Chapter VI

INFLUENCE OF CORTICOSTERONE OR GONADOTROPHIC HORMONES ALONE OR IN COMBINATION, ON CARBOHYDRATE METABOLISM IN FERAL PIGEONS IN THE QUIESCENT PHASE.

Previously it was shown that circannual variations in carbohydrate metabolism could be related to the reproductive phases in the male pigeons (chapter III).In keeping with the positive relationship between adrenal and testes in the pigeon, adrenocortical activity and serum CORT level were shown to parallel testicular activity (Patel et al.,1986; Ramachandran and Patel, 1987). Functional manipulation of adrenocortical activity was seen to alter carbohydrate metabolism such that, hypo-corticalism in the breeding season lowered blood glucose level and increased tissue glycogen content and, hypercorticalism in the regression and quiescent phases elevated blood glucose level and decreased tissue glycogen content, changes which are characteristic of regression and recrudescence respectively (chapter III). These observations tended to confirm the importance of CORT as an important metabolic hormone related to testicular functions in the pigeon. In the previous investigation, it was revealed that during the quiescent phase, the testes retained sensitivity to gonadotrophic hormones as, administration of gonadotrophic hormones induced testicular

activation and further the importance of CORT in the same was also highlighted (chapter V). In this respect, it was imperative to see whether testicular activation induced by gonadotrophic hormones and corticosterone are paralleled by concomitant changes in carbohydrate metabolism. Hence in the present study the influence of CORT or gonadotrophic hormones administrated either alone or in combination has been assessed in terms of blood glucose level, tssue glycogen contents and hepatic phosphorylase and G-6-P'ase activities.

MATERIALS AND METHODS

Procurement, maintenance of pigeons and methodology of evaluation are as outlined in chapter III.

Experimental setups are as outlined in chapter V.

RESULTS.

Blood glucose: The blood glucose level in the control birds was 158.29 $\pm$ 5.16 mg/dl. Treatment with CORT or FSH.LH induced significant hyperglycemia. However, treatments with FSH or CORT + FSH (P or S) induced hypoglycemia.
(Table VI; Figs 6A to 6C)

Tissue glycogen content : Hepatic glycogen depletion was the

Treatments	Blood glucose (mg/dl)	Tissue glycogen (mg/100mg tissue			Hepatic Phosphorylase (μg PO_4 released /mg Protein/15 min	Hepatic G-6-P'ase (μ moles PO_4 released/mg Protein/15 min)
		Liver	Muscle	Testis		
Control	160.29 ± 4.98	5.11 ± 0.33	1.84 ± 0.03	0.26 ± 0.04	13.82 ± 1.18	0.12 ± 0.001
CORT	170.81* ± 5.52	4.13* ± 0.13	2.00* ± 0.02	0.28 ± 0.01	17.78* ± 1.84	0.23* ± 0.01
FSH	142.61* ± 2.28	3.34* ± 0.12	1.96* ± 0.14	0.05 ± 0.001	11.98 ± 1.40	0.13 ± 0.002
FSH.LH	172.61* ± 2.82	4.54* ± 0.61	1.87 ± 0.14	0.15 ± 0.004	14.02 ± 1.87	0.10 ± 0.001
CORT+FSH(P)	154.18 ± 4.84	3.49* ± 0.30	2.17* ± 0.02	0.10 ± 0.003	12.61 ± 1.21	0.12 ± 0.001
CORT+FSH(S)	143.55* ± 1.04	4.86* ± 0.73	2.62* ± 0.06	0.14 ± 0.01	11.91 ± 0.81	0.19* ± 0.01
CORT+FSH.LH	179.21* ± 4.64	3.92* ± 0.27	1.85 ± 0.04	0.13 ± 0.01	15.62* ± 0.62	0.18* ± 0.02

Table VI. Alterations in blood glucose, tissue glycogen content and activity levels of hepatic phosphorylase and G-6-P'ase of pigeons treated with gonadotrophins alone or with corticosterone in the quiescent phase.
(* Significant at $P < 0.05$; values are $\bar{x} \pm$ SEM)

