CHAPTER X

Photoperiod-Adrenal interactions on carbohydrate metabolism in the feral pigeon during the recrudescent phase.

Adaptive seasonal alterations in carbohydrate metabolism have been reported to occur in pigeons (Patel et al.,1983; Ramachandran and Patel,1987). In addition, many other factors like pinealectomy (Patel et al.,1983, 1988; Ramachandran and Patel, 1987), pCPA and pineal indoles (Chapter II), induced functional alteration of adrenal (Ayyar,1987) as well as long photoperiod (Chapter VIII), have all been shown to affect carbohydrate metabolism. It is also seen that functional alteration of adrenal and photoperiod has significant effects on testicular recrudescence in the pigeon (Chapter IX). Since it has been observed that tissue glycogen content and blood glocose level vary between breeding and non-breeding phases (Patel et al.,1983),it was thought pertinent to study the effect of photoperiod - adrenal interaction on carbohydrate metabolism. Moreover, there is a total lack of information on metabolic response to such experimental manupulations despite the fact that the factors which regulate annual gonadal cyclicity may be intimately related with alterations in overall metabolic strategy in seasonal animals.

Materials and Methods :

Procurement and maintenance of pigeons as outlined in chapter I,

Lighting and lighting schedules- as outlioned in chapter VIII.

Parameters and methodology of evaluation - as outlined in chapter II and

Experimental set-ups & preparation of corticosterone (CORT.)and Dexamethasone (DXM) as outlined in Chapter IX.

Results : (Table 10.1, Fig 10A,B)

Blood glucose and hepatic and muscle glycogen.

The blood glucose level which was higher during the recrudescent phase tended to increase under LD 18:6. However, CORT treatment in birds exposed to either NLD or LD 18: 6 decreased the blood glucose level with, the decrease being more pronounced in the latter. However, DXM treatment increased the blood glucose level in both NLD as well as LD 18:6 birds. The hepatic and muscle glycogen contents were significantly lowered in all experimental groups.

Hepatic phosphorylase and G-6-P'ase :

Treatments	Blood Glucose (mg/dl)	Tissue Glycogen (mg/100mg Tissue)		Hepatic G-6-P'ase (u moles PO_4 released /mg Protein/15 min)	Hepatic Phosphorylase (ug PO_4 released/mg Protein/15 min)
		Liver	Muscle		
N 12:12	199.43 ± 11.05	6.44 ± 0.29	0.52 ± 0.07	0.87 ± 0.05	10.83 ±1.61
N 18:6	203.99 ± 21.00	2.60 ± 0.12	0.15 ± 0.02	1.70 ± 0.08	4.66 ± 0.71
CORT 12:12	184.97* ± 18.93	2.65* ± 0.15	0.11* ± 0.01	1.25* ± 0.06	4.94* ± 0.47
CORT 18:6	153.11* ± 17.91	1.52* ± 0.08	0.28* ± 0.04	1.70 ± 0.07	7.06* ± 0.95
DXM 12:12	208.98 ± 21.55	4.22* ± 0.25	0.33* ± 0.06	0.94 ± 0.05	5.93* ± 0.30
DXM 18:6	219.23 ± 19.19	2.30* ± 0.15	0.39* ± 0.03	1.02* ± 0.04	5.51* ± 0.37

Table 10.1 : Alterations in blood glucose,tissue glycogen content and activity levels of G-6-P'ase and phosphorylase in pigeons treated with CORT or DXM and exposed to NLD or LD 18:6 in the pre-recrudescent phase .
(* = Significant at $P < 0.05$; values are $\bar{x} \pm SD$)

