced a reduction in the absolute and relative weight of spleen and bursa of Fabricius. There was significant increase in the weight of testes in DXM treated chicks while a reverse effect of decreased weight was the feature in CORT treated chicks. Both the DXM doses induced regressive changes in the adrenal cortex with a concomitant reduction in the cortico-medullary ratio. CORT treatment did not alter the cortico-medullary ratio; though a tendency for hypertrophy of cortical cells could be seen. The testis of DXM(L) treated chicks tended to show better organized seminiferous cords and interstitium with lumenation in many of the cords. Such progressive changes were not discernible in DXM(H) and CORT treated chicks. All the steroid treatments induced a stimulatory change on the thyroid gland marked by increased follicular epithelial cell height and decreased colloid content. These changes are taken to indicate that adrenal corticoids do exert definite influence on post-hatched growth and maturation of various organs in chicks and the observations are discussed in detail in the text.

CHAPTER III

Alterations in carbohydrate metabolism in terms of tissue glycogen contents, phosphorylase activity, hepatic glucose-6-phosphatase activity and blood gluocse have been evaluated in 30 day old White Leghorn chicks under induced chronic hypocorticalism (by dexamethasone-DXM) and hypercorticalism (by corticosterone). DXM treatment showed increased tissue glycogen contents and hypoglycemia with decreased phosphorylase activity while corticosterone treatment produced reverse set of changes. Both the steroid treatments increased hepatic G-6-Pase activity. These observations have been taken to indicate a definite role for glucocorticoids in regulating carbohydrate metabolism in neonatal chicks. It is suggested that hypo- or hypercorticalism influence carbohydrate metabolism by affecting the secretory/activity ratio of pancreatic hormones.

CHAPTER IV

Alterations in tissue protein and ascorbic acid (AA) contents have been assessed in 30 day old White Leghorn chicks under induced chronic adrenocortical insufficiency (by dexamethasone - DXM) and excess (by corticosterone). Corticosterone treatment increased the AA content and decreased protein content in all the tissues while DXM treatment decreased AA content in adrenal and testis and increased the protein content in liver and muscle. These results are discussed in terms of the gluconeogenic action of corticosteroids with protein as the source and also on the possible influence on protein synthesis and/or break-down. Further, it is presumed that hypercorticalism can induce a positive AA balance while hypocorticalism can have reverse influence and induce negative AA balance.

CHAPTER V

Changes in total lipids and cholesterol fractions in tissues and serum of 30 day old White Leghorn chicks with induced chronic hypocorticalism (with dexamethasone-DXM) and hypercorticalism (with corticosterone) have been evaluated. Adrenal lipid content was increased with DXM treatment and decreased with corticosterone treatment while the serum lipid content was increased under both treatments. In the adrenal, the content of total and esterified fractions of cholesterol was increased with DXM low dose and decreased with DXM high dose while only free cholesterol was decreased with corticosterone. In liver, both total and esterified fractions of cholesterol were increased with both DXM treatments while free cholesterol was reduced with only DXM low dose. Corticosterone increased only the total cholesterol content significantly. Significant increase in all the cholesterol fractions was the feature under DXM treatment while only free cholesterol content was increased under corticosterone treatment. Based on these observations it has been concluded that lipid content may probably serve as a better index in assessing steroidogenic activity of adrenal and that DXM may have a dose dependent effect on ACTH release with concomitant effect on cholesterol metabolism of adrenal by way of differential sensitivities of enzymes of cholesterol metabolism to ACTH levels. It has also been concluded that both DXM and corticosterone are potent in inducing lipid and cholesterol biogenesis by liver with the former being more potent. The increased steatogenesis and fat deposition observed have been discussed in terms of correlated permissible alterations in thyroid and pancreatic hormones at the mechanistic level.

CHAPTER VI

Effect of induced chronic adrenocortical deficiency (by dexamethasone - DXM) and excess (by corticosterone) on certain glycolytic and oxidative enzymes (Aldolase, LDH and SDH) has been assessed in 30 day old White Leghorn chicks. The activity levels of all the three enzymes were reduced in liver and muscle of DXM treated chicks while aldolase and SDH activities were increased and LDH decreased in corticosterone treated chicks. Aldolase and LDH activities were increased in the testis with DXM treatment and SDH activity decreased with corticosterone treatment. These alterations in enzyme activity have been correlated with suppression of both glycolytic and oxidative metabolism in liver and muscle under hypocorticalism. Further, hypocorticalism induced stimulation of glycolytic metabolism in testis is also considered to provide a favourable influence on its post-natal growth and maturation.

CHAPTER VII

Effect of chronic dexamethasone (DXM) induced hypocorticalism and corticosterone induced hypercorticalism on activity levels of ATPases, phosphomonoesterases and phosphodiesterase (PDE) has been assessed in 30 day old White Leghorn chicks. Muscle ATPase activity did not show much change with any of the treatments, while hepatic total and Ca^{++}-Mg^{++}-ATPase activity were decreased with low dose of DXM and enzyme activity in general was increased with both high dose of DXM and corticosterone. Total and Ca^{++}-Mg^{++}-ATPases were increased in testis of corticosterone treated chicks. Acid phosphatase activity of testis was increased with DXM(H) and decreased with DXM(L) while the enzyme

activity in all the three tissues was increased with corticosterone. Muscle alkaline phosphatase activity was decreased with DXM treatments while that of testis was decreased with both DXM(H) and corticosterone treatments. Hepatic PDE activity was decreased with DXM and increased with corticosterone while muscle PDE activity was decreased under both DXM(H) and corticosterone treatments. Based on the prsent observations it has been concluded that both hypo- and hypercorticalism can induce tissue specific differential alterations in phosphomonoesterases, ATPases and PDE during early phases of post-natal development of chick which are discussed in the text in terms of related observations.

CHAPTER VIII

Alterations in histochemical distribution and localization of steroid dehydrogenases and lipids in the testis of 30 day old White Leghorn chicks with chronic hypocorticalism induced by dexamethasone (DXM) and hypercorticalism induced by corticosterone have been studied. Reduced content of lipids and increased activity of 3β- and 17β-hydroxy steroid dehydrogenases and decreased 3∝-HSDH activity in both seminiferous cords and interstitium were the feature in chicks treated with low dose of DXM. On the other hand both high dose of DXM and corticosterone treatments induced more or less reverse set of changes. It has been concluded from the observations that adrenocortical insufficiency in the neonatal period could hasten up the process of tubular differentiation and organization by way of testosterone production within the seminiferous cords and increased local build up of progesterone and androstenedione. The observations also suggest that adrenocortical excess

could delay the process of testicular maturation by preventing the build up of the above steroids. These aspects are discussed in detail in the text.

CHAPTER IX

Alterations in glucose tolerance and, insulin, glucagon and adrenaline responses in dexamethasone (DXM) treated (induced hypocorticalism) chicks have been evaluated chronologically during the first month of post-hatched development of White Leghorn breed of chicks. Definite alteration in glucose tolerance and, insulin resistance and glucagon and adrenaline responses in dexamethasonised chicks have been recorded. Moreover, even control chicks depicted differential glucose tolerance and, insulin, glucagon and adrenaline responses on a chronological basis. The observations taken as a whole suggest increased insulin and adrenaline sensitivity in the first fortnight and gradually increasing glucagon response in the second fortnight in neonatal chicks. Further, increased insulin sensitivity and attenuated glucagon responsiveness have also been associated with DXM induced hypocorticalism. These aspects have been discussed in detail in the text taking into consideration other relevant related observations.