PHOTOPERIOD-ADRENAL INTERACTIONS ON CARBOHYDRATE METABOLISM IN THE FERAL PIGEON DURING THE RECRUDESCENT PHASE

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Abstract: The possible influence of photoperiodism on carbohydrate metabolism has been evaluated in the feral pigeons. Both Dexamethasone (DXM) and Corticosterone (CORT) treated pigeons were exposed to NDL or LD 18:6 photoperiodic schedules and their tissue glycogen contents, blood glucose and hepatic phosphorylase and G-6-P’ase activity assayed. Both DXM & Corticosterone treatment increased tissue glycogen content under both photic schedules. Plasma glucose level increased under LD 18:6 while, CORT treatment decreased and, DXM treatment increased further the glycemic level under both photic schedules. The activity of hepatic phosphorylase decreased in all experimental birds while that of G-6-P’ase increased. From the results it can be concluded that gonadal recrudescence induces depletion of tissue glycogen content and hyperglycemic, which are potentiated by long photic schedule. Chronic adreno-cortical suppression or activation has differential effects on glycemic status probably through their action on glucoregulatory center or by the altering mechanism involved in glucose absorption, uptake and utilization.

Key Words: Dexamethasone, Corticosterone, Photoperiodism, Metabolism, Adrenal.

INTRODUCTION

Seasonal cyclic activities like reproduction may be divided into different life history stages and morphological, physiological and behavioral traits expressed in any life history stage will be regulated by characteristic hormonal mechanisms (Jacobs and Wingfield, 2000; Wingfield et al., 2002). Accordingly, 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 para-chloro phenyl alanine (pCPA) and pineal indoles, induced functional alteration of adrenal as well as long photoperiod have all been shown to affect carbohydrate metabolism (Ayyar et al., 1999; Patel et al., 1983, 1988, 2003; Ramachandran and Patel, 1987). It is also seen that functional alteration of adrenal and photoperiod have significant effects on testicular recrudescence in pigeon (Patel et al., 1983). Since it has been observed that tissue glycogen content and blood glucose level vary between breeding and non-breeding phases (Patel et al., 1983; Ayyar et al., 1999), 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 manipulations despite the fact that factors that 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: Adult feral blue rock pigeons, Columba livia in the weight range of 250-300 Gms, procured from a local animal dealer were used for present study. The birds were housed in well ventilated aviary with food and water ad libitum. After a week of acclimation, the birds were sexed using a laproscope and only males with similar testicular conditions were used for the experiments.

Lighting and lighting schedules: The cages used for housing the experimental birds were illuminated using cool day light fluorescent tubes. The light intensity employed was of 2000 Lux units which was measured with the help of Lux – meter (Weston Electrical Instrument Corporation, N.J., U.S.A.). The cage temperature varied by only 1°C from that of the room temperature. Experimental birds were exposed to two different photic conditions.
schedules namely:

1. Normal light – dark (NLD or LD 12:12) – i.e. 12 hours of light followed by 12 hours of darkness.
2. Long photoperiod (LD 18:6) – i.e. 18 hours of light followed by 6 hours of darkness.

For LD 12:12 lights were switched on at 06:00 hrs and put off at 18:00 hrs where as for LD 18:06 the lights were switched on at 06:00 hrs and put off at 24:00 hrs.

**Experimental set ups:**

**Group I** (N 12:12): Intact pigeons were exposed to LD 12:12 and served as control for CORT 12:12 and DXM 12:12.

**Group II** (CORT 12:12): These birds were given daily injections of Corticosterone at 09:00 hours and exposed to LD 12:12.

**Group III** (DXM 12:12): These birds were given daily injection of DXM at 17:00 hours and exposed to LD 12:12.

**Group IV** (N 18:06): Intact birds were exposed to LD 18:06 and served as control for CORT 18:06 and DXM 18:06.

**Group V** (CORT 18:06): These birds were given daily injection of Corticosterone at 09:00 hours and exposed to long photoperiod of LD 18:06.

**Group VI** (DXM 18:06): These birds were given daily injection of DXM at 17:00 hours and exposed to LD 18:06.

**Parameters and methods of evaluation:**

**Blood glucose:** Prior to decapitation of pigeons, 0.1 ml of blood was drawn from brachial vein by a needle prick. Blood glucose was estimated by method of Winckers and Jacobs (1971). The glucose concentration was expressed as mg/dl. Decapitation was done under mild anesthesia.

