CHAPTER VI

EFFECT OF INDUCED ADRENOCORTICAL DEFICIENCY AND EXCESS ON SOME GLYCOLYTIC AND OXIDATIVE ENZYMES (ALDOLASE, LDH AND SDH)IN POST-HATCHED WHITE LEGHORN BREED OF CHICKS

Changes in the activities of enzymes involved in carbohydrate, lipid and amino acid metabolisms occurring in embryonic and growing chicks have been documented (Goodridge, 1968; Raheja et al., 1971a). These studies have illustrated occurrence of adaptive alterations in the enzyme activities in keeping with the metabolic adjustments required due to transitions in developmental phases. Similar changes in metabolic strategy occurring during the course of post-natal development in mammals (rat) have also been recorded (Beebee and Carty, 1983).

Such changes in metabolic activities during post-natal development could be considered to be under the purview of endocrine secretions and could well be considered a crucial phase during vertebrate development as both metabolic feature and hormonal balance are in the process of maturation and acquisition of the characterestic adult pattern. In this context, exogenous hormones or suppression of endocrine glands cause disturbances in the overall physiological homeostasis at this curcial phase of avian development more than in the adult condition. Investigations on influences of such hormonal manipulations have been restricted to a single observation with reference to PTU induced hypothyroidism during post-natal chick development (Raheja et al., 1971b) and another on effect of thyroxine in adult Japanese quail (Koneka and Majewska, 1981).

Previous chapters have demonstrated definite alterations in various biochemical and metabolic parameters due to induced adrenocortical suppression and excess during post-natal development of White Leghorn breed of chicks. In the present study, alterations in activity levels of aldolase, LDH and SDH in liver, muscle and testis of dexamethasone (hypocorticalism) and corticosterone (hypercorticalism) treated chicks have been evaluated to ascertain the possible impact of corticoid lack or excess on glycolytic and oxidative metabolisms.

MATERIAL AND METHODS

As outlined in Chapter I.

RESULTS

Activity levels of Aldolase, LDH and SDH in liver, muscle and testis

The results obtained depicted in Table 1; Figs. 1 & 2 revealed significant reduction in the activity levels of aldolase, LDH and SDH in the liver and muscle of adrenocortical suppressed chicks compared to that of control chicks. Corticosterone treated chicks showed a significant increment in the activities of aldolase and SDH in liver and muscle while LDH was decreased significantly in the case of liver and non-significantly in the case of muscle.

The activity levels of the enzymes showed a variable response in testis.

Table 1 Enzyme activity levels of aldolase, LDH and SDH in liver, muscle and testis of chicks treated with dexamethasone and corticosterone for 30 days.

	Aldolase μ moles FDP cleaved/ mg protein/60 min			LDH μ moles lactate oxidised/ mg protein/15 min			SDH μg formozan formed/ mg protein/30 min		
	LIVER	MUSCLE	TESTIS	LIVER	MUSCLE	TESTIS	LIVER	MUSCLE	TESTIS
CONTROL	0.481 ±0.038	0.404 ±0.033	0.502 ±0.029	381.00 ±21.37	581.85 ±21.41	203.50 ±15.17	26.06 ±1.92	17.04 ±1.29	25.98 ±1.70
DXM(L)	0.272[a] ±0.020	0.280[b] ±0.016	0.724[a] ±0.028	248.12[a] ±13.30	412.20[a] ±10.36	232.96 ± 7.15	14.39[a] ±0.68	9.75[a] ±0.91	29.04 ±1.09
DXM(H)	0.140[a] ±0.023	0.276[b] ±0.006	1.025[a] ±0.077	230.21[a] ± 7.37	417.83[a] ±23.94	328.93[a] ±19.41	14.05[a] ±0.61	6.84[a] ±0.64	30.37 ±2.15
CORT	0.966[a] ±0.054	1.359[a] ±0.055	0.479 ±0.029	294.39[b] ±15.45	532.86 ±12.99	224.61 ±19.76	34.14[b] ±1.66	23.58[d] ±1.89	19.47[b] ±1.29

DXM(L) : dexamethasone low dose; DXM(H) : dexamethasone high dose; CORT : corticosterone
Values are mean ±SE of not less than 8 birds.
a : $P < 0.001$; b : $P < 0.01$; d : $P < 0.05$

