## Identification of *c-mos* Homologue in the Sawfly *Athalia rosae ruficornis* (Hymenoptera) and Its Expression Analysis<sup>\*</sup>

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In vertebrates, the mechanisms of meiotic cell cycle arrest during egg maturation have become increasingly clear in recent years (Tunquist and Maller, 2003). Mos, the product of *c-mos* proto-oncogene is known as a key protein functioning as the cytostatic factor (CSF) in vertebrate meiosis. Mos activates mitogen-activated protein kinase (MAPK) cascades, and causes the arrest of meiosis at the second metaphase (MII) before fertilization. On the other hand, the mechanisms still remain largely unknown in invertebrates including whether common and conserved mechanisms as in vertebrates exist. We have been analyzing the mechanisms of meiotic cell cycle regulation in insects using *Athalia rosae ruficornis* (Hymenoptera, Symphyta, Tenthredinidae) as a model organism. We have shown that a MAPK cascade participated in egg maturation in *A. rosae* as in vertebrates, and suggested that the Mos-like protein was a regulator of the MAPK cascade (Yamamoto *et al.*, 2004).

We now have successfully cloned the *c-mos* gene homologue of *A. rosae* (Ar mos) and have done some preliminary examinations. *Ar mos* encodes 313 amino acids in a single open reading frame (ORF) and the ORF is comprised of four exons. Sex-, tissue- and stage-specific expression of Ar mos was examined using the RT-PCR. The ovary was the organ Ar mos was expressed most strongly. Further examinations by Western blot analysis using anti-A. rosae Mos antisera, showed that Mos was present in mature unfertilized eggs, but not in immature eggs with germinal vesicle. Once meiosis resumed upon artificial egg activation, Mos then disappeared. These results suggest that Mos participates in the meiotic cell cycle arrest and regulates the MAPK cascade in *A. rosae*.

## References

Tunquist, B.J. and J.L. Maller (2003) Genes Dev., 17, 683–710.
Yamamoto, D.S., J.M. Lee, K. Tachibana and M. Hatakeyama (2004) Proc. Arthropod. Embryol. Soc. Jpn., 39, 71.

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