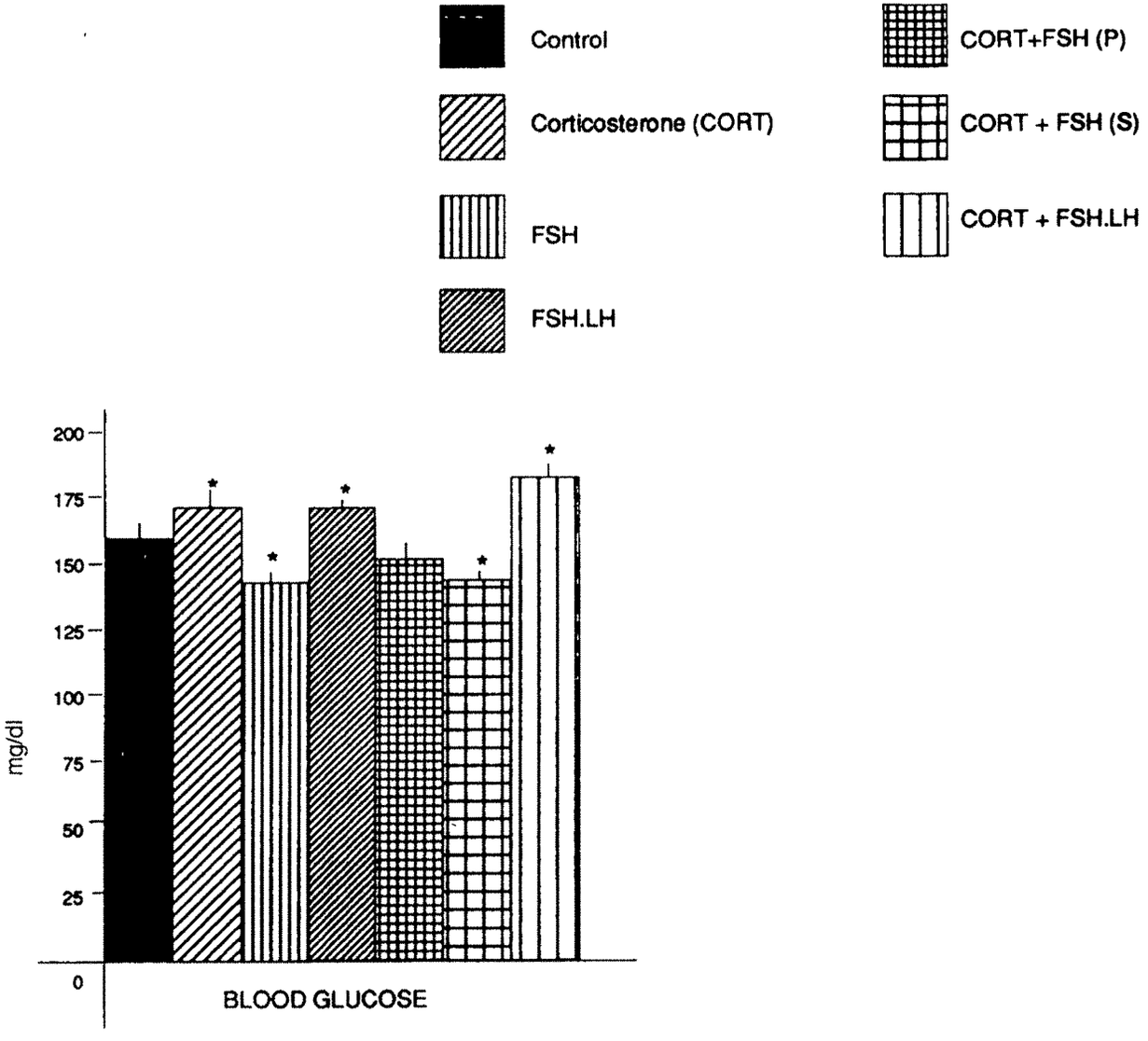

Fig 6 A · Alterations in blood glucose level of pigeons treated with gonadotropins alone or with corticosterone (CORT) in the quiescent phase (* Significant at $P < 0\ 05$, values are $\bar{x} \pm$ SEM).