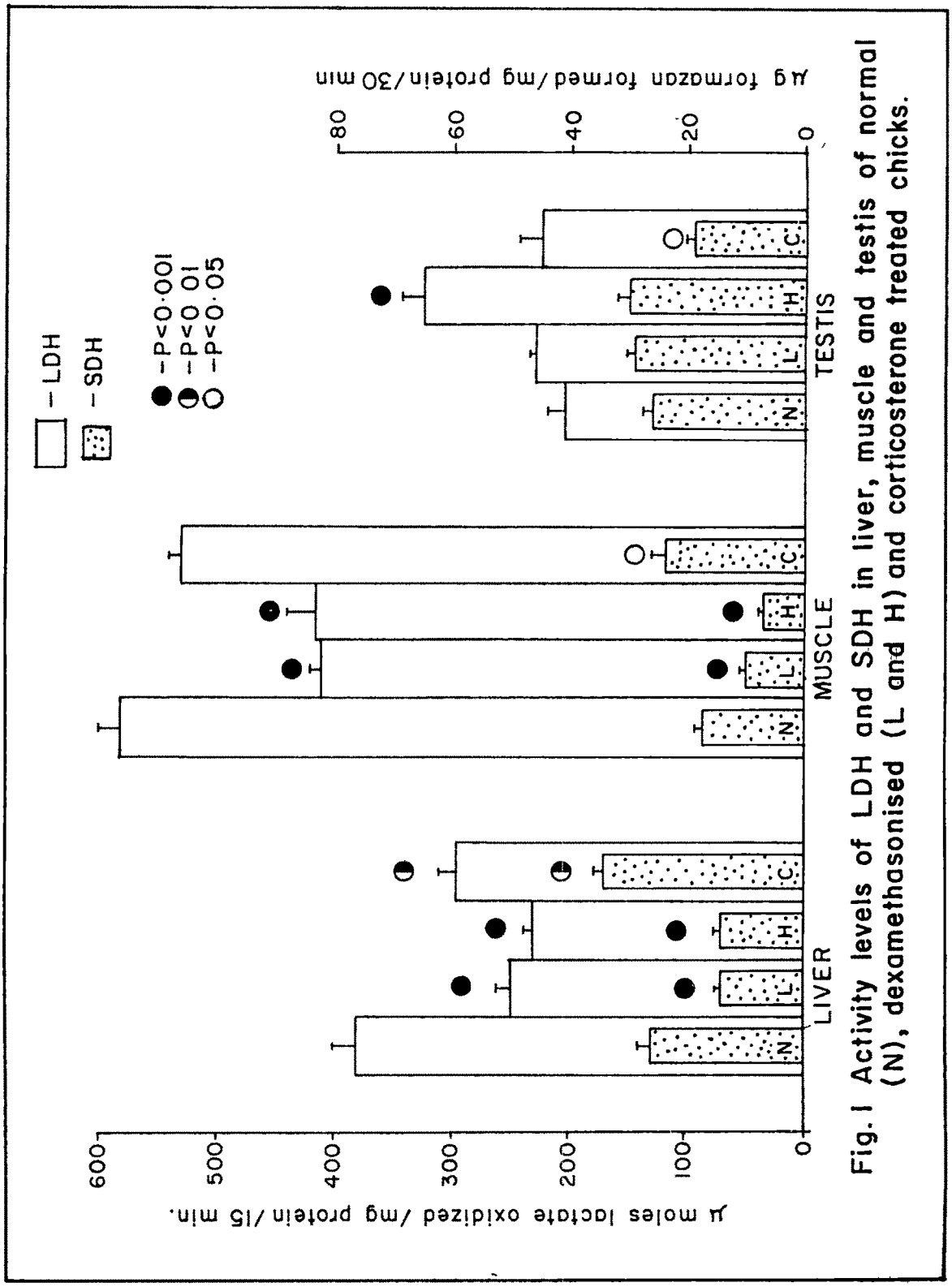

Fig. 1 Activity levels of LDH and SDH in liver, muscle and testis of normal (N), dexamethasonised (L and H) and corticosterone treated chicks.

