SUMMARY

Levels of cardiac glycogen were determined by biochemical assays in turkey poults from an experimental flock with a known high incidence of round heart disease. Levels of cardiac glycogen were significantly higher ($p < .05$) than in commercial turkeys, but not significantly different in the turkeys that developed spontaneous round heart disease. The high level of cardiac glycogen in the poults of the experimental flock may be a manifestation of abnormal glycogen metabolism, but has an unclear role, if any, in the development of the round heart syndrome.

INTRODUCTION

Round heart disease in turkey poults is characterized by right, left, or bilateral ventricular dilatation and a thinning of the ventricular wall (17) consistent with an interstitial myocarditis (14). Microscopic lesions consistently present include myocardial congestion and degeneration, hemorrhage, and epicardial fibrosis (17). Of particular interest are the degenerative changes and their relationship to the metabolic state of the myocardium.

Foci of abnormal glycogen deposition were observed in regions of damaged myocardial fibers obtained from turkeys afflicted with the round heart syndrome (Czarnecki, unpublished). For study of cardiac glycogen deposition in cases of cardiac dilatation, turkey poults were fed toxic levels of furazolidone (FZ). The levels of glycogen in the hearts of these poults were elevated 2–3 times over the levels in control poults (2). This led to speculation that abnormal glycogen metabolism was a contributing factor to the round heart syndrome. To test the validity of this theory, myocardial tis-

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Cardiac glycogen in round heart disease

Table 1. Average glycogen content of left ventricular wall of turkey hearts at 5 weeks of age (μg/mg of tissue wet weight).

<table>
<thead>
<tr>
<th>Group^A</th>
<th>Total (KOH + TCA)</th>
<th>Acid-soluble (TCA)</th>
<th>Acid-insoluble (KOH)</th>
<th>TCA/KOH + TCA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hatch 1:</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>N</td>
<td>1.9 ± 0.4^B (9)^C</td>
<td>1.1 ± 0.3</td>
<td>0.9 ± 0.2</td>
<td>56</td>
</tr>
<tr>
<td>Male</td>
<td>1.9 ± 0.5 (5)</td>
<td>1.1 ± 0.4</td>
<td>0.9 ± 0.2</td>
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<tr>
<td>Female</td>
<td>1.8 ± 0.3 (4)</td>
<td>1.0 ± 0.2</td>
<td>0.8 ± 0.1</td>
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</tr>
<tr>
<td>R</td>
<td>2.3 ± 0.4 (8)</td>
<td>1.2 ± 0.3</td>
<td>1.0 ± 0.1</td>
<td>55</td>
</tr>
<tr>
<td>Hatch 2:</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>N</td>
<td>2.5 ± 0.9 (6)</td>
<td>1.5 ± 0.7</td>
<td>1.1 ± 0.2</td>
<td>58</td>
</tr>
<tr>
<td>R</td>
<td>2.2 ± 0.6 (15)</td>
<td>1.1 ± 0.4</td>
<td>1.1 ± 0.2</td>
<td>51</td>
</tr>
<tr>
<td>Male</td>
<td>2.2 ± 0.4 (9)</td>
<td>1.1 ± 0.2</td>
<td>1.1 ± 0.2</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2.2 ± 0.8 (6)</td>
<td>1.1 ± 0.6</td>
<td>1.0 ± 0.2</td>
<td></td>
</tr>
</tbody>
</table>

^A N = normal; R = round heart.
^B S.D. of the mean.
^C Number of poults.

MATERIALS AND METHODS

Small white-breasted turkey poults were obtained from an experimental flock with a known high incidence of round heart disease. The experimental flock has been described in a previous publication (10). Poults from two different hatches were sacrificed at 5 weeks of age. Poults were selected for sacrifice on the basis of EKG data as described by Jankus et al. (9). At sacrifice, a portion of the left ventricular wall from base to apex was removed and quenched in liquid nitrogen as described previously (1). Myocardial tissue from each heart was minced into pieces approximately 1 mm³ in size, randomly divided into 5 samples, weighed on a Sartorius balance, and stored in a low-temperature freezer (−50 C) until the day of extraction. Acid-soluble and acid-insoluble fractions of glycogen were determined as described by Czarnecki (1).

RESULTS

Table 1 shows data from the glycogen assays of the myocardial tissue. The most striking observation was the wide variation in levels of cardiac glycogen in poults classed as normal (N) and round heart (R) by EKG recordings (9) and confirmed by gross examination of the heart. The greatest variance was noted in the acid-soluble fraction in both N and R poults. In poults from the first hatch, the levels of the acid-soluble fraction ranged from 0.63 to 1.49 μg/mg in the N poults and from 0.84 to 1.62 μg/mg in the R poults. Levels of the acid-insoluble fraction varied from 0.74 to 1.25 μg/mg in N turkeys and from 0.83 to 1.21 μg/mg in R turkeys. How-
ever, there were no significant differences (P < 0.05) in the glycogen levels of N vs. R poults or in male vs. female poults.

