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Centrosomes in asymmetric cell division

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Asymmetric cell division (ACD) is a strategy for achieving cell diversity. Research carried out over the last two decades has shown that in some cell types that divide asymmetrically, mother and daughter centrosomes are noticeably different from one another in structure, behaviour, and fate, and that robust ACD depends upon centrosome function. Here, I review the latest advances in this field with special emphasis on the complex structure-function relationship of centrosomes with regards to ACD and on mechanistic insight derived from cell types that divide symmetrically but is likely to be relevant in ACD. I also include a comment arguing for the need to investigate the centrosome cycle in other cell types that divide asymmetrically.

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Introduction

This short piece is intended to offer a concise view of the most recent articles published on ‘centrosomes in asymmetric cell division’. It is therefore not suitable as an introduction to this subject, which can be obtained from previous, comprehensive reviews on asymmetric cell division or centrosomes in general [1–5] and others on the specific subject of centrosomes in asymmetric cell division [6–9].

Pioneer work on diving grasshopper neuroblasts showing that unequal asters bring about unequally sized daughter cells was probably the first hint that centrosome activity may be tailored to the specific needs of cells that divide asymmetrically [10]. This was solidly substantiated two decades later by the discovery that mother and daughter

centrosomes segregate according to cell fate and have different roles in ACD in *Drosophila* germline stem cells (GSCs) and neuroblasts as well as neural progenitors in vertebrates (NBs) [11–15].

Although belonging to different lineages from different species these cells share four traits that altogether make them distinct: (i) they undergo a particular type of ACD in which one of the daughters is a renewed version of the mother and can go through repeated rounds of such a ‘self-renewing’ ACD; (ii) during interphase the centrosome is close to the cell membrane, away from the nucleus; (iii) the interaction between centrosome and plasma membrane takes place at the apical side, which is fated to remain in the daughter cell that becomes the renewed stem/progenitor; and (iv) the fate of each centrosome correlates tightly with centrosome age.

Relevant news from ‘symmetric’ divisions

The last few years have seen a great deal of new insight on centriole duplication, basal body function, PCM assembly, and so on that derive from cell types that divide symmetrically but may eventually help to understand centrosome asymmetry in the context of ACD [16–23]. A remarkable example is the new concept of PCM as a molecular assembly formed via liquid–liquid phase separation [24**]. This exciting conceptual revolution is a paradigm change that is already exerting a strong influence on centrosome biology [25] and ACD (reviewed in Ref. [26]) and may be specially relevant in asymmetric centrosome cycles where mother and daughter centrosomes become conspicuously unequal as far as PCM recruitment is concerned. The reader is also referred to an interesting article adding a pertinent note of caution [27*].

Another one is targeted co-translation. In different zebrafish and human cell types Pericentrin (PCNT), Abnormal Spindle Microcephaly-related (ASPM), Hyaluronan Mediated Motility Receptor (HMMR), and Nuclear Mitotic Apparatus Protein 1 (NUMA1) mRNAs have been found to localize to the centrosome where they are translated during mitosis [28**,29**]. In situ translation of key scaffold proteins may optimise centrosome maturation not only by speeding up their arrival at the destination but also by facilitating specific protein interactions that may help folding and protein-complex assembly. Moreover, targeted translation minimises the chances of assembly of ectopic PCM aggregates. It is tempting to speculate that such supramolecular ribonucleoprotein aggregates made of mRNAs, ribosomes and nascent peptides may also have a significant role in PCM phase

