Previous paleobiogeographic analyses were ambiguous as to the region, as many of the primitive branches in the Late Cretaceous Alvarezsauridae were South American, yet *Haplocheirus* was from Central Asia [18]. That these new specimens are from China helps to secure the region of origin for the Alvarezsauria within Asia. That said, given that the alvarezsaurian nature of fragmentary specimens such as *Aorun* and *Tugulusaurus* escaped notice until now suggests the possibility that similar such fossils might exist unrecognized in collections from other parts of the world.

Although the new species increase our knowledge of these intermediate steps in the history the alvarezsaurian dinosaurs, unanswered questions remain. The skull and blade-like teeth of *Haplocheirus* are consistent with it being a feeder on small vertebrates, whereas those of the Late Cretaceous alvarezsaurids appear to have been adapted to insectivory (or possibly herbivory). The skulls of the new species are only poorly known at present, so determining their feeding mode is uncertain. Furthermore, there remains a gap of tens of millions of years between *Haplocheirus* and these later Early Cretaceous species, and between these and their Late Cretaceous kin. Much remains to be uncovered in the evolution of these enigmatic and unusual small dinosaurs.

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Cell Division: Here Comes the Kinesin Cavalry

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A new study finds that a spindle motor makes an unexpected contribution to kinetochore–microtubule attachments and chromosome segregation.

Cell division requires coordination of a complex series of events that lead to one cell becoming two. During cell division, a molecular super-structure called the kinetochore assembles on each sister chromatid and binds to spindle microtubules to ensure that the genome is equally distributed. Under the microscope objective, kinetochores appear as diffraction-limited spots, but one shouldn't be fooled into thinking that all is simple and serene in those spots. In fact, every time a cell divides, a battle is waged at the kinetochore between opposing molecules and the fidelity of chromosome segregation is at stake. Decades of work has revealed the layout and composition of the kinetochore battlefield and the warring parties of kinases and phosphatases. While the battle nearly always concludes in a timely manner with a positive outcome — equal chromosome segregation — it wasn't evident how the cell avoided a stalemate.