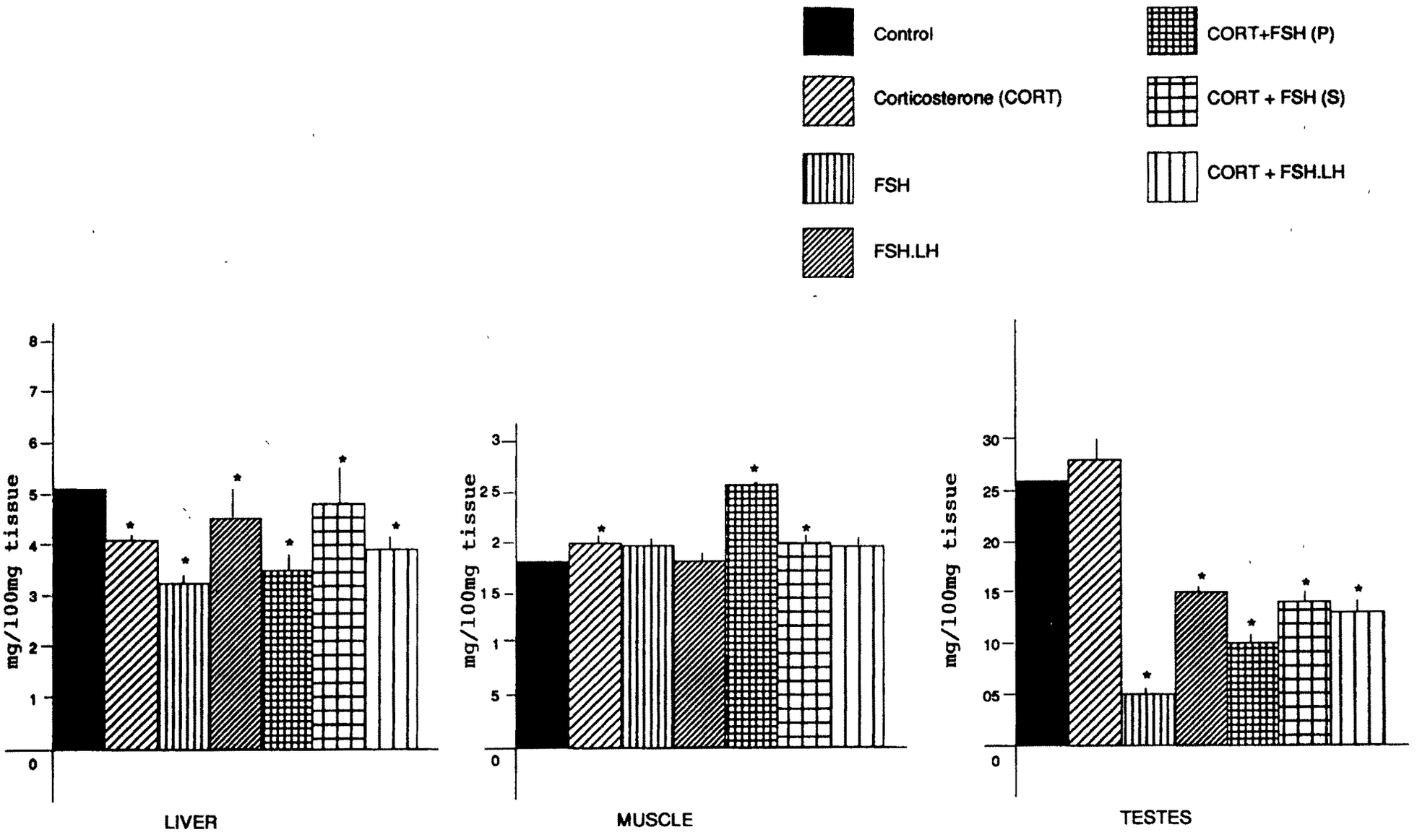

Fig. 6 B Alterations in tissue glycogen contents of pigeons treated with gonadotropins alone or with corticosterone (CORT) in the quiescent phase.
(* Significant at $P < 0.05$; values are $\bar{x} \pm$ SEM)