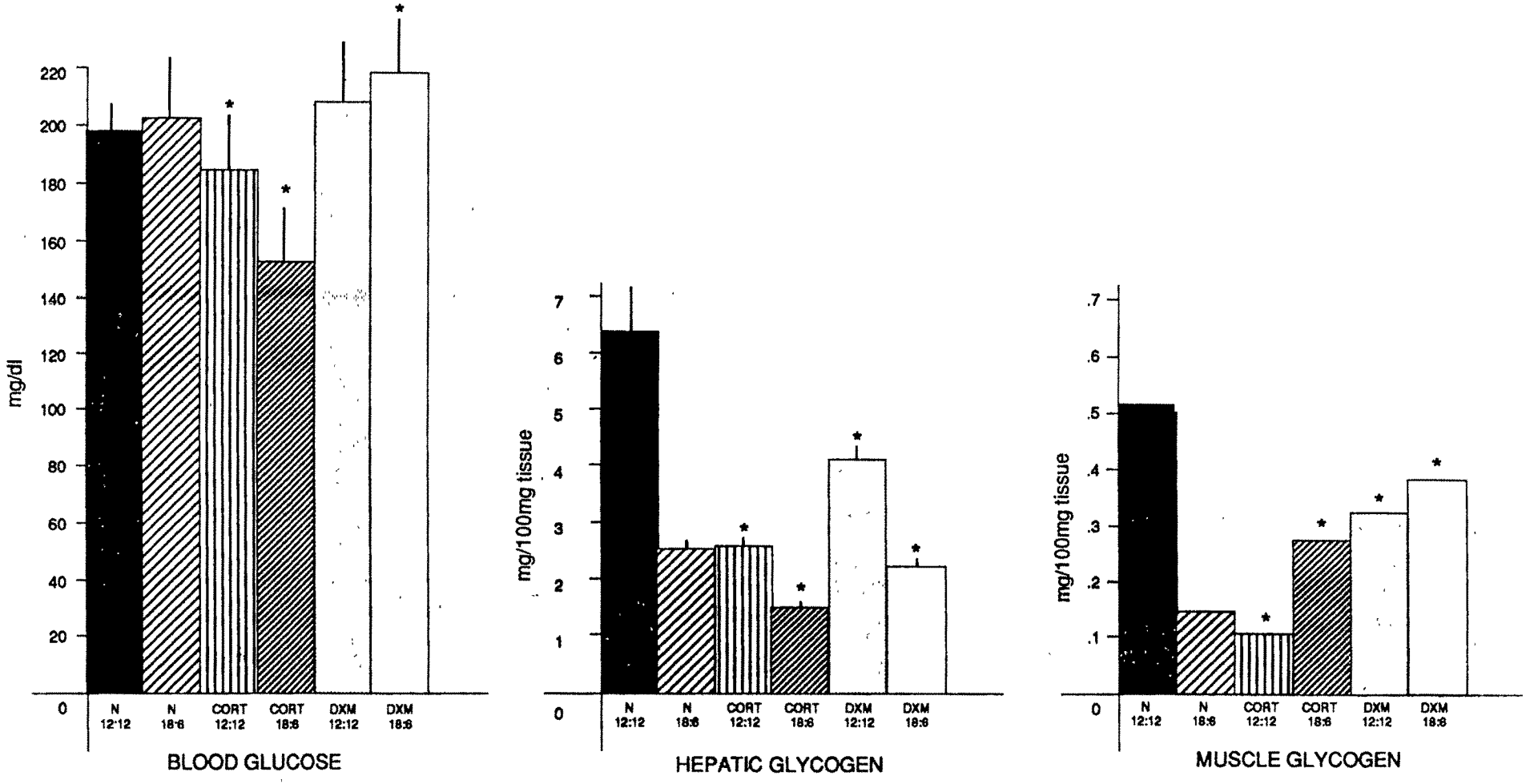

Fig. : 10.A: Alterations in blood glucose and tissue (hepatic and muscle)glycogen content of pigeons treated with CORT or DXM and exposed to NLD or LD 18:6 in the pre-recrudescent phase.
(* = Significant at $p < .05$; values are $\bar{x} \pm$ SEM)