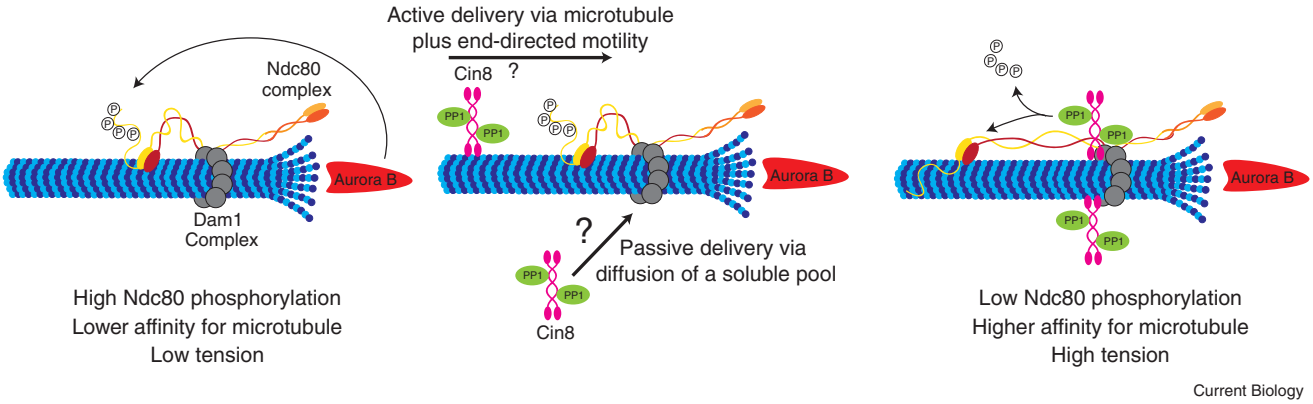

Figure 1. Model for how Cin8 shifts the phospho-regulatory balance in favor of phosphatase activity at kinetochores.
(A) In early mitosis, centromere-enriched Aurora B kinase dominates over phosphatases, resulting in hyper-phosphorylation of the Ndc80 tail, reduced affinity for microtubules, and lower tension at kinetochores. (B) Cin8 shifts the balance in favor of phosphatase activity by bringing PP1 to the kinetochore through two possible non-exclusive mechanisms: active delivery via microtubule plus-end-directed motility or passive diffusion of a soluble pool. (C) Cin8 localization requires the microtubule, the Ndc80 complex, and the Dam1 complex, although the motor directly binds the microtubule and the Dam1 complex. At mature attachments, Cin8-delivered PP1 allows the phosphatase activity to dominate, likely resulting in dephosphorylation of the Ndc80 tail (and other components), increased affinity of the kinetochore for microtubules, stable tension-supporting attachments, and satisfaction of the spindle assembly checkpoint.

In other words, it was unclear what turned the tide. A new study by Suzuki and Gupta *et al.* [1] reported in this issue of *Current Biology* reveals that a kinesin motor brings phosphatase reinforcements to the fight and, in doing so, shifts the balance in favor of stable, tension-supporting kinetochore–microtubule attachments.

The kinetochore is a wonderfully complex structure that fulfills two essential functions during cell division: firstly, as the physical link between DNA and spindle microtubules and, secondly, as the coordinator of a signaling pathway called the spindle assembly checkpoint (SAC) that delays anaphase onset until every chromosome is properly attached to spindle microtubules. A network of proteins mediates the microtubule attachment and SAC regulatory functions of the kinetochore. A central player is the Ndc80 complex, which directly binds to microtubules via a basic unstructured amino-terminal tail and neighboring calponin-homology (CH) domain, and regulates SAC signaling through binding to the checkpoint kinase Mps1 [2]. In budding yeast, the Ndc80 complex also recruits a microtubule-binding, ring-assembling structure called the Dam1 complex to end-on kinetochore–microtubule attachments [3]. The affinities of the Ndc80 and Dam1 complexes for microtubules are reduced by phosphorylation and a major modifier of both complexes is the inner centromere-enriched Aurora B kinase (ABK) [4–6]. Protein phosphatase 1 (PP1) counteracts ABK, and PP1 levels are higher at attached/aligned kinetochores than at unattached/mis-aligned kinetochores [7]. Clearly the balance of ABK and PP1 activities is critically important to cell division, but it has been unclear what events and molecules tip the balance of power between these two opposing activities.

Apart from kinetochore proteins, motor proteins also play an important role in cell division. Kinesin-5 motors function during mitosis by cross-linking microtubules and sliding anti-parallel microtubules apart to promote spindle bipolarity [8]. Budding yeast has two kinesin-5 family members (Cin8 and Kip1) with redundant bipolarity-supporting functions [9], although Cin8 (but not Kip1) has also been implicated in regulating the SAC [10]. In this issue of *Current Biology*, Suzuki and Gupta *et al.* define the molecular basis of the Cin8 SAC phenotype and uncover an important role for the kinesin-5 motor at kinetochores. In pairing elegant live-cell imaging and *in vitro* techniques, the research team was able to explain how a moving piece of the kinetochore puzzle plays a central role in the phospho-regulation of kinetochore–microtubule attachments.

Cin8 localized to kinetochores throughout the cell cycle, peaking at metaphase, when it localized near the amino terminus of the Ndc80 complex, and dropped significantly during anaphase. Its kinetochore localization was dynamic (t1/2 = ~40 s) and depended on microtubules, the Ndc80 complex, and the Dam1 complex. Interestingly, Cin8 directly interacted with the Dam1 complex, but not the Ndc80 complex *in vitro*. Since Dam1 complex recruitment to the kinetochore requires the Ndc80 complex, the requirement of Ndc80 for Cin8 localization is probably indirect. Given that Cin8 has a microtubule-binding motor domain, the main contact points responsible for its kinetochore enrichment are likely the microtubule and the Dam1 complex, although the precise nature of Cin8 recruitment warrants further research. Along these lines, the authors posited that there may be multiple Cin8-binding proteins at kinetochores since some Cin8 remained at kinetochores in absence of Dam1.

