Contribution ID: **96** Type: **Oral Communications**

ORAL PRESENTATION - Radiopharmacological studies of leptin and thyroid hormones relationship in white adipose tissue

Monday 17 September 2012 12:00 (15 minutes)

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The metabolism of thyroid hormones (TH) in white adipose tissue (WAT) is so far poorly characterized. Therefore, we followed changes in TH metabolism in mice, which were associated with the development of high-fat (HF) diet-induced obesity, with the aid of several radioanalytical methods –radiometric enzyme assays and radioimmunoanalysis (RIA), among others. Possible changes in activities of the key enzymes of TH metabolism in murine WAT were measured not only during obesogenic treatment of the mice but also in response to caloric restriction or leptin administration. Adult male C57BL/6J mice were subjected to these three different treatment protocols. Subcutaneous and epididymal WAT and interscapular brown fat (BAT) depots were dissected and used for morphometric and enzymatic analyses. In addition, blood and samples of liver were also collected (and the latter used as controls for enzymatic determinations). Plasma levels of leptin, as well as total and free thyroxine (T4) and triiodothyronine (T3) concentrations were determined using RIA kits. Enzyme activities of iodothyronine deiodinases of the types 1 (D1), 2 (D2) and 3 (D3) in the liver, WAT and BAT were measured with the aid of our newly developed radiometric enzyme assays. We found that D1 enzyme activity in WAT was stimulated by a high-fat-diet feeding, which also increased plasma levels of leptin. However, D1 or D2 activities in BAT did not change. On the contrary, caloric restriction decreased D1 activity in WAT, but not in the liver, and reduced leptin levels. In return, leptin injections increased D1 activity in WAT. In summary, our results demonstrate, for the first time, changes in D1 activity in WAT under the conditions of changing adiposity, and a stimulatory effect of leptin on D1 activity in WAT. Support from the Academy of Sciences of the Czech Republic (Project No. AV0Z50110509) and from the Czech Science Foundation (GACR Grant No. 304/08/0256) is acknowledged.

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Session Classification: Session 1 - Radiopharmaceutical Chemistry (radiodiagnostics, radiotherapy, theragnostics)

Track Classification: Radiopharmaceutical chemistry, radiodiagnostics, radiotherapy, theragnostics