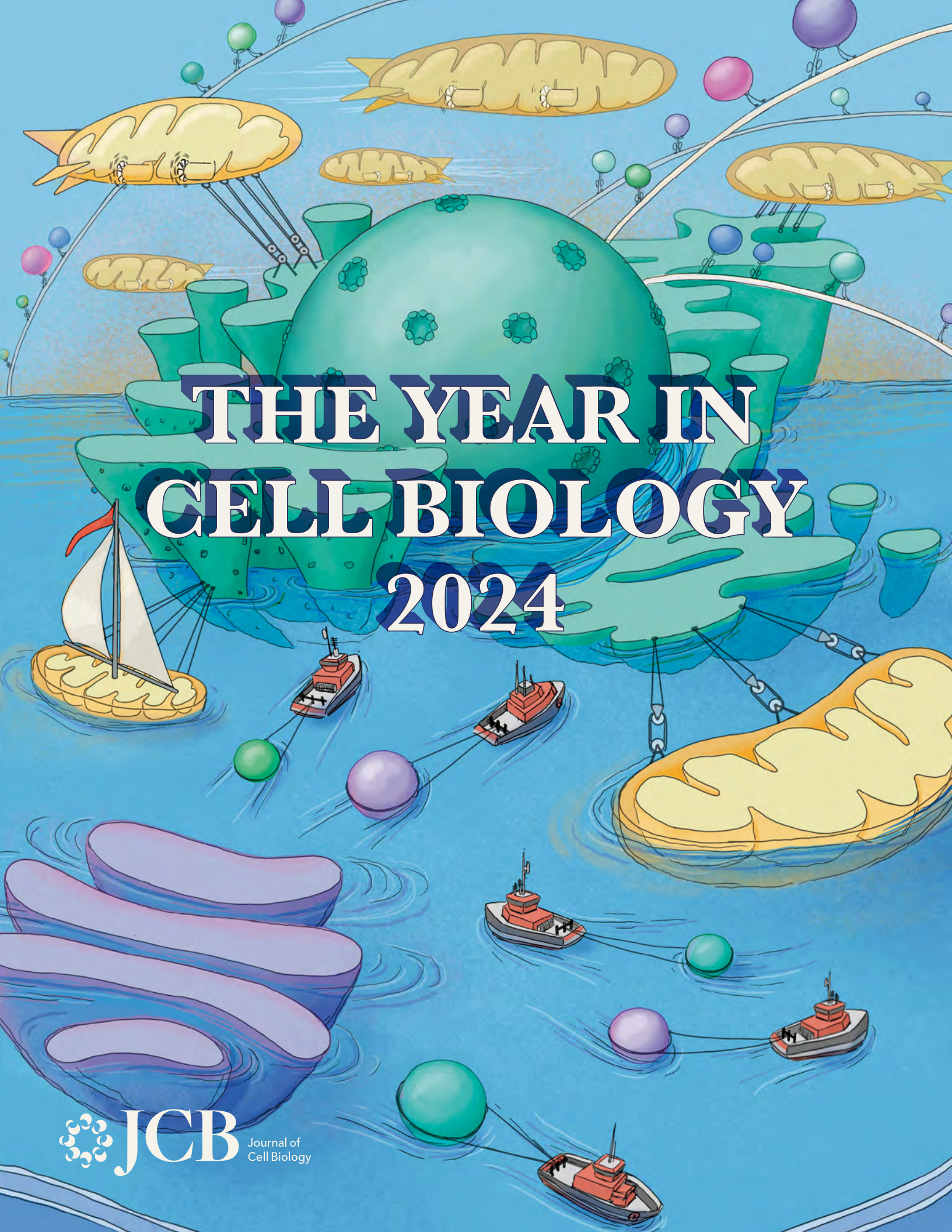

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Print ISSN: 0021-9525.
Online ISSN: 1540-8140

Rockefeller University Press

The Year In Cell Biology 2024

The *Journal of Cell Biology* is pleased to present our annual collection of outstanding papers that most captivated our readers over the past year.

