Follicle cell calmodulin: transcript accumulation in vitellogenic follicles of {\it Blattella germanica\/} is regulated by juvenile hormone

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Abstract

Calmodulin (CaM) is a major intracellular calcium receptor. There is abundant calmodulin (CaM) in the oocytes and eggs of B. germanica during vitellogenesis and early embryogenesis. The accumulation of CaM in oocytes may be for immediate use in the oocytes and/or in preparation for later stages of their development. Previous investigation from this laboratory suggested that maternal follicle cells are the most likely source of this CaM. Tissue culture labeling with $\sp{35}$S methionine showed a 13-fold higher rate of synthesis of CaM in the follicle cells than in oocyte preparations (Zhang & Kunkel, 1994). The high rate of biosynthesis of CaM in the follicle cells, and the absence of extracellular CaM in transit in the hemolymph suggested that CaM is made in the follicle cells and transferred to the oocytes. In order to obtain more information about the site of CaM synthesis I isolated total RNA from different tissues that could potentially contribute to the high amounts of follicular CaM and measured the amounts of CaM transcripts during development. I show that isolated whole follicles accumulate more transcripts for CaM than the fat body. The steady state levels of CaM transcripts increases 150 fold during the 4 day developmental period under study. This is in addition to a 32 fold increase in total follicle RNA during the period. Steady state levels of CaM transcripts in whole follicles also show a pattern of increase disproportionate to the increase in volume of the whole follicle. In comparison steady state levels of actin transcripts increase 35 fold during the same developmental period. At 96 hr post feeding, in a given amount of total RNA, follicle cell total RNA contains 3 times more CaM transcripts than whole follicle total RNA, and 70 times more CaM transcripts than the fat body tissue. The oocyte total RNA collected from material expelled from the whole follicle contains less than 10% of the amount of CaM transcripts

available in the follicle cells. The fat body tissue preparation shows little developmental increase in steady state levels of CaM transcripts despite a 4 fold increase in total RNA. In my investigation into the control of the accumulation of this transcript I found that deprivation of JH, by head ligation, not only causes atresia of the follicles, but also reduces CaM transcript accumulation. Reconstituting JH titer by injection allows a selected population of follicles to develop to full size and also reinstates steady state CaM transcript levels above that of unligated controls. The results of my study makes the CaM gene a potentially important target for the study of JH action in follicle cells during oogenesis. ^

Subject Area

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