

SPOTLIGHT

Satellite view: Dissection of PCM1 assembly processes reveals centriolar satellite structures

Ruth M. Kearney^{1*} , Anushka Sharma^{1*} , and Ciaran G. Morrison¹ 

Centriolar satellites (CS) are dynamic and heterogeneous granular assemblies that concentrate around centrosomes and contribute to ciliogenesis. In this issue, Begar et al. (<https://doi.org/10.1083/jcb.202509238>) examine the CS scaffold protein PCM1 to dissect CS assembly and structure during the cell cycle and ciliogenesis.

Centriolar satellites (CS) are nonmembranous, granular protein assemblies that concentrate around centrosomes during interphase (1). They help to move associated client proteins toward the centrosome along microtubules (MTs) and are required for efficient ciliogenesis (2), with further roles in cell stress responses (3) and protein quality control (4). CS can vary in size, although generally described as being 70–100 nm in diameter, and subcellular location, even within the same cell. They are highly dynamic, being disassembled during mitosis (5), and changing their composition and subcellular localization during ciliogenesis (6) and in response to cell stress (3). These complexities mean that significant questions remain open regarding molecular details of the composition, assembly mechanism, size regulation, and dynamic positioning of CS. In this issue of *JCB*, Begar, Firat-Karalar, and colleagues present a detailed analysis of CS that reveals their underlying architecture and assembly (7).

CS are defined by the large (2024 amino acids) protein, PCM1, which acts as a scaffold and is required for their formation (Fig. 1) (5, 6, 8). PCM1-deficient animal models show developmental and cognitive deficiencies that reflect defects in CS-regulated processes (2), such as ciliogenesis, consistent with the implication of CS gene mutations in such disorders in humans. Association with PCM1 has been used to identify CS components, and proteomic analyses have revealed the complex makeup

of CS, with upwards of 600 potential interactors of known CS components. However, the extent and duration of some interactions may be limited so that CS membership remains to be confirmed for a large number of candidates (9, 10). Furthermore, CS are not homogeneous, with different satellites containing different protein subpopulations (9).

Begar et al. expressed truncation mutants of PCM1 in PCM1 knockout cells to define large subregions of the protein that are required for CS granule assembly. They show that the N-terminal 1,200 amino acids are key for the multimeric assembly that underpins CS, consistent with previous work highlighting that this part of PCM1 contains regions needed for multimerization (5, 6). Forced dimerization of PCM1 drove its assembly into fewer, larger granules. These granules allowed formation of cilia, albeit with defective signaling capacity. They also persisted during and interfered with mitosis, demonstrating the importance of a regulated assembly process for PCM1 multimers.

Tet-inducible PCM1 expression in PCM1 null cells was then used to analyze the de novo assembly of CS, an insightful approach that circumvented the technical challenges of visualizing these small and dynamic structures. Full-length PCM1 rescued ciliary assembly and function, although the N-terminal region alone did not support fully functional cilia, despite allowing the formation of CS granules and cilia. Combining the inducible expression of PCM1 with time-lapse microscopy, Begar et al.

followed the kinetics of CS assembly. They observed a triphasic process, in terms of granule number and size, of centrosome-proximal initiation, growth, and plateau. The truncation mutants assembled fewer, larger granules, showing that CS regulatory elements lie within full-length PCM1. Next, Begar et al. used this inducible CS assembly model to follow how nine CS client proteins associate with PCM1 scaffolds over time. Strikingly, they found an ordered recruitment that points to a structured and regulated assembly of CS through PCM1 association of clients. In addition, the properties of CS granules change over time, transitioning from a dynamic initial phase to increasingly stable, mature structures.

Testing the hypothesis that this ordered recruitment indicated a defined internal organization of the satellites, antibodies raised to different regions of PCM1 were used in expansion microscopy experiments to reveal the regional substructure of CS. The location of individual satellite client proteins was then mapped onto PCM1. Client proteins showed discrete localization patterns within individual PCM1 scaffolds, providing further evidence for CS having a defined structure. For technical reasons, larger granules formed by the N-terminal region of PCM1 were examined in more detail, allowing a time- and space-defined map of client assembly to be generated. Begar et al. propose an underlying architecture for CS structures that invokes compartmentalization, a notion of potential relevance in

¹Centre for Chromosome Biology, School of Biological and Chemical Sciences, University of Galway, Galway, Ireland.

*R.M. Kearney and A. Sharma contributed equally to this paper. Correspondence to Ciaran G. Morrison: ciaran.morrison@universityofgalway.ie.

© 2026 Kearney et al. This article is distributed under the terms as described at <https://rupress.org/pages/terms102024/>.