In order to characterize Cin8 kinetochore functions, Suzuki and Gupta studied *Cin8Δ* cells that, in agreement with prior studies [9,11], were significantly delayed in mitosis. To explain why there was a delay in SAC satisfaction in the absence of Cin8, the research team employed a powerful tool that they had previously developed [12]: a FRET-based tension sensor inserted into Ndc80 that exhibits higher FRET under low/no tension and reduced FRET when Ndc80 is under tension. Interestingly, the Ndc80 biosensor was under significantly less tension (approaching zero tension) in

mitosis in *Cin8Δ* cells compared to control cells. The amino-terminal tail of Ndc80 is required to support tension [12] and cells expressing an Ndc80 tailless mutant phenocopied *Cin8Δ* in that both mutants were delayed in mitosis with very low Ndc80 tension. Since double mutants (*Cin8Δ* + Ndc80 tailless) had the same mitotic delay as either single mutant, Suzuki and Gupta hypothesized that Cin8 was most likely regulating Ndc80 amino-terminal tail function.

How does Cin8 contribute to the generation of Ndc80 tension? First, Suzuki and Gupta investigated if kinetochore assembly was defective in *Cin8Δ* cells and found no change in the stoichiometry of core components in purified kinetochores. Additionally, FRAP measurements revealed that microtubule dynamicity was unaffected in *Cin8Δ cells*. Although further investigation into kinetochore–microtubule attachment stability in the absence of Cin8 is needed, the microtubule dynamicity measurements indicated that Ndc80 tension was low even in the presence of functional end-on kinetochore–microtubule attachments. The authors reasoned that perhaps Cin8 could affect phospho-regulation of the Ndc80 complex and found that Cin8 contained a putative PP1-binding motif. Indeed, PP1 co-immunoprecipitated with Cin8 and its association with both Cin8 and purified kinetochores was lost upon mutating the PP1 binding motif in Cin8. A Cin8 PP1-binding motif mutant phenocopied the mitotic delay observed in *Cin8Δ* mutants and these cells exhibited a comparable loss of Ndc80 tension to *Cin8Δ* and Ndc80 amino-terminal tailless mutants that don't support tension.

Early in mitosis, phosphorylation of kinetochore components dominates, which reduces the kinetochore's affinity for microtubules and contributes to SAC signaling. As mitosis progresses, PP1 becomes enriched on aligned kinetochores, counteracting Mps1 kinase to promote SAC satisfaction and likely increasing the affinity of the Ndc80 complex for microtubules by countering ABK. Since multiple kinetochore components, most notably KNL1 (Spc105 in budding yeast), recruit PP1 during mitosis [13–16], it was surprising that Suzuki and Gupta found that PP1 was completely absent from purified kinetochores in *Cin8Δ* cells. Like Ndc80, KNL1 is a substrate of the ABK–PP1 phospho-regulatory loop such that ABK-mediated phosphorylation of an amino-terminal PP1-binding motif in KNL1 reduces its affinity for PP1 [13]. The absence of kinetochore-bound PP1 in *Cin8Δ* cells supports the hypothesis that Spc105 at low tension kinetochores cannot bind PP1, most likely due to ABK activity, and would suggest that Cin8 (or an analogous motor in other cell types) is the key component that shifts the balance away from ABK and in favor of PP1. Once Cin8 delivers its initial shots of PP1, Ndc80 may be able to establish stronger tension-supporting microtubule attachments that, in turn, liberate Spc105 to recruit more PP1 that strengthens existing attachments and contributes to silencing the SAC by opposing Mps1 activity (Figure 1) [17]. More work is certainly needed to flesh out the details, but these findings provide a new way to think about the contribution of motor proteins to kinetochore–microtubule attachment stability and checkpoint satisfaction.

