THE EFFECTS OF OBESITY ON THE PANCREATIC BETA CELL FUNCTION AND MORPHOLOGY IN WHISTAR RATS

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Key words: rat, obesity, beta cell dysfunction, hypertrophy, hyperplasia

SUMMARY

The aim of the present article is the study of obesity and consecutive insulin resistance in the beta cell dysfunction and in the pathogenesis of diabetes mellitus.

Experimental subjects: 20 male Whistar rats, age of approx. 150 days. The rats of each lot were housed five in a cage, on sawdust, kept 12 hours light and 12 hours dark, with water at discretion.

Lot 1 (n=10) – the normal witness lot was fed with standard rat chow once for a day;
Lot 2 (n=10) – the obese ones were fed with high caloric chow at discretion, 7 months;

Obesity was appreciated by weight (W) and body mass index (BMI) measurements. The oral glucose test (OGTT) and an insulin tolerance test (ITT) were applied after 7 month in order to determine the insulin resistance and the beta cell function. The morphology of Langerhans islets was appreciated comparatively at the two lots after 7 months. Identification of B cells was attempted by the tricrom Mallory staining method. The following were counted and measured at the histology exam for the each subject of the experiment: main dimension of islet cut surface of 20 islets, main number of endocrine cells in 20 islets, average of 40 beta cell cut surface diameter. Statistical comparison of the values was made, using the Student-t test.

Results: a significant obesity was obtained at the lot 2 in just 7 month of overfeeding. The functional testes for determining the islet function revealed insulin resistance and a certain degree of glucose intolerance. The glycemia decrease was slighter at the obese lot after insulin challenge meaning an insulin resistance at the obese lot.

<table>
<thead>
<tr>
<th>Weight gr</th>
<th>BMI kg/m2</th>
<th>OGTT Glycemia mg/dl</th>
<th>ITT Glycemia mg/dl</th>
<th>Main dim of islets µm</th>
<th>Main number of B cells/islet</th>
<th>Main dimension of B cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lot 1</td>
<td>279±18</td>
<td>19.49±0.5</td>
<td>92±14</td>
<td>58±11</td>
<td>109±29</td>
<td>102±25</td>
</tr>
<tr>
<td>Lot 2</td>
<td>406±36</td>
<td>28.2±0.7</td>
<td>120±18</td>
<td>78±21</td>
<td>134±36</td>
<td>129±28</td>
</tr>
</tbody>
</table>

Table 1. Representation of main values of functional and morphological differences between the normal (Lot 1) and obese (Lot 2) rats.

The pathology exam of the pancreas demonstrated the adaptation of the beta cell function to the insulin resistance by morphological changes. Hypertrophy and hyperplasia of the beta cells were observed in the obese lot.

Conclusions: the insulin resistance of the obese rats caused an adaptation in the beta cell function and morphology characterized by hyper function to compensate the periphery insulin resistance and morphologically, it caused hypertrophy and hyperplasia as well. The rat endocrine islets have more adaptation resources comparing to the human pancreas.