

17th World Congress of the Academy of Human Reproduction

15–18 March 2017
Rome, Italy

TITLE

MOLECULAR MECHANISMS OF THYROID HORMONE ACTION DURING BLASTOCYST IMPLANTATION

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ABSTRACT

Context: A clinical association between implantation failure and thyroid dysfunction has been extensively reported, although the molecular mechanism governing this correlation has not been elucidated yet. TH machinery is expressed in the fetomaternal unit at implantation, suggesting a local action of TH.

Objective: We have investigated the role of TH in embryo implantation, focusing on the molecular regulation of blastocyst hatching and outgrowth.

Methods: Mouse blastocysts were cultured on a feeder-layer of primary endometrial cells or on plastic, with or without TH supplementation, and hatching and outgrowth evaluated. TH stimulation was also studied on endometrial cell cultures without blastocysts. By qRT-PCR, the expression of ISP1 and ISP2 (two proteases involved in blastocyst hatching in mice), and MMPS was studied.

Results: TH supplementation significantly increased the number of hatching blastocysts cultured on the endometrial cells, while had limited effect on blastocysts cultured on plastic. Isp1 and Isp2 were significantly up-regulated in both blastocysts and endometrial cells, independently analyzed after an overnight co-culture with TH; a less pronounced effect was observed in both blastocysts and endometrial cells cultured alone. TH also induced a significant up-regulation of several MMPs in endometrial cells, independently from the presence of the blastocysts, although the co-culture induced a much higher increase. TH also significantly increased the expansion of trophoctodermal cells in both culture conditions, however expansion was more pronounced in the presence of the endometrial feeder layer.

Conclusions: TH plays a key role in the bidirectional crosstalk between the competent blastocyst and the receptive endometrium at the time of implantation. TH supplementation may improve implantation success.

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