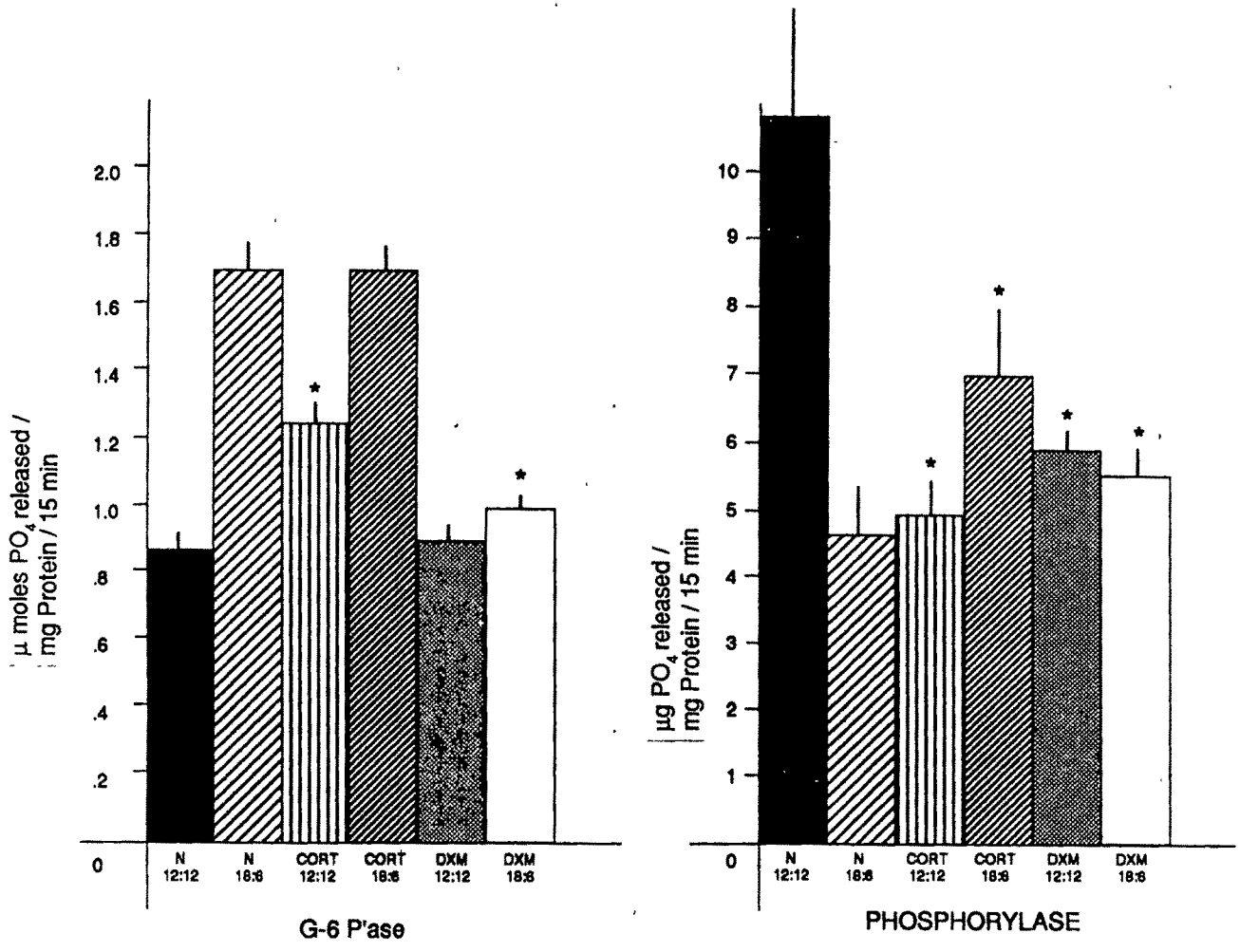

Fig. 10 B : Alterations in the activity levels of hepatic G-6 P'ase and phosphorylase of pigeons treated with CORT or DXM and exposed to NLD or LD 18:6 in the pre-recrudescent phase.
(* Significant at $p < 0.05$; values are $\bar{x} \pm SD$)

All the experimental set-ups registered decrease in phosphorylase activity and increase in G-6-P'ase activity.

DISCUSSION :

It is evident from the present results that long photoperiod tends to decrease tissue glycogen content and elevates blood glucose, changes characteristic of active phase of gonad (Patel et al.,1983). Utilization of tissue glycogen in various bodily functions associated with gonadal recrudescence seems to be an important feature in the pigeon. Though,this aspect is evident in all experimental set-ups in the present study, chronic treatment with DXM or CORT seems to have differential effect on glycemic level. Whereas CORT caused hypoglycemia, DXM caused hyperglycemia with either change being more pronounced in LD 18:6 than in NLD. Apparently photoperiod seems to potentiate the effect of both, DXM and CORT on the glycemic status. In view of the known gluconeogenic role of corticosterone, the presently recorded hypoglycemic effect of CORT and hyperglycemic action of DXM are apparently contradictory. However, it may be pertinent to keep in mind the fact that the effect on glycemic level due to either chronic adreno-cortical excess or insufficiency are likely to be different from that due to either acute or short term treatment schedules. The possibility of chronic adreno-cortical excess or

insufficiency altering the sensitivity or functions of the hypothalamic glucoregulatory centre cannot be discounted. Confirmation to this thinking comes from previous studies from this laboratory showing hypoglycemia due to DXM treatment and hyperglycemia due to CORT treatment for only 15 days (Ayyar,1987).

The increased G-6-P'ase activity recorded in the present study by CORT or DXM treatment seems to be due to a common effect of both in inducing G-6-P'ase activity as inferred earlier (Joseph and Ramachandran, 1992).

Though the reduced glycogen contents and reduced phosphorylase activity may appear contradictory, it is likely that the reduced enzyme activity is consequent to decreased glycogen content. Presumably,the earlier periods may have had higher phosphorylase activity leading to rapid glycogen depletion and with the attainment of low glycogen content the phosphorylase activity also decreased to establish a steady state. Apart from the earlier discussed reasons for the observed glycemic changes due to CORT or DXM treatment, alterations in other aspects like food intake, absorption, and tissue uptake of glucose and peripheral utilization also need to be considered.

Overall, it can be concluded that :-

1. Stored tissue glycogen is utilized during gonadal recrudescence,
2. Increased photoperiod potentiates the same
3. Chronic adreno-cortical suppression or activation have altered effects on glycemic status and,
4. These effects of DXM or CORT are potentiated under long photoperiod.