Results were similar from assays on turkeys from the second hatch. Here, again, variance in glycogen levels was greatest in the acid-soluble fraction, indeed varying considerably more than in poults from the first hatch. The range of this fraction was 0.42–2.08 μg/mg in N poults and 0.51–1.99 μg/mg in R poults. Variations observed in the acid-insoluble fraction were nearly identical to those noted in the first hatch, 0.74–1.28 μg/mg for N poults and 0.72–1.38 μg/mg for R poults. Again, no significant differences (p < .05) could be accounted for either on the basis of sex or EKG classification. Likewise, there were no significant differences between the poults from the two hatches.

Slightly more than half (51–58%) of the total cardiac glycogen was present as the acid-soluble fraction. However, in individual turkeys the proportion of the total glycogen extractable as the acid-soluble fraction varied considerably. In poults with low levels of cardiac glycogen, there were proportionately lesser amounts of the acid-soluble fraction. For instance, in one N poult the concentration of total glycogen was 1.2 μg/mg, with only 36% of this amount extractable as the acid-soluble fraction. This can be contrasted with another N poult in which the acid-soluble fraction comprised 64% of the total glycogen assayed at a concentration of 3.2 μg/mg. The proportion of total glycogen present as the acid-soluble fraction was similar in R poults and in N poults.

DISCUSSION

The wide variation in levels of cardiac glycogen and lack of significant differences between N and R turkeys is somewhat disturbing. In a previous study conducted on commercially available turkey poults in which cardiac dilatation was induced by toxic levels of FZ, the levels of cardiac glycogen were more uniform and obvious differences were apparent between unaffected poults fed low doses of the drug and poults fed toxic doses (2).

Cardiac glycogen levels in the experimental flock were significantly greater (p < .05) in N poults than in N poults obtained commercially (1,2). The difference was greatest in the acid-soluble fraction which was elevated by at least a factor of 2. Such a large amount of glycogen present as a highly mobilizable fraction lends credence to the supposition that abnormal glycogen metabolism may be a factor in round heart disease. Sufficient documentation
supports the current belief that glucose is not the principal fuel utilized by the myocardium under normal conditions (5,11,12,15). It, therefore, seems unlikely that such large stores of glycogen would be necessary to satisfy the usual energy requirements of the myocardium.

The findings that cardiac glycogen levels are increased in diabetic conditions (13,16,18), in myocardial infarctions (19), in glycogen storage diseases (3), and in FZ-induced cardiomyopathy (2) lend further support to the theory that the abnormal amount of cardiac glycogen observed in N poults of the experimental flock is an expression of a disturbance in glycogen metabolism. Such an anomalous condition could be the result of inheritable enzymatic defects. Since toms appear to be more susceptible than hens to the round heart syndrome, inheritance has been proposed as an etiologic factor to be investigated further (4,6,7,8,14). In the present study there were no significant differences between toms and hens in levels of cardiac glycogen. This indicates that the level of cardiac glycogen per se appears not to be the primary factor in predisposing turkeys to the round heart condition.

The cardiac glycogen levels (2) were significantly higher (p < .05) in FZ-induced cardiomyopathy than in the R poults of the experimental group. The effects of the drug appeared to be twofold: 1) alteration of the cardiac physiology to simulate the round heart syndrome; and 2) significant (p < .01) elevation of cardiac glycogen levels. The latter effect is presumed to be due to altered glycogen metabolism, though the actual mechanism has not been determined. The role, if any, that this may play in increasing the turkey poult's susceptibility to the round heart syndrome is unknown, but bears further investigation. The lack of correlation in levels of cardiac glycogen between spontaneous round heart disease and FZ-induced cardiomyopathy is probably due to the effect, as yet unknown, of the drug on glycogen metabolic pathways. That the drug influences glycogen metabolism was demonstrated in the previous study (2) by allowing affected turkeys to recover spontaneously following removal of the drug. After a recovery period of 7 weeks, the cardiac glycogen returned to normal levels even though the EKG pattern and gross morphologic characteristics were typically those seen in the round heart condition.

Caution should be exercised in any attempt to equate the spontaneous round heart condition with FZ-induced cardiac dilatation.
While both disease states exhibit a number of similarities, present knowledge is insufficient to establish definitively that the FZ-induced condition is a true replication of the spontaneous condition.

REFERENCES