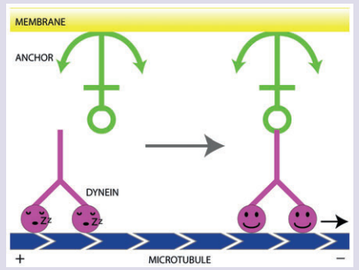
Highlights from this issue

Regulation of cytoplasmic dynein by anchor proteins

Vaishnavi Ananthanarayanan reviews how cytoplasmic dynein – the primary minus-end directed motor protein in the cell – is regulated (see pages 514–525). Anchor proteins play a vital role in this process. The fungal proteins Num1 (*S. cerevisiae*), Mcp5 (*S. pombe*) and ApsA (*A. nidulans*), for example, ensure binding of dynein to the cell membrane, a requirement for nuclear organization. The author also discusses possible mechanisms by which the regulation occurs – not only in these fungal model organisms but also in metazoans such as flies, worms and mammals. – *kb*

Highlighted article: Activation of the motor protein upon attachment: Anchors weigh in on cytoplasmic dynein regulation. Vaishnavi Ananthanarayanan ↗ dx.doi.org/10.1002/bies.201600002



Mitochondrial DNA: Of cells, organisms and the tragedy of the cytoplasmic commons

On pages 549–555, David Haig reviews the evolution of mitochondrial DNA at different levels: from the cellular to the organismal level, and between germline and somatic cells. He uses a nice analogy by likening the mitochondria/mitochondrial DNA within a cell to a herd and the nuclear factor regulators to herdsmen. As mitochondria are predominantly transmitted maternally, female germ cells can then be regarded as the stud farms that stock the mitochondrial herds of the next generation. The author also draws an analogy to the tragedy of the commons; replication is a private good of individual mtDNAs, but an individual mtDNA's contribution to cellular (or even organismal) function and fitness is shared. However, this problem has been solved for most mitochondrial genes by their transfer to the nucleus. – *kb*

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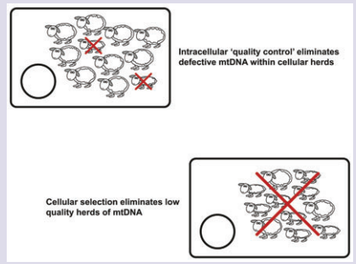
Cover Photograph

Evolutionary conservation of hindbrain segmentation in vertebrates. On pages 526–538 of this issue, Parker *et al.* explore the origin and diversification of the *Hox* gene regulatory network for vertebrate hindbrain segmentation and patterning. The cover depicts transgenic embryos of three different species – sea lamprey, mouse and zebrafish – expressing fluorescent proteins in the developing hindbrain through the activity of segment-specific *cis*-regulatory elements. Segmental enhancers from mouse and zebrafish can function when introduced into lamprey, highlighting the conservation of this gene regulatory network to the vertebrate base. Photo credits: Hugo Parker and Mark Parrish. Illustration: Mark Miller.

Highlighted article: Intracellular evolution of mitochondrial DNA (mtDNA) and the tragedy of the cytoplasmic commons. David Haig ↗ dx.doi.org/10.1002/bies.201600003



Unraveling the basis of complex diseases

Aravinda Chakravarti and Tychele Turner propose that studying rare, extreme-phenotype families may help the understanding of complex diseases (see pages 578–586). These diseases are characterized by the interplay of multiple genes often in combination with lifestyle and environmental factors. The authors hypothesize that these various genes are all functionally united through gene regulatory networks (GRNs). The mutant phenotype consequently arises from the perturbation of one or more rate-limiting steps and this in turn affects the function of the entire GRN. The authors suggest that genomic analyses of extreme phenotypes represent a valuable tool to identify these GRNs. – *kb*

Highlighted article: Revealing rate-limiting steps in complex disease biology: The crucial importance of studying rare, extreme-phenotype families. Aravinda Chakravarti and Tychele N. Turner ↗ dx.doi.org/10.1002/bies.201500203