These papers showcase the exceptional range and quality of research published by *JCB*, from organelle repair and contact sites to bacterial wall synthesis to dietary influences on the heart. These articles convey exciting new discoveries in neuroscience, plant biology, and cell mechanics, as well as a method to detect phosphoinositides in living cells. Overall, these findings, which reveal the structures and operations within and among cells, illustrate the power of understanding the living world from a cellular perspective.

The editorial team at *JCB* is honored by the enduring interest of our readers, the dedication and rigor of our reviewers, and of course the trust that our authors place in *JCB* as a premier outlet for their most important findings. We are delighted to have served as an independent venue for the awe-inspiring world of cell biology for 70 years, and we look forward to publishing more exciting discoveries in 2025.

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NUCLEAR HUNTINGTIN AGGREGATES RUPTURE THE NUCLEAR ENVELOPE

Huntington's disease (HD) is caused by a polyglutamine expansion of the huntingtin protein, resulting in the formation of polyglutamine aggregates. The mechanisms of toxicity that result in the complex HD pathology remain only partially understood.

We show that nuclear polyglutamine aggregates induce nuclear envelope (NE) blebbing and ruptures that are often repaired incompletely. These ruptures coincide with disruptions of the nuclear lamina and lead to lamina scar formation. Expansion microscopy enabled resolving the ultrastructure of nuclear aggregates and revealed polyglutamine fibrils sticking into the cytosol at rupture sites, suggesting a mechanism for incomplete repair. Furthermore, we found that NE repair factors often accumulated near nuclear aggregates, consistent with stalled repair.

These findings implicate nuclear polyQ aggregate-induced loss of NE integrity as a potential contributing factor to Huntington's disease and other polyglutamine diseases.

ORIGINAL PAPER

Korsten, G., M. Osinga, R.A. Pelle, A.K. Serweta, B. Hoogenberg, H.H. Kampinga, and L.C. Kapitein. 2024. Nuclear poly-glutamine aggregates rupture the nuclear envelope and hinder its repair. *J. Cell Biol.* 223 (11): e202307142. https://doi.org/10.1083/jcb.202307142

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A PLANT TRANSCRIPTION FACTOR FORMS LIGHT-INDUCED NUCLEAR CONDENSATES

The functional importance of nuclear protein condensation remains often unclear. The bHLH FER-like iron deficiency-induced transcription factor (FIT) controls iron acquisition and growth in plants. Previously described C-terminal serine residues allow FIT to interact and form active transcription factor complexes with subgroup Ib bHLH factors such as bHLH039. FIT has lower nuclear mobility than mutant FITmSS271AA.

We show that FIT undergoes a light-inducible subnuclear partitioning into FIT nuclear bodies (NBs). Using quantitative and qualitative microscopy-based approaches, we characterized FIT NBs as condensates that were reversible and likely formed by liquid–liquid phase separation. FIT accumulated preferentially in NBs versus nucleoplasm when engaged in protein complexes with itself and with bHLH039. FITmSS271AA, instead, localized to NBs with different dynamics.

FIT colocalized with splicing and light signaling NB markers. The NB-inducing light conditions were linked with active FIT and elevated FIT target gene expression in roots. FIT condensation may affect nuclear mobility and be relevant for integrating environmental and Fe nutrition signals.

ORIGINAL PAPER

Trofimov, K., R. Gratz, R. Ivanov, Y. Stahl, P. Bauer, and T. Brumbarova. 2024. FER-like iron deficiency-induced transcription factor (FIT) accumulates in nuclear condensates. *J. Cell Biol.* 223 (4): e202311048. https://doi.org/10.1083/jcb.202311048

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TRIPARTITE MEMBRANE CONTACTS REGULATE MITOCHONDRIAL DYNAMICS AND PI(4)P DISTRIBUTION

The mitochondria–ER–cortex anchor (MECA) forms a tripartite membrane contact site between mitochondria, the endoplasmic reticulum (ER), and the plasma membrane (PM). The core component of MECA, Num1, interacts with the PM and mitochondria via two distinct lipid-binding domains; however, the molecular mechanism by which Num1 interacts with the ER is unclear.

