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The Endocrine Society's 91st Annual Meeting

Filename: 850367

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Member ID #: 238543 Not an Active Member or Emeritus Member.

Professional Role: Basic Researcher

Abstract Format and Category

Session Type: Regular Abstract Session

Presentation Type: Consider for Oral Presentation

Category: 43. Thyroid Hormone Action/HPT Axis Biology (excluding receptor structure & function)

Research Type: Translational

I am a trainee who is the first and presenting author of the submitted abstract. If NOT chosen for an Oral Presentation I would like to be considered for the Presidential Poster Competition.

Keyword 1: Thyroid
Keyword 2: Iodide
Keyword 3: Membrane

Suggested Keyword: transporter

Awards:

Travel Grant Awards

You consider yourself a member of this U.S. underrepresented group:

N/A

Abbott Thyroid Research Clinical Fellowship & Mentor Awards

Name of mentor/training director: Samuel Refetoff

Award - Education Status: *Postdoctoral/ Research Fellowship*

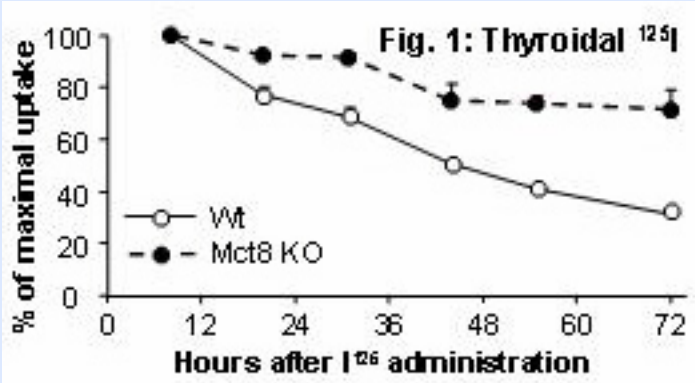
I have read and confirm that I meet all the criteria of each award for which I have applied.

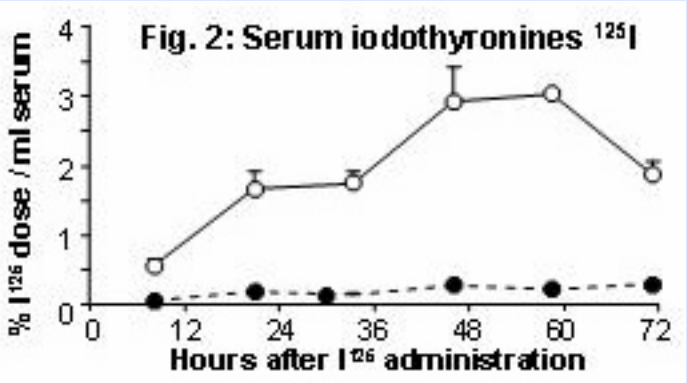
Sponsor Info - I am an international non-member and have been unable to secure a sponsor for my abstract. I request that the Annual Meeting Steering Committee review my abstract.

Title: A novel role for MCT8: Control of thyroid hormone secretion

C Di Cosmo[1], X Liao[1], AM Dumitrescu[1], NJ Philp[3], RE Weiss[1] and S Refetoff[1,2]. [1]Medicine, University of Chicago, Chicago, IL, United States, 60637; [2]Pediatrics and Genetics, University of Chicago, Chicago, United States and [3]Pathology, Anatomy and Cell Biology, Thomas Jefferson University, Philadelphia, PA, United States, 19107.

The mechanism of thyroid hormone (TH) secretion from the thyroid gland into the blood is unknown. We used the Mct8 deficient mouse (Mct8KO) to determine if MCT8 has a role in this process. While MCT8 is known to transport TH into cells, several observations suggest that it also controls TH secretion: (1) Humans and mice deficient in MCT8 have a low serum T4 level, which cannot be fully explained by increased deiodination; (2) Our preliminary data show that TH secretion in Mct8KO mice is delayed following the release of endogenous hormone suppression with methimazole and perchlorate; (3) MCT8 is localized at the basolateral membrane of thyrocytes. RESULTS: Thyroid glands of Mct8KO mice contained 2.1-fold and 2.3-fold more free T4 and T3 than wild-type (Wt) mice ($P<0.001$). This was independent of deiodination as comparable increases were also found in Mct8KO mice that lacked the types 1 and 2 deiodinases. Next we determined the rate of iodothyronine secretion in mice thyroid glands after administration of ^{125}I. Peak thyroidal ^{125}I uptake occurred at 8 h in both genotypes. However, in Mct8KO mice there was a significant reduction in the rate of decrease in thyroidal ^{125}I (Fig. 1) and in the appearance of iodothyronines in serum as TCA-precipitable radioactivity (Fig. 2).





Results were confirmed by measurement of the stable T4 levels in serum following injection of 2 mU of bovine TSH to Mct8KO and Wt mice in which endogenous TSH and T4 levels had been suppressed by the administration of L-T3. The T4 response was significantly reduced in the Mct8KO mice as compared with the Wt mice (1.65±0.1 vs 2.02±0.06 μg/dl; P<0.03). CONCLUSIONS: MCT8 is involved in the secretion of TH from the thyroid gland. The defect in the thyroid efflux contributes the low serum T4 level observed in MCT8 deficiency.

Sources of Support: NIH grant DK 15070 and the Sherman family

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