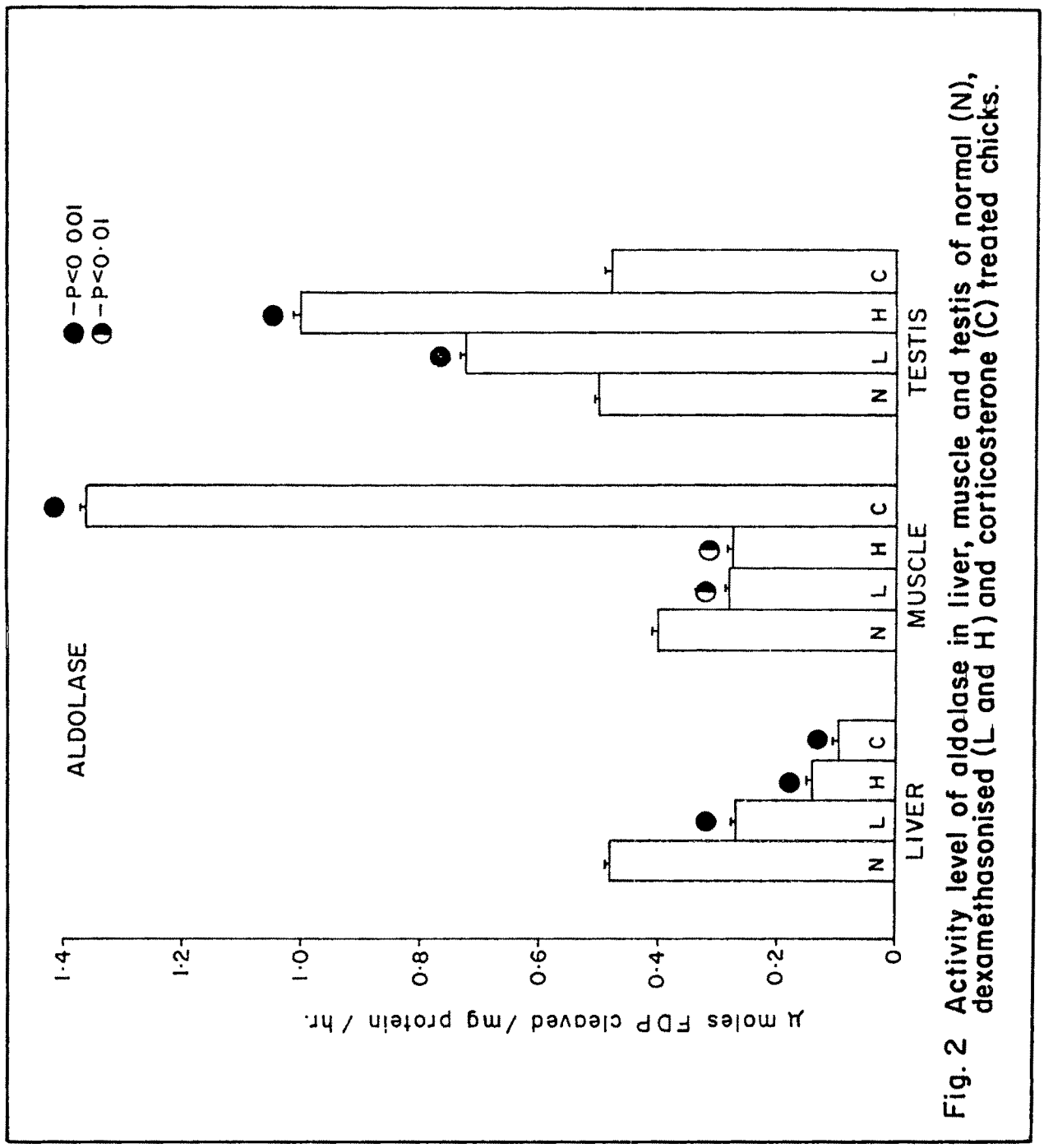

Fig. 2 Activity level of aldolase in liver, muscle and testis of normal (N), dexamethasonised (L and H) and corticosterone (C) treated chicks.

There was an increase in the activity levels of Aldolase and LDH in dexamethasone treated chicks without any alteration in SDH activity.

Whereas Aldolase and LDH activities remained unaltered, SDH activity was significantly decreased in corticosterone treated chicks.

