followed by surface warming in Kennett *et al.*'s data suggests a potential lead of catastrophic methane release over Northern Hemispheric warming. Could methane have triggered the climate change from glacial to interglacial? Raynaud *et al.* (*14*) list several points against such an impact of methane. First, deglaciation in Antarctica started several millennia before the fast methane increase (see the figure) (*10*). Second, the contribution of methane to greenhouse warming is much smaller than the one from CO_2, which rises simultaneously with southern temperature (*15*). Further, the methane increase lags the drastic temperature increase in Greenland by up to a few decades (*16*).

Kennett *et al.*'s study shows that methane releases linked to climate change do occur. It is reasonable to assume that such methane release from hydrates intensifies during times of fast temperature increase. However, because of the diversity of the source and its dependence on parameters such as sea level and temperature changes linked to ocean circulation, it is difficult to judge its contribution to fast CH_4 increases in the climate record. The timing of climate and methane changes suggests, however, that methane did not play a more active role in past climate change than has been accounted for so far.

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NOTA BENE: DEVELOPMENT

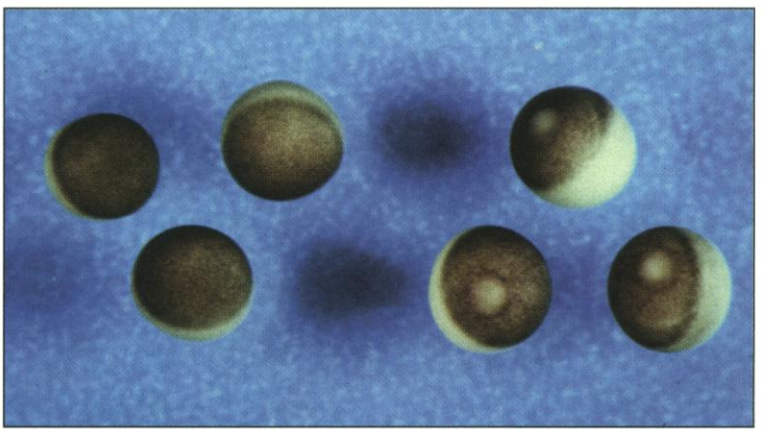
Awakening Aurora

The vertebrate oocyte is a veritable sleeping beauty, for it can remain slumbering in the G_2/M phase of meiosis for months or, in the case of humans, decades. In the frog *Xenopus*, the steroid hormone progesterone is the prince that awakens the oocyte, activating a signaling pathway that enables the oocyte to reenter the cell cycle and to mature into a sperm-responsive egg. Exactly how progesterone awakens the sleeping oocyte (see the figure) is not clear, but a recent report from Richter, Ruderman and their colleagues (*1*) reveals that an elaborate cast of characters helps progesterone in its quest.

Slumbering oocytes contain large stockpiles of ribosomes and mRNAs that encode proteins vital for oocyte maturation and for early development of the embryo after fertilization. Unlike mRNAs in somatic cells, which carry long tails of polyadenylic acid [poly(A)] and are readily translated into protein, these mRNAs have short, stubby poly(A) tails and are not translated. In response to gonadotrophin hormones, follicle cells surrounding the oocyte release progesterone, which binds to an unidentified receptor on the oocyte surface. This results in a rapid decrease in adenylyl cyclase activity, cyclic AMP levels and protein kinase A activity. But the next steps in the signaling pathway that awakens the oocyte are still shrouded in mystery.

Earlier work by the Ruderman group (*2*) on frog oocytes (favored because of their accessibility and large size) reported that activation of *c-mos* mRNA—which encodes the protein kinase Mos, a key component of the MAP kinase signaling pathway—required Eg2 (a serine/threonine kinase of the Aurora family). The researchers found that within 30 minutes of progesterone stimulation, Eg2 became activated through acquisition of a phosphate group (phosphorylation) and then somehow elicited the translation of *c-mos* mRNA into Mos, with subsequent activation of the MAP kinase signaling cascade. This cascade culminates in the activation of M-phase promoting factor (a complex of cyclin B and cdc2), leading to breakdown of the nuclear envelope and reentry of the oocyte into the cell cycle.

But exactly how does Eg2 elicit translation of *c-mos* mRNA? The Ruderman and Richter groups already knew the identity of the key character in the next chapter of the Aurora story: the cytoplasmic polyadenylation element binding factor (CPEB). In their new work (*1*), they discovered that CPEB was phosphorylated at a single site (serine-174) soon after progesterone stimulation but before translation of *c-mos* mRNA; phosphorylation of other CPEB sites occurred much later and depended on Mos activity. The investigators then demonstrated that Eg2 was responsible for the early phosphorylation of CPEB on serine-174, and that this in turn promoted the addition of a long poly(A) tail onto *c-mos* mRNA, resulting in its translation. They suggested that the later Mos-dependent phosphorylation of CPEB might result in its degradation, its task now being complete.



Next, the investigators plan to nail down the identity of the elusive oocyte progesterone receptor, discover the components that connect the receptor to the protein that activates Eg2, and find out how early phosphorylation of CPEB results in recruitment of the enzyme that adds a long poly(A) tail onto *c-mos* mRNA.

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