Effect of prolactin on tissue cholesterol levels of male albino rats

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Abstract. Prolactin modulated tissue cholesterol distribution of male albino rats. Testis and epididymis had elevated cholesterol levels which was correlated to increased androgenesis. Both prostate and seminal vesicles had depleted levels, might be due to elevated seminal plasma secretion. Since heart and dorsal aorta recorded significant accumulation of cholesterol in response to prolactin administration, involvement of prolactin in cardiovascular disorders in rats was envisaged.

Keywords. Prolactin; cholesterol; testis; androgenesis; heart; cardiovascular disorders.

1. Introduction

Prolactin (PRL) stimulates androgenesis in the testis (Hafiez et al 1972a, b), leading to elevated spermatogenesis and fertility (Bartke 1966, 1967; Bartke and Lloyd 1970). Since PRL influences androgenesis which requires cholesterol, PRL induced cholesterol mobilization can be envisaged in the tissues of rats. Cholesterol deposition induces cardiovascular disorders (Thomas et al 1968; Brecher et al 1974) and hence modulations in cholesterol distribution by PRL might suggest the possible role played by PRL on cardiovascular disorders of the animals besides its effect on androgenesis.

2. Material and methods

Wistar Strain adult male albino rats weighing 200 ± 20 g were used as the experimental material for the present study. Experimental group of rats received 1·0 µg of PRL/g body weight subcutaneously (Ramachandra Rao et al 1978), while control group of rats received similar volume of physiological saline daily, for five days. After post-treatment day one, all the rats were sacrificed by decapitation and tissues like testis, epididymis, seminal vesicles, prostate gland, liver,
kidney, adrenal, brain, heart and dorsal aorta and serum were isolated. The total cholesterol content was estimated in all the tissues by the method of Natelson (1971) and cholesterol content was expressed in mg/g wet weight of the tissue.

3. Results and discussion

The data presented in table 1 reveals the influence of PRL administration on the cholesterol distribution in the tissues of adult male albino rats. The testicular cholesterol content was considerably elevated in PRL treated rats. Since testis is concerned with androgenesis (Dorfman and Shipley 1956), cholesterol forms raw material for androgenesis (Hall et al 1963; Hall 1963) and PRL is known to increase spermatogenesis and androgenesis (Bartke 1966, 1967; Bartke and Lloyd 1970; Hafiez et al 1972a, b), it is likely that elevated cholesterol level of testis in PRL administered rats might be due to its de novo synthesis, which might be diverted towards the formation of sex steroids.

Epididymis elevated cholesterol content in the PRL treated rats. Since epididymis is concerned with steroidogenesis (Prasad and Rajalakshmi 1976), the accumulated cholesterol might suggest the induced steroidogenesis in the tissue. Besides, maturation of spermatozoa is known to occur in epididymis and the spermatozoal maturation requires cholesterol (Mann 1964), and hence elevated cholesterol content in the tissues is suggestive of increased maturation processes in the presence of PRL. Both prostate gland and seminal vesicles showed depleted cholesterol content in the experimental rats. Prostate gland and seminal

Table 1. Effect of PRL administration on tissue total cholesterol distribution in reproductive and non-reproductive tissues of adult male albino rats.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Control</th>
<th>Experimental</th>
<th>% difference</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testis</td>
<td>1.4 ± 0.03</td>
<td>1.58 ± 0.06</td>
<td>+12.86</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Epididymis</td>
<td>1.33 ± 0.03</td>
<td>2.92 ± 0.08</td>
<td>+119.55</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Seminal vesicles</td>
<td>1.30 ± 0.07</td>
<td>1.07 ± 0.04</td>
<td>-17.69</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Prostate gland</td>
<td>1.69 ± 0.03</td>
<td>0.7 ± 0.01</td>
<td>-34.58</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Serum</td>
<td>39.27 ± 5.4</td>
<td>62.17 ± 4.7</td>
<td>+53.31</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Liver</td>
<td>4.90 ± 0.25</td>
<td>5.21 ± 0.31</td>
<td>+6.33</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Kidney</td>
<td>4.50 ± 0.21</td>
<td>6.13 ± 0.17</td>
<td>+36.22</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Adrenal</td>
<td>71.85 ± 0.12</td>
<td>79.49 ± 2.15</td>
<td>+10.63</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Brain</td>
<td>20.82 ± 0.17</td>
<td>34.42 ± 0.47</td>
<td>+65.32</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Dorsal Aorta</td>
<td>3.46 ± 0.42</td>
<td>7.61 ± 0.38</td>
<td>+119.94</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Heart</td>
<td>1.77 ± 0.06</td>
<td>2.11 ± 0.08</td>
<td>+19.21</td>
<td>P &lt; 0.001</td>
</tr>
</tbody>
</table>

(Mean ± S.D.; + and – indicate increase and decrease over control respectively).
vesicles are involved in the seminal plasma secretion (Mann 1964) and seminal plasma contains cholesterol (Scott 1945). Hence the depleted cholesterol content in seminal vesicles and prostate gland might suggest the PRL induced accelerated seminal plasma secretion. Since PRL is known to increase fertility in male rats (Bartke 1966, 1967; Bartke and Lloyd 1970) such possibility of elevated seminal plasma formation by these sex accessories can be envisaged.

In view of increased spermatogenesis and fertility in males, in the presence of PRL (Bartke 1966, 1967; Bartke and Lloyd 1970), there might have been changes in the cholesterol distribution of non-reproductive tissues. The serum cholesterol content is elevated in the PRL treated rats, suggesting its addition from the synthetic sites (Dungan and Porter 1977). The cholesterol content of liver, kidney and adrenal glands is elevated. Since these tissues are concerned with the synthesis of cholesterol (Harper 1975) it is likely that the cholesterol synthesized in them might have been accumulated in response to PRL. Brain, heart and dorsal aorta had elevated cholesterol content in PRL treated rats, maximum increase being in dorsal aorta. Elevated cholesterol in these tissues might be due to the active uptake of plasma cholesterol. These observations suggested the possibility of deposition of cholesterol on the walls of dorsal aorta in the presence of PRL, which might lead to cardiovascular disorders in rats.

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