DISCUSSION

The present observations indicate suppression of both glycolytic and oxidative metabolisms in the liver and muscle of DXM treated chicks suggesting the importance of corticosterone in maintaining glycolytic and oxidative metabolisms in post-hatched chicks. Importance of corticosterone in oxidative metabolism and oxygen uptake of various tissues in rat has been documented (Bottoms and Goetsch, 1968). The decrease in the activity levels of both aldolase and SDH in liver and muscle of chicks treated with both doses of DXM bear testimony to this. The decrease in total LDH activity observed in these two tissues of DXM treated chicks suggest an overall decrement in both glycolytic (M type) and oxidative (H type) isozymic forms of the enzyme. This is corroborated by the reported correlation between cortisol level and serum LDH activity in human subjects (Rehiman et al., 1986) which has been correlated with increased oxidative LDH isozymic forms. The less significant decrement in total LDH activity of corticosterone treated chicks in the present study may be indicative of reduction in glycolytic form of LDH isozyme only. This is in contrast to the recent report of an increase in LDH enzyme activity and decrease in SDH activity

in liver of DXM treated rats and decrease in both LDH and SDH activities with corticosterone treatment (Amando et al., 1988). The above workers have suggested a functional duality of the two types of glucocorticoids at the hepatic level in rat, and the present observations tend to corroborate the same but in reverse sense, as in chicks both LDH and SDH activities were decreased under DXM treatment and SDH was increased and LDH decreased under corticosterone treatment. Possible alterations in LDH isozymic forms suggested herein due to manipulation in adrenocortical activity of post-hatched chicks is strengthened by the reported developmental changes in total LDH content of liver and kidney of rat neonates during the first 56 days (Beebee and Carty, 1983). Their study involving both in vivo and in vitro methods of evaluating LDH subunits had indicated regulation of LDH activity to occur at some point(s) during both their biosynthesis and degradation. The decreased glycolytic and oxidative metabolisms of liver and muscle in DXM induced hypoadrenocorticalism as observed in the present study is well reflected in the observed decrease in phosphorylase activity and increased glycogen content (Chapter III) and increased lipid content (Chapter V). However, the increased SDH activity in the liver and muscle of corticosterone treated chicks in the wake of increased lipid content (Chapter V) and depleted glycogen and protein contents (Chapter III & IV) tend to suggest increased oxidation of glycogen and protein reserves. Whereas previous observations (Chapter II, III, & V) purport that both DXM and corticosterone have a common action in decreasing physiologically active T_3 generation, present observations indicate that corticosterone has a direct action

in inducing SDH activity in liver and muscle of chicks in spite of the fact that thyroid hormone has been generally accepted as the inducer of SDH activity (Lee and Lardy, 1965; Hoch, 1974; Hulbert, 1976).

In contrast, the changes in testicular enzyme activities appear to be of a differential nature. The increased aldolase and LDH activities in the testis of DXM treated chicks with no alteration in SDH activity could suggest increased glycolytic metabolism. Though the increased testicular glycogen content and decreased phosphorylase activity under this condition (Chapter III) do not favour this postulate, the prevailing hypoglycemic condition tends to suggest plasma glucose as the choice metabolite. This metabolic activation of the testis is well reflected in the advanced maturational changes affecting the testis (Chapter II & VIII). The reverse change of decreased SDH activity together with unaltered aldolase and LDH activities in the testis of corticosterone treated chicks tend to corroborate the fact that corticosterone excess in the early post-natal development is inhibitory to testicular development (Chapter II & VIII) and metabolism. These are in contrast to the reported influence of DXM on rat testicular and epididymal enzyme activities (Valivullah et al., 1981, 1983, 1985; Balasubramanian et al., 1987). In all the above studies, DXM has been shown to be acting as corticosterone while, in the present study on chicks, DXM and corticosterone seem to have differential effect in keeping with the purported function of DXM as inducing adrenocortical suppression and corticosterone inducing adrenocortical excess. The present results therefore tend to suggest DXM induced adrenocortical suppression to be more favourable

in inducing testicular development and differentiation in chicks while corticosterone administration has a retardatory effect. Apparently, native corticosteroids from the adrenal cortex somehow retard testicular development and differentiation in the early post-natal development of chicks. In this context, it is reasonable to ponder as to whether DXM induced adrenocortical insufficiency in post-hatched chicks has a favourable influence on FSH/PRL release or their action? Apparently, corticosterone seems to have a maturation delaying effect on testis of immature chicks as has been noted with reference to other observations as well in the present study (Chapters II, IV, V, VI, VII & VIII).