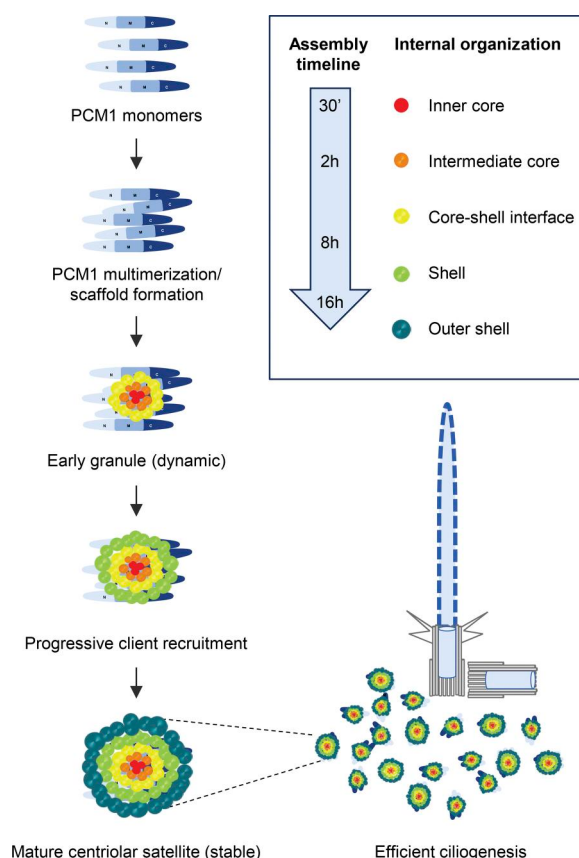


Figure 1. Model for hierarchical assembly of CS driven by PCM1 multimerization. The cartoon shows how the initial multimerization of PCM1 provides a scaffold for the ordered assembly of CS components, as described by Begar and colleagues (7). PCM1 monomers self-associate to initiate scaffold formation, followed by early granule formation (30'), progressive recruitment of client proteins (2–16 h), and maturation into larger, stable satellite assemblies. An internal organization of mature satellites is shown with a layered architecture consisting of an inner core (shown in red), intermediate zone (orange), core-shell interface (yellow), shell (light green), and outer shell (dark green). The components of the different layers do not all assemble at the same time. Mature CS localize around the centrosome and facilitate ciliogenesis.

terms of regulation of CS functions. Interestingly, distinct changes in CS organization were observed during ciliogenesis and after DNA-damaging treatment, indicative of regulated CS responses at the structural level.

In support of their analysis of PCM1's cellular roles, Begar and colleagues observed concentration-dependent, *in vitro* assembly of recombinant PCM1 into granule-like assemblies at physiological concentrations. These granules bound to the CS client protein, MIB1, and to MTs *in vitro*, forming larger structures in the presence of stabilized MTs. Pharmacological disruption of MTs in cells disrupted *de novo* CS assembly,

dispersing the satellites away from the centrosome, consistent with an initial PCM1 scaffold nucleation event being facilitated by MTs.

PCM1 contains few defined protein motifs, but many intrinsically disordered regions, which flag its potential for assembly through a condensation mechanism (11). Begar et al. noted only a limited impact on PCM1 granules of chemical or enzymatic treatments that disrupted condensates such as stress granules, although there were some differences in behavior among the client proteins examined. This suggests that CS are not wholly dynamic or soluble aggregates and, combined with the authors' findings of

an ordered assembly process, underscores the need to define further the PCM1-scaffolded mechanism of CS formation. Clearly, additional challenges lie in understanding how CS interact with other nonmembranous organelles, notably the pericentriolar material, during ciliogenesis, in cellular stress responses, or in developmental contexts in which the satellites play important roles. A possibility is that human disease may result not only from deficiency of satellite-associated proteins, but also from subtle perturbations in CS assembly dynamics that compromise their functional integrity.

Acknowledgments

The authors declare no competing interests exist. This publication has emanated from research conducted with the financial support of Taighde Éireann/Research Ireland under grant number 23/FFP-A/11620.

Author contributions: Ruth M. Kearney: conceptualization and writing—original draft, review, and editing. Anushka Sharma: conceptualization and writing—original draft, review, and editing. Ciaran Morrison: conceptualization, funding acquisition, supervision, and writing—original draft, review, and editing.

Disclosures: The authors declare no competing interests exist.

References

- Prosser, S.L., and L. Pelletier. 2020. *J. Cell Sci.* <https://doi.org/10.1242/jcs.239566>
- Hall, E.A., et al. 2023. *Elife.* <https://doi.org/10.7554/eLife.79299>
- Villumsen, B.H., et al. 2013. *EMBO J.* <https://doi.org/10.1038/emboj.2013.223>
- Prosser, S.L., et al. 2022. *Nat. Cell Biol.* <https://doi.org/10.1038/s41556-022-00869-0>
- Dammermann, A., and A. Merdes. 2002. *J. Cell Biol.* <https://doi.org/10.1083/jcb.200204023>
- Kubo, A., et al. 1999. *J. Cell Biol.* <https://doi.org/10.1083/jcb.147.5.969>
- Begar, E., et al. 2026. *J. Cell Biol.* <https://doi.org/10.1083/jcb.202509238>
- Odabasi, E., et al. 2019. *EMBO Rep.* <https://doi.org/10.15252/embr.201947723>
- Gheiratmand, L., et al. 2019. *EMBO J.* <https://doi.org/10.15252/emboj.2018101109>
- Quarantotti, V., et al. 2019. *EMBO J.* <https://doi.org/10.15252/emboj.2018101082>
- Banani, S.F., et al. 2017. *Nat. Rev. Mol. Cell Biol.* <https://doi.org/10.1038/nrm.2017.7>