**Hepatic and muscle glycogen content:** The glycogen content was estimated employing the method of Seifter et al. (1950). Small pieces of tissues were dropped in preweighed test tubes containing 2 ml of 30% KOH. Glycogen was precipitated with 95% alcohol. The diluted precipitates were treated with anthrone reagent and colour intensity was read colourimetrically at 620 nm. Glycogen content was expressed as mg/100mg tissue weight.

**Hepatic glucose-6-phosphatase (G-6-P’ase/ E.C.3.1.3.9):** Liver tissue homogenate was prepared in cold citrate homogenate buffer. Enzyme activity was assayed by the method of Harper (1963). Glucose-6-phosphate disodium salt (sigma chemicals, USA) was used as a substrate. Inorganic phosphate released was estimated as per the method of Fiske and Subbarow (1925) and the color intensity was read at 660nm on a colorimeter. Enzyme activity was expressed as µg phosphate released/mgprotein/15 min.

**Hepatic phosphorylase (E.C.2.4.1.1):** Total phosphorylase activity was assayed by the method of Cahill et al. (1957) using glucose-1-phosphate dipotassium salt (Sigma chemicals, USA) as the substrate. The inorganic phosphate released was measured by the method of Fiske and Subbarow (1925). Enzyme activity was expressed as µg phosphate released/mgprotein/15 min.

**Hormone assays:** Insulin and glucagon levels were assayed using kits from Linco Research Chemicals U.S.A in a private pathological laboratory.

**RESULTS**

**Blood glucose and hepatic and muscle glycogen:** The blood glucose level which was higher during the recrudescence phase tended to increase under LD 18:06. However, CORT treatment in birds exposed to either NLD or LD 18:06 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 18:06 birds. The hepatic and muscle glycogen contents were significantly lowered in all experimental groups (Table 1).

**Hepatic phosphorylase and G-6-P’ase:** Pigeons under all experimental schedules showed significantly reduced phosphorylase activity and increased glucose-6-phosphatase activity (Table 1).

**Table 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 18:06 in the pre-recrudescent phase. (values are mean ± SD) (* significant at p<0.05)

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Blood Glucose (mg/dl)</th>
<th>Hepatic Glycogen (mg/100 mg tissue)</th>
<th>Muscle Glycogen (mg/100 mg tissue)</th>
<th>Hepatic G-6-P’ase activity (µ moles PO₄ released/mg protein/15min)</th>
<th>Hepatic Phosphorylase activity (µg PO₄ released/mg protein/15 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N 12:12</td>
<td>199.4± 11.05</td>
<td>6.44± 0.29</td>
<td>0.52±0.07</td>
<td>0.87± 0.05</td>
<td>10.83± 1.61</td>
</tr>
<tr>
<td>N 18:06</td>
<td>203.9± 21.00</td>
<td>2.60± 0.12</td>
<td>0.15± 0.02</td>
<td>1.70± 0.08</td>
<td>4.66± 0.71</td>
</tr>
<tr>
<td>CORT 12:12</td>
<td>184.97± 18.93</td>
<td>2.65± 0.15</td>
<td>0.11± 0.01</td>
<td>1.25± 0.06</td>
<td>4.94± 0.47</td>
</tr>
<tr>
<td>CORT 18:06</td>
<td>153.11± 17.91</td>
<td>1.52± 0.08</td>
<td>0.28± 0.04</td>
<td>1.70± 0.07</td>
<td>7.06± 0.95</td>
</tr>
<tr>
<td>DXM 12:12</td>
<td>208.98± 21.55</td>
<td>4.23± 0.25</td>
<td>0.33± 0.06</td>
<td>0.94± 0.05</td>
<td>5.93± 0.30</td>
</tr>
<tr>
<td>DXM 18:06</td>
<td>219.23± 19.19</td>
<td>2.30± 0.15</td>
<td>0.39± 0.03</td>
<td>1.02± 0.04</td>
<td>5.51± 0.37</td>
</tr>
</tbody>
</table>
Serum glucagon and insulin levels: The serum titres of insulin and glucagon show changes under all experimental schedules. In general all experimental schedules tended to decrease insulin level and increase glucagon level (Table 2).

**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 18:06 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 et al., 1999). 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 including 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. In this context the increasing glycogen levels and decreasing insulin levels under experimental conditions are relevant.

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 has altered effects on glycemic status and, 4. These effects of DXM or CORT are potentiated under long photoperiod.

**REFERENCES**