We demonstrate that Num1 contains a FFAT motif in its C-terminus that interacts with the integral ER membrane protein Scs2. While dispensable for Num1's functions in mitochondrial tethering and dynein anchoring, the FFAT motif is required for Num1's role in promoting mitochondrial division. Unexpectedly, we also reveal a novel function of MECA in regulating the distribution of phosphatidylinositol-4-phosphate (PI(4)P). Breaking Num1 association with any of the three membranes it tethers results in an accumulation of PI(4)P on the PM, likely via disrupting Sac1-mediated PI(4)P turnover.

This work establishes MECA as an important regulatory hub that spatially organizes mitochondria, ER, and PM to coordinate crucial cellular functions.

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ORIGINAL PAPER
Casler, J.C., C.S. Harper, A.J. White, H.L. Anderson, and L.L. Lackner. 2024. Mitochondria–ER–PM contacts regulate mitochondrial division and PI(4)P distribution. *J. Cell Biol.* 223 (9): e202308144. https://doi.org/10.1083/jcb.202308144

APOE REGULATES LIPID DROPLET SIZE AND COMPOSITION IN ASTROCYTES

The *E4* variant of *APOE* strongly predisposes individuals to late-onset Alzheimer's disease. We demonstrate that in response to lipogenesis, apolipoprotein E (APOE) in astrocytes can avoid translocation into the endoplasmic reticulum (ER) lumen and traffic to lipid droplets (LDs) via membrane bridges at ER–LD contacts.

APOE knockdown promotes fewer, larger LDs after a fatty acid pulse, which contain more unsaturated triglyceride after fatty acid pulse-chase. This LD size phenotype was rescued by chimeric APOE that targets only LDs. Like APOE depletion, APOE4-expressing astrocytes form a small number of large LDs enriched in unsaturated triglyceride. Additionally, the LDs in *APOE4* cells exhibit impaired turnover and increased sensitivity to lipid peroxidation.

Our data indicate that APOE plays a previously unrecognized role as an LD surface protein that regulates LD size and composition. *APOE4* causes aberrant LD composition and morphology. Our study contributes to accumulating evidence that *APOE4* astrocytes with large, unsaturated LDs are sensitized to lipid peroxidation, which could contribute to Alzheimer's disease risk.

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ORIGINAL PAPER
Windham, I.A., A.E. Powers, J.V. Ragusa, E.D. Wallace, M.C. Zanellati, V.H. Williams, C.H. Wagner, K.K. White, and S. Cohen. 2024. APOE traffics to astrocyte lipid droplets and modulates triglyceride saturation and droplet size. *J. Cell Biol.* 223 (4): e202305003. https://doi.org/10.1083/jcb.202305003

FzlA COORDINATES *CAULOBACTER* DIVISION

To divide, bacteria must synthesize their peptidoglycan (PG) cell wall, a protective meshwork that maintains cell shape. FtsZ, a tubulin homolog, dynamically assembles into a midcell band, recruiting division proteins, including the PG synthases FtsW and FtsI. FtsWI are activated to synthesize PG and drive constriction at the appropriate time and place. However, their activation pathway remains unresolved.

In *Caulobacter crescentus*, FtsWI activity requires FzlA, an essential FtsZ-binding protein. Through time-lapse imaging and single-molecule tracking of *Caulobacter* FtsW and FzlA, we demonstrate that FzlA is a limiting constriction activation factor that signals to promote conversion of inactive FtsW to an active, slow-moving state. We find that FzlA interacts with the DNA translocase FtsK and place FtsK genetically in a pathway with FzlA and FtsWI. Misregulation of the FzlA-FtsK-FtsWI pathway leads to heightened DNA damage and cell death.

