



**Fig. 3** S<sub>1</sub> nuclease mapping of *mos* transcripts. **a**, S<sub>1</sub> mapping using probe B. Lane 1, 25 μg total testis; lane 2, 2 μg of poly(A)<sup>+</sup> testis RNA plus 30 μg of yeast tRNA; lane 3, 3 μg of embryonic poly(A)<sup>+</sup> RNA plus 30 μg of yeast tRNA; lane 4, 30 μg of yeast tRNA. The samples were hybridized to probe B at 60 °C overnight. **b**, S<sub>1</sub> mapping using probe C. Lane 1, 25 μg of total testis RNA; lane 2, 30 μg of yeast tRNA. The samples were hybridized to probe C at 52 °C overnight. For analysis of DNA/RNA hybrids and preparation of RNA samples, see Fig. 2 legend. The X-ray film (XAR-5; Kodak) was exposed for 14 days at -70 °C in the presence of an intensifying screen.

transcript size is overestimated by Northern analysis or there is RNA processing 5' and/or 3' to the protected regions.

While the embryonic RNA transcripts protect a discrete ~925-bp fragment of probe A (Fig. 2a, lanes 3 and 4) and a ~480-bp fragment of probe B (Fig. 3a, lane 3), they do not give a detectable signal with probe C (not shown), indicating that they differ from the testis RNA transcripts in the region 5' to the *mos* ATG. Similarly, S<sub>1</sub> mapping of the ovarian RNA transcripts indicates that they also differ from both the testis and embryo transcripts at the 5' end (not shown). Collectively, these results suggest that transcripts from the single-copy *mos* locus are expressed in a tissue-specific manner. In addition, these transcripts are either promoted from different sites and/or are spliced differently in testes, ovaries and embryonic tissues.

We have shown that *c-mos* is not efficiently activated by a downstream long terminal repeat (in contrast to *v-mos*<sup>1</sup>); this has been attributed to the presence of an upstream transcription termination sequence termed UMS<sup>21</sup> (M. L. McGeady and G.F.V.W., in preparation) and to the lack of a promoter between UMS and *c-mos* that can function in NIH 3T3 cells<sup>21</sup>. How then can *mos* be expressed? One suggestion<sup>21</sup> is that *mos* expression can be *trans*-activated. The presence of *mos* transcripts in tissues like testes and ovaries may indicate a *trans*-activation mechanism mediated by hormonal control. We also note that an additional 60 codons are proximal to the conserved *mos* ATG in all three *mos* genes that have been sequenced<sup>8,22</sup>. Thus, in addition to tissue-specific regulation of the size of the *mos* transcripts expressed, the presence or absence of these codons (or possibly other exons) could give rise to functionally different protein products in specific tissues.

Finally, the higher levels of *mos* expression detected in gonadal tissues and the differences in transcript size between ovaries and testes suggest that *mos* proto-oncogene expression is regulated in a sex-linked manner and may be associated with expression of primary and/or secondary sex-related phenotypes.

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**Note added in proof:** We have also detected a 6-kb *mos* transcript in poly(A)<sup>+</sup> RNA from embryos and epididymis. In addition, *mos* transcripts are present in rat embryos and testes.

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## Errata

### Does interocular transfer occur in visual navigation by ants?

R. Wehner & M. Müller  
*Nature* **315**, 228-229 (1985)

The third sentence of the last column on page 229 should read "In honey bees, IOT has been inferred . . ."

### Identification and comparison of two sequence elements that confer cell-type specific transcription in yeast

A. M. Miller, V. L. MacKay & K. A. Nasmyth  
*Nature* **314**, 598-603 (1985).

In the eighth line of paragraph two,  $\alpha/\alpha$  diploid should read  $a/\alpha$  diploid, and in the upper part of Fig. 5a and in the third line of the Discussion,  $\alpha 1/\alpha 2$  should read  $a 1/\alpha 2$ .