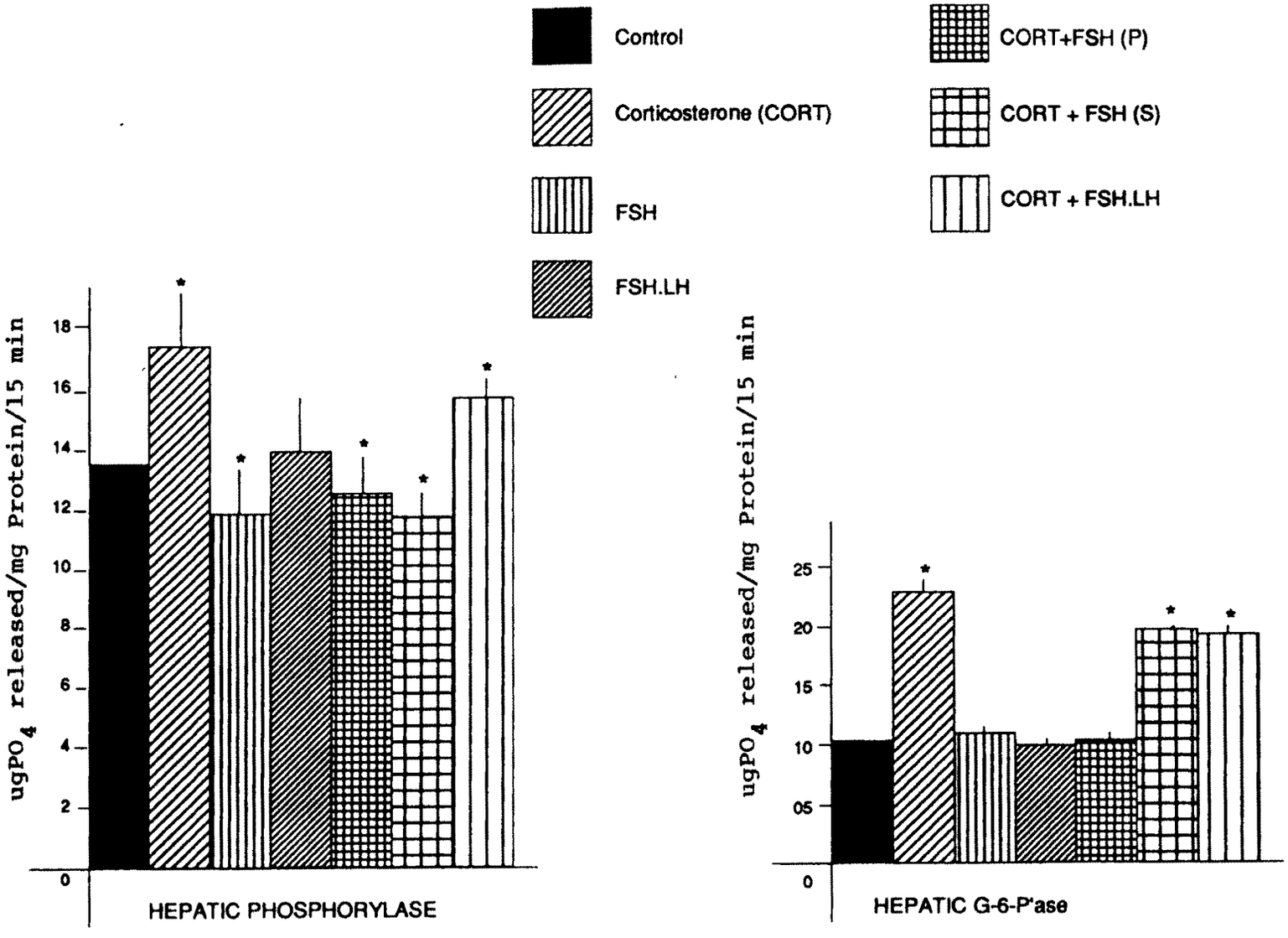

Fig. 6 C . Alterations in activity levels of hepatic phosphorylase and G-6-P'ase of pigeons treated with gonadotropins alone or with corticosterone (CORT) in the quiescent phase (* = Significant at $P < 0.05$; values are ± SEM)

common feature under all experimental schedules. Treatments with CORT or CORT + FSH (P or S) increased muscle glycogen content significantly, while there was no change under other experimental manipulation. Testis glycogen content decreased under all experimental conditions with a maximum decrement occurring under FSH treatment.

Hepatic phosphorylase and G-6-P'ase :

Hepatic phosphorylase activity decreased under all experimental conditions except in CORT treatment where there was an increase. G-6-P'ase activity was also increased under all experimental conditions except in FSH.LH and CORT + FSH (P) under which there was no significant change.

DISCUSSION.