While Suzuki and Gupta found that a Cin8 motor domain mutant with reduced affinity for microtubules still co-immunoprecipitated PP1 from yeast cell extracts, the contribution of Cin8 motility to Ndc80 tension and mitotic progression was not directly investigated. The role of motility in the motor–PP1 regulatory pathway is an open question since kinesin-8 motors in fission yeast bind PP1 and contribute to spindle assembly checkpoint satisfaction independent of motor activity [14]. Motor-based PP1 delivery to the kinetochore is conserved outside budding and fission yeast as human kinesin-8 (Kif18A) directly interacts with PP1 [18] and a recent study found that the human Kif18A–PP1 complex functions similarly to Cin8-PP1 in promoting stable kinetochore-microtubule attachments by dephosphorylating Ndc80 [19]. The Dam1 complex, which is found in budding and fission yeast, is the main Cin8-binding partner. Since humans do not have a Dam1 complex, delivery of PP1 to human kinetochores may depend to a greater extent on Kif18A motility or use a different kinetochore receptor. Regardless of whether kinesin–PP1 complexes arrive at the kinetochore via active delivery or passive diffusion, the kinesin cavalry is coming and it will turn the tide towards stable, tension-supporting kinetochore–microtubule attachments.

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Reproductive Strategies: Eat Your Kids to Restart Your Sex Life

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A recent study shows that filial cannibalism is not merely a desperate survival tactic for hungry fathers. Rather, brood destruction triggers sexual physiology, enabling males to restart courtship with new partners. Like abandonment and infanticide by males and females across taxa, cannibalism generates evolutionary conflict between fathers, mothers, and offspring.

One of European art's most shocking images is Goya's depiction of the Titan Cronos devouring his children (Figure 1). The term 'filial cannibalism' conjures something so desperate and beyond the moral pale as to defy explanation. Animal behavior is interesting in no small part because it holds up a mirror to human experience, but giving technical meaning to fraught terms — choice, rape, cuckoldry, divorce — makes for what Bateson [1] called "unconscious punning" and a tendency to seek special explanations for behaviors that evoke abhorrence.

Infanticide is a case in point: until Sarah Blaffer Hrdy's [2] seminal papers in the late 1970s, scientists strove to explain away the killing of dependent offspring as a rare, pathological behavior. Hrdy proposed a classic set of testable hypotheses for the adaptive benefits of infanticide; for genetic parents, it pays to kill (or abandon) young offspring and restart reproduction if the costs associated with offspring care exceed the benefits. Filial cannibalism — eating one's genetic progeny — is a form of infanticide found across taxa, and is particularly well-documented in fishes with male parental care. In many species, individual males defend nests or spawning sites. One or more females spawn with the male in his territory, leaving him to care for the young. Parental care often involves little more than keeping intruders away from the nest, particularly other fish species likely to eat eggs [3].

Yet Dad is often the worst predator of his own eggs. Partial filial cannibalism, like parental infanticide in birds [4], may be beneficial for both parents if thinning the brood increases average offspring fitness, for example, by increasing oxygen availability [5] or skewing sex ratios [4]. But what about total filial cannibalism, in which a male consumes the whole brood? There is abundant evidence that less-valuable clutches — those that are smaller, earlier in development [6], or of mixed paternity [7] — are more likely to be cannibalized.

Why do males eat these eggs instead of just soliciting matings from more females? The consensus until now has been that cannibalism is driven by the direct benefit of eating eggs: a caviar snack serves as an alternative food source for hungry males. Total filial cannibalism is adaptive if the nutritional benefit of eating eggs, applied to future courting and guarding, has a greater fitness benefit than guarding the current clutch. It therefore pays a father to reap the energy benefit from the eggs, at the expense of mother and progeny [6]. The unique prediction of the so-called energy-based hypothesis is that cannibalism will be sensitive to nutritional state and food availability: hungrier males will be more likely to cannibalize, and cannibalism will confer a nutritional benefit. This prediction is poorly supported by empirical data [8].