We propose that FzlA integrates the FtsZ ring, chromosome segregation, and PG synthesis to ensure robust and timely constriction during *Caulobacter* division.

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ORIGINAL PAPER
Mahone, C.R., I.P. Payne, Z. Lyu, J.W. McCausland, J.M. Barrows, J. Xiao, X. Yang, and E.D. Goley. 2024. Integration of cell wall synthesis and chromosome segregation during cell division in *Caulobacter*. *J. Cell Biol.* 223 (2): e202211026. https://doi.org/10.1083/jcb.202211026

GDP-TUBULIN POLYMERIZES INTO STABLE MICROTUBULES

Microtubules are dynamic polymers that interconvert between phases of growth and shrinkage, yet they provide structural stability to cells. Growth involves hydrolysis of GTP-tubulin to GDP-tubulin, which releases energy that is stored within the microtubule lattice and destabilizes it; a GTP cap at microtubule ends is thought to prevent GDP subunits from rapidly dissociating and causing catastrophe.

Using in vitro reconstitution assays, we show that GDP-tubulin, usually considered inactive, can itself assemble into microtubules, preferentially at the minus end, and promote persistent growth. GDP-tubulin-assembled microtubules are highly stable, displaying no detectable spontaneous shrinkage. Strikingly, islands of GDP-tubulin within dynamic microtubules stop shrinkage events and promote rescues.

Microtubules thus possess an intrinsic capacity for stability, independent of accessory proteins. This finding provides novel mechanisms to explain microtubule dynamics.

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ORIGINAL PAPER
Bagdadi, N., J. Wu, J. Delaroche, L. Serre, C. Delphin, M. De Andrade, M. Carcel, H. Nawabi, B. Pinson, C. Vérin, Y. Couté, S. Gory-Fauré, A. Andrieux, V. Stoppin-Mellet, and I. Arnal. 2024. Stable GDP-tubulin islands rescue dynamic microtubules. *J. Cell Biol.* 223 (8): e202307074. https://doi.org/10.1083/jcb.202307074

JCB Journal of Cell Biology

DIET-INDUCED FATTY ACID OXIDATION POTENTIATES CARDIAC ECM REMODELING

Context-dependent physiological remodeling of the extracellular matrix (ECM) is essential for development and organ homeostasis. On the other hand, consumption of high-caloric diet leverages ECM remodeling to create pathological conditions that impede the functionality of different organs, including the heart. However, the mechanistic basis of high-caloric diet–induced ECM remodeling has yet to be elucidated.

Employing in vivo molecular genetic analyses in *Drosophila*, we demonstrate that high dietary sugar triggers ROS-independent activation of JNK signaling to promote fatty acid oxidation (FAO) in the pericardial cells (nephrocytes). An elevated level of FAO, in turn, induces histone acetylation–dependent transcriptional up-regulation of the cytokine Unpaired 3 (Upd3). Release of pericardial Upd3 augments fat body-specific expression of the cardiac ECM protein Pericardin, leading to progressive cardiac fibrosis.

Importantly, this pathway is quite distinct from the ROS-Ask1-JNK/p38 axis that regulates Upd3 expression under normal physiological conditions. Our results unravel an unknown physiological role of FAO in cytokine-dependent ECM remodeling, bearing implications in diabetic fibrosis.

ORIGINAL PAPER

Gera, J., D. Kumar, G. Chauhan, A. Choudhary, L. Rani, L. Mandal, and S. Mandal. 2024. High sugar diet–induced fatty acid oxidation potentiates cytokine-dependent cardiac ECM remodeling. *J. Cell Biol.* 223 (9): e202306087. https://doi.org/10.1083/jcb.202306087

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RECOMBINANT BIOSENSORS TO STAIN PHOSPHOINOSITIDES

Phosphoinositides are a small family of phospholipids that act as signaling hubs and key regulators of cellular function. Detecting their subcellular distribution is crucial to gain insights into membrane organization and is commonly done by the overexpression of biosensors. However, this leads to cellular perturbations and is challenging in systems that cannot be transfected.