The role of corticosterone in glucose homeostasis and carbohydrate metabolism has been well studied in mammals (Sistare and Haynes, 1985). Alterations in carbohydrate metabolism have been recognised to occur under both adrenal insufficiency (Ivarsson et al.,1983; Kitabachi et al., 1986) and hypercorticalism (Cann and Fajans, 1956; Kitabachi et al.,1973; Olefsky and Kimmerling, 1976). The influence of corticosterone on carbohydrate metabolism in birds has been essentially studied in the domestic species, especially the

poultry birds. Most of these studies have remained restricted largely to an understanding of the influence of CORT on growth of organs, or the body as a whole or, fat content (Nagra et al.,1963; Mangnall and Bartey, 1973; Magdi and Huston, 1974; Bartov et al.,1980; Freeman, 1983; Brake et al.,1988; Kafri et al.,1988). In contrast, there are only few studies designed to understand the role of CORT on carbohydrate metabolism (Davison et al.,1983; Simon, 1984; Saadoun et al., 1987). In this context, the present study as well as the previous ones from this laboratory have revealed a state of hypoglycemia and tissue glycogen deposition under induced hypocorticalism and vice-versa under hypercorticalism (Ayyar, 1987). The elevated serum CORT level and decreased tissue glycogen contents coupled with elevated blood glucose level in the recrudescent phase of pigeons have been viewed as a positive influence of CORT on carbohydrate utilization during gonadal recrudescence (chapter III).Though the metabolic effect of CORT is understandable, it is for the first time that the metabolic actions of the gonadotrophins are being revealed in the present study. Interestingly, a combination of FSH.LH could induce hypoglycemia and hepatic glycogen depletion much like CORT. However, though FSH alone also caused hepatic glycogen depletion, the glycemic level was lowered. This hypoglycemic action of FSH was evident even in those cases where it was given in combination with corticosterone. The elevated

phosphorylase and G-6-P'ase activity with CORT treatment is in keeping with the known ability of CORT to induce these enzymes (Exton et al.,1973; O'Neill and Langslow, 1978; Ballard,1979; Pierluissi et al.,1986; Natarajan, 1987) and correlate well with the observed hepatic glycogen depletion and hypoglycemia. It was previously shown that FSH and FSH.LH either given alone or in combination with CORT in the quiescent phase could induce testicular recrudescence, though a combination of CORT and FSH.LH had better quantitative effect (chapter V). The observed hepatic glycogen depletion in response to gonadotrophic hormones in the present case is understandable as, such a feature was related with testicular recrudescence during the annual cycle of the pigeon (chapter III). However, the exact mode of action of gonadotrophic hormones in inducing hepatic glycogenolysis remains enigmatic though an indirect role through potentiation of CORT and glucagon action may be speculated. Such a suggestion gains credence from the previously reported adreno-cortical activation in response to gonadotrophins (chapter V).

The ability of gonadotrophic hormones to promote carbohydrate utilization in association with testicular recrudescence is well reflected in the significantly decreased testicular glycogen content. In this respect, FSH seems to have a more pronounced effect. The ability of CORT to induce insulin

resistance and hyperphagia has been reported (Nagra and Meyer, 1963; Taouis et al.,1993). Interestingly, a previous study from this laboratory had demonstrated increased insulin sensitivity in chicks rendered hypocorticalic (John,1990). Presently also, CORT treated birds were noted to show hyperphagia. In this context, the decreased hepatic glycogen content in CORT treated pigeons can be accounted for by the increased insulin resistance coupled with potentiation of CORT and glucagon action while the hyperglycemic state is mainly due to the observed hyperphagia and decreased peripheral uptake due to insulin resistance. Similar mechanism may be envisaged for birds treated with FSH.LH.In contrast, it is likely that FSH might potentiate peripheral utilization of glucose either directly or through insulin and, also nullify the CORT effect on peripheral utilization, as can be inferred from the observed hypoglycemic condition in birds treated with either FSH alone or in combination with CORT as well as the increased muscle glycogen content.

Finally, from the present results it emerges that gonadotrophic hormones can modulate systemic carbohydrate metabolism in addition to its acceptable role in gonadal glycogen depletion in relation to seasonal testicular activity. Since this is the first report of its kind, more careful scrutinyof this aspect is warranted.