threshold level of the protein. Second, when CSF preparations from crushed frog eggs were depleted of pp39^{mos} with monoclonal antibodies, they lost their CSF activity. Control antibodies did not diminish the CSF content of the cell-free extracts. A similar conclusion can be drawn from recent experiments in mouse oocytes, where ablation of c-*mos* mRNA with antisense oligonucleotides prevented meiosis II arrest[12].

There is a close relationship between CSF and MPF despite their clearly different identities. CSF is in effect a specialized entity designed to stabilize MPF in metaphase-arrested eggs. CSF does not advance the cell cycle in microinjected *Xenopus* blastomeres; it simply locks the cell in metaphase once metaphase has been achieved by the action of MPF. The key question is therefore: how is MPF turned on and off, and how can its level be kept high? This issue was recently discussed by Andrew Murray in these pages[13]. The figure presents a speculative summary. There is no doubt that MPF is turned on by a protein phosphatase[14] and turned off by a protease that digests its cyclin subunit. A key point is that cyclin is only unstable during mitosis, which means that MPF must itself activate the protease, probably indirectly as shown in the model. This provides a negative feedback loop that makes mitosis normally a self-limiting process. The circuit almost certainly contains a positive feedback loop as well; there are reasons to suspect that the phosphatase that turns on MPF is itself turned on by MPF as shown by the positive feedback arrow in the figure.

One interpretation of Sagata *et al.*'s results is that the putative cyclin-specific protease of the model is kept switched off by pp39^{mos}-mediated phosphorylation. According to this view, the protease is activated by a phosphatase, which by a simplifying guess could be the same phosphatase that activates and keeps MPF active. The idea that the cyclin protease is turned on and off by reversible phosphorylation is attractive, because it could explain why cyclin is only unstable during a narrow window that opens just before the onset of anaphase and closes once MPF activity has been lost. The alternative hypothesis is that cyclin itself can be made protease-resistant by c-*mos*-catalysed phosphorylation. The species specificity of c-*mos* might arise by co-evolution with the surprisingly variable amino-terminus of cyclin, which is required for destruction[15] and which shows rapid evolutionary drift. It is not yet clear if cyclin is a substrate for pp39^{mos} and, if so, whether pp39^{mos}-phosphorylated cyclin resists destruction. This is of course an active area of investigation.

The puzzling variability of the cell-cycle arrest states shown by oocytes and eggs of different animals is now somewhat easier to understand. Molluscan oocytes are fertilized directly in first meiotic prophase, frog and mouse eggs arrest in second meiotic metaphase, while sea-urchin eggs are blocked in the G1 phase of the first post-meiotic mitotic cell cycle. It used to be difficult to see how such variability could evolve without extensive changes to the cell-cycle control machinery, which as we now appreciate shows high evolutionary conservation. The c-*mos* results give an example of how this can be achieved; a protein kinase appears in the right place at the right time, phosphorylates a key cell-cycle regulator, and then vanishes.

How can c-*mos* act as an oncogene if its primary role is to *block* the cell cycle? The answer is not clear. One possibility is that pp39^{mos} indeed blocks somatic cells in metaphase, which may sometimes result in chromosome breakage and hence aneuploidy when cells escape the block. Another possibility is that c-*mos* could stabilize 'start' cyclins in the G1 phase of the cell cycle and thereby cause cells to proliferate inappropriately. Conceivably some sort of side reaction — for example the inappropriate phosphorylation of a protein quite unconnected with cell-cycle regulation — might lead to the transformed phenotype, but the effects of c-*mos* expression in cells will surely come under new scrutiny in relation to the cell cycle. It would be particularly interesting to know whether appropriate levels of pp39^{mos} expression block normal somatic cells in mitotic metaphase; was this the basis of its previously noted toxicity[16]? Paradoxically, the original finding that pp39^{mos} can promote frog oocyte maturation is now even harder to understand than it was at first report[17]. The new results make much more sense. □

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David Jones