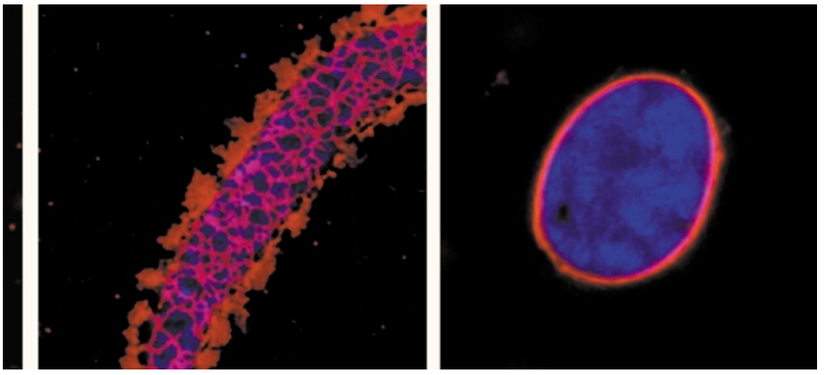

and colleagues concluded that p97 acts together with Ufd1–Npl4 at early stages, up to closure of the nuclear envelope, whereas it functions in combination with p47 at a later stage, during nuclear envelope growth.

The precise mechanism through which p97 acts during nuclear envelope formation is still not clear, but these results provide a starting point for further investigation. In particular, although it is likely that the interaction of p97 with p47 leads to a SNARE-mediated fusion event, as is the case during Golgi or ER fusion, the mechanism by which p97 regulates fusion through the Ufd1–Npl4 complex is a mystery. Indeed, the Ufd1–Npl4 complex has been linked with ubiquitin-dependent protein degradation, so p97 could function differently in combination with this adaptor complex.

Raluca Gagescu

References and links

ORIGINAL RESEARCH PAPER Hetzer, M. *et al.* Distinct AAA-ATPase p97 complexes function in discrete steps of nuclear assembly. *Nature Cell Biol.* **3**, 1086–1091 (2001)

FURTHER READING Burke, B. The nuclear envelope: filling in gaps. *Nature Cell Biol.* **3**, E273–E274 (2001)

intracellular domain of Sef (which contains the conserved tyrosine residue). Although the precise mechanism of Sef action is, as yet, unclear, these two studies have identified a new antagonist of the FGF signalling pathway.

Sarah Greaves, Senior Editor,
Nature Cell Biology

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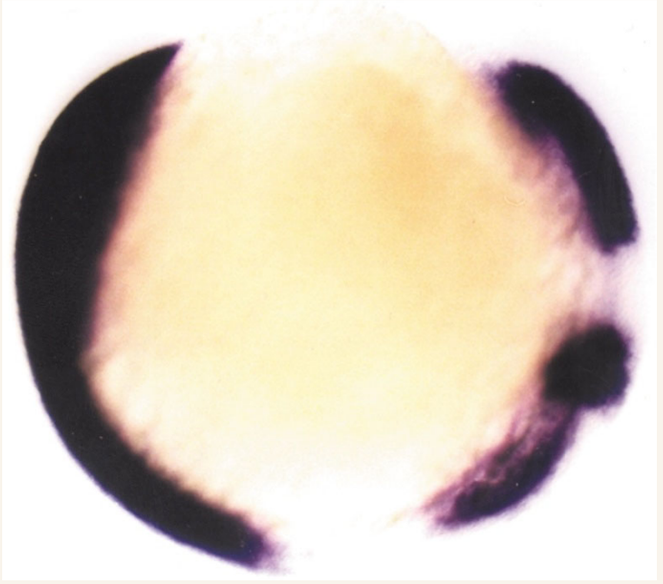
ORIGINAL RESEARCH PAPERS Tsang, M., Friesel, R., Kudoh, T. & Dawid, I. B. Identification of Sef, a novel modulator of FGF signalling. *Nature Cell Biol.* **4**, 165–169 (2002) | Fürthauer, M., Lin, W., Ang, S.-L., Thisse, B. & Thisse. C. Sef is a novel feedback-induced antagonist of Ras/Raf/MEK/MAPK mediated FGF signalling. *Nature Cell Biol.* **4**, 170–174 (2002)

WEB SITE

Encyclopedia of Life Sciences: http://www.els.net/FGF



IN BRIEF

CELL CYCLE

Calcium, calmodulin, and CaMKII requirement for initiation of centrosome duplication in *Xenopus* egg extracts.

Matsumoto, Y. & Maller, J. L. *Science* **295**, 499–502 (2002)

Centrosomes are duplicated once — and only once — per round of cell division. Studies from *Xenopus* egg extracts have shown that the cyclin-dependent kinase 2 (Cdk2) is required for many rounds of centrosome duplication. However, inactivation of Cdk2 does not block the initial duplication event. Matsumoto and Maller now show that chelation of calcium, or specific inactivation of the calcium/calmodulin-dependent protein kinase II (CaMKII), blocks all centrosome duplications, including the first one.

NUCLEAR ARCHITECTURE

Directed proteomic analysis of the human nucleolus.

Andersen, J. S. *et al. Curr. Biol.* **12**, 1–11 (2002)

Paraspeckles: a novel nuclear domain.

Fox, A. H. *et al. Curr. Biol.* **12**, 13–25 (2002)

The most important function of the nucleolus is in the generation of ribosomal subunits, but could it have other functions? To address this, the authors of these papers undertook a proteomic analysis of human nucleoli. They identified over 250 proteins, two-thirds of which (including ribosomal proteins that process factors and other transcriptional components) have functions that are associated with the known role of the nucleolus. Among the new proteins, Paraspeckle protein 1 (PSP1), PSP2 and p54/nrb were found to accumulate in new compartments — paraspeckles — that correspond to discrete regions in the heterochromatin nucleoplasmic space. All primary and transformed cell lines that were studied contained 10–20 paraspeckles. The three proteins move dynamically between paraspeckles and the nucleolus, but relocalize in nucleolar caps within the nucleolus when transcription is blocked. Combined with the fact that they all have related RNA-binding domains, this indicates that they might have a role in transcriptional regulation.

SIGNAL TRANSDUCTION

Leptin stimulates fatty-acid oxidation by activating AMP-activated protein kinase.

Minokoshi, Y. *et al. Nature* **415**, 339–343 (2002)

Leptin is secreted by adipocytes to lower appetite and increase energy expenditure, but the signals that mediate these effects are not well known. Here, the authors report that leptin can prevent lipids accumulating in non-adipose tissue by stimulating the phosphorylation and activation of 5'-AMP-activated protein kinase (AMPK). By inhibiting acetyl coenzyme A carboxylase activity, AMPK stimulates fatty-acid oxidation, initially by a direct effect on the cell, but later requires the hypothalamic–sympathetic nervous system axis.