Iron metabolism disorder in prepubertal obese children with and without NAFLD

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Background
Childhood obesity is associated with non-alcoholic fatty liver disease (NAFLD). Previous studies in obese adult and pubertal children with NAFLD have shown that chronic inflammation/oxidative stress and insulin resistance might induce iron metabolism disorders, characterized by increased Hepcidin and Ferritin levels and decreased serum Iron levels. However, data evaluating these findings in a well selected population of obese prepubertal children are still missing.

Aims of the study
- we aimed to characterize iron metabolism in a group of 40 obese prepubertal children with and without NAFLD defined by ultrasonography, compared to 40 healthy prepubertal age- and gender matched peers
- we also investigated correlations between iron metabolism and both oxidative stress and metabolic markers

Materials and methods

STUDY POPULATION
80 prepubertal children

20 with NAFLD
11Male/9Female

20 without NAFLD
12Male/8Female

40 healthy prepubertal matched peers
22Male/18Female

Anthropometric measurements were determined
- Fasting blood samples were collected for measurement of insulin, glucose, lipid profile
- HOMA-IR was calculated as Insulin Resistance Index
- ALT, AST and iron profile including iron concentration, ferritin and hepcidin
- Lag-phase and MDA were evaluated as markers of oxidative stress

Statistical Analysis
- All values were expressed as means and SD
- Differences across the three groups were evaluated by One-way Anova test
- Post-hoc assessment was calculated by Bonferroni test
- In obese subjects a Pearson’s correlation was used for searching correlations between Hepcidin and other parameters
- Significant values P <0.05

Results

<table>
<thead>
<tr>
<th></th>
<th>Lean Healthy Controls</th>
<th>Obese without NAFLD</th>
<th>Obese with NAFLD</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>40</td>
<td>20</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>8.8±1.9</td>
<td>8.9±1.6</td>
<td>9.1±1.9</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>22/18</td>
<td>12/8</td>
<td>11/9</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (Kg)</td>
<td>30.7±18.8</td>
<td>48.5±13.1</td>
<td>56.4±15.2</td>
<td>&lt;0.01; †</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>17.8±3.6</td>
<td>25.1±3.4</td>
<td>27.6±4.3</td>
<td>&lt;0.01; †</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>57±5</td>
<td>78±12</td>
<td>86±11</td>
<td>&lt;0.01; †</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>103±9</td>
<td>110±25</td>
<td>112±13</td>
<td>0.03; †</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>59±5</td>
<td>63±8</td>
<td>63±8</td>
<td>NS</td>
</tr>
</tbody>
</table>

Obese without NAFLD versus Lean Healthy Controls‡
Obese with NAFLD versus Lean Healthy Controls‡
Obese without NAFLD versus Obese with NAFLD †

Conclusions
Obese prepubertal children show impaired iron metabolism disorders, especially in those subjects with NAFLD. The correlation between Hepcidin levels and increased oxidative stress activity in obese prepubertal children suggest a role of these components in the early pathogenesis of NAFLD in prepubertal children.