diphosphate (GDP) or guanosine triphosphate (GTP), and the switch from GDP to GTP is catalysed in the nucleus by an 'exchange factor' called RCC1. Ran–GTP interacts with Importin-β (Fig. 1), causing it to release its import cargo because binding of Importin-β to its cargo and to Ran–GTP are mutually exclusive.

In mitosis, however, the nuclear envelope of vertebrate cells breaks down, and during this time, Importin-β binds and inhibits a variety of 'spindle assembly factors'[3]. As before, Ran–GTP can affect these interactions. It is widely believed that RCC1 binds to chromosomes during mitosis and increases the local concentration of Ran–GTP, thereby activating spindle assembly factors near the chromosomes to guide the spatial organization of the spindle. As a result of its capacity to activate spindle assembly factors, the addition of Ran–GTP to extracts from eggs of *Xenopus laevis* frogs (a popular model system for studying spindle formation) is sufficient in itself to cause the formation of spindle-related microtubule structures called asters in the absence of DNA.

Blower *et al.*[2] used an activity-based assay to purify spindle assembly factors that bind to Importin-β in *Xenopus* egg extracts. They isolated a protein called Rae1. Rae1 was previously shown to be essential for exporting messenger RNAs (mRNAs) from the nucleus in yeast[4], although its precise role is poorly understood. Rae1 localizes to the spindle poles and to chromosomes in egg extracts[2], and its depletion abolished both spindle assembly around sperm DNA and the formation of asters in response to Ran–GTP in DNA-free extracts.

Further analysis of the egg extracts showed that Rae1 associates with a number of proteins that are parts of large RNA–protein complexes called ribonucleoproteins (RNPs)[2]. These RNP components included the Maskin protein, which is recruited to mRNAs to control translation of the RNA code into protein during early *Xenopus* development. To test whether the integrity of the RNPs affects Rae1 function, Blower *et al.* treated egg extracts with an enzyme that destroys RNA (RNase). Remarkably, they found that this treatment disrupted spindle assembly around chromosomes and aster formation in response to Ran–GTP.

Treatment with protein-synthesis inhibitors did not affect spindle formation under the same conditions. The authors therefore argue that the effect they observed is not due to the translation of the mRNAs. However, it should be noted that other researchers have found that protein synthesis may be important later in the process of spindle assembly[5]. Blower and colleagues' findings for the first time implicate RNA as an essential component of the spindle, and imply that RNPs have a structural or regulatory role during early steps in spindle formation that can be distinguished from their role in protein synthesis.

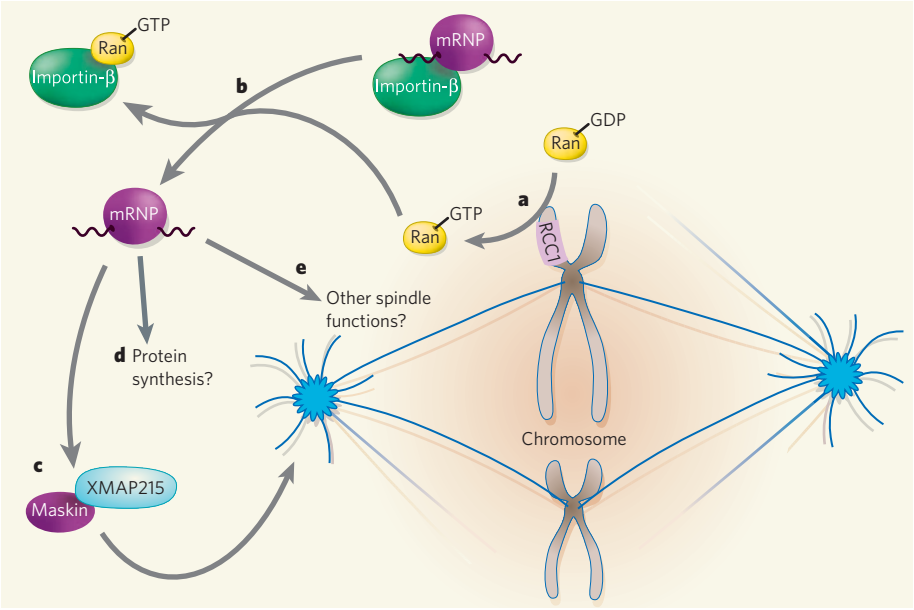

Figure 1 | RNPs and spindle assembly. **a**, Ran–GTP is generated by the chromosome-associated exchange factor, RCC1. **b**, Blower *et al.*[2] find that Ran–GTP releases the direct binding of Importin-β to Rae1-associated messenger RNA–protein complexes (mRNPs). **c–e**, RNPs such as Maskin may have many functions during cell division, including targeting XMAP215 to the spindle poles[3] (**c**), regulation of protein synthesis (**d**) and other structural or regulatory roles in spindle assembly (**e**).

A major question is how RNPs function in spindle assembly. One possible mechanism is suggested by another recent study by O'Brien *et al.*[5], who investigated the role of Maskin in spindle assembly. Maskin is a member of the 'transforming acidic coiled coil' (TACC) protein family, and is the first TACC protein to have been found in *Xenopus*[6]. In mammals and flies, TACC proteins reside at centrosomes, and interact with the XMAP215 and Aurora A proteins[6,7]. XMAP215 is a microtubule-associated protein that modulates microtubule dynamics[8], and Aurora A controls its activity[7]. Aurora A is also a downstream target of Ran, through an Importin-α/β-regulated spindle assembly factor called TPX2 (ref. 7).

O'Brien *et al.*[5] found that Maskin interacts with XMAP215 in a manner that seems similar to how other TACC proteins act: although XMAP215 can bind to microtubules in Maskin-depleted egg extracts, it fails to accumulate at spindle poles. The Maskin-depleted extracts form undersized asters in response to Ran–GTP and assemble highly disorganized spindles around added sperm chromosomes. These findings show that Maskin's control of XMAP215, and perhaps of other spindle components, is essential for proper spindle assembly. Notably, RNase treatment, Maskin depletion and Rae1 depletion give related but distinct outcomes, possibly suggesting that many different RNPs have diverse roles in spindle assembly[2,5].

Collectively, these data hint at a complex web of interactions at spindle poles that are carefully regulated to achieve balanced microtubule assembly and spindle organization. Naturally, many questions remain to be addressed, including how Ran regulates these interactions at such relatively large distances from RCC1 activity on mitotic chromosomes, whether the incorporation of RNPs in the spindle might modulate the translation of individual mRNAs or their distribution to daughter cells, and whether these RNA-based mechanisms also occur in non-embryonic systems and among different species. ■

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CORRECTIONS

● In the News & Views article "Plant biology: Auxin action" by Judy Callis (*Nature* **435,** 436–437; 2005), editorial error introduced the implication that auxins are proteins. They are not. Naturally occurring auxins are low-molecular-weight compounds derived from the amino-acid tryptophan or a tryptophan precursor.

● In "Organic chemistry: Fast reactions 'on water'" by Jaap E. Klijn and Jan B. F. N. Engberts (*Nature* **435,** 746–747; 2005), the page numbers for the main paper under discussion (ref. 2) were wrong in the print edition. The correct reference, which appears in the online edition, is Narayan, S. *et al. Angew. Chem. Int. Edn* **44,** 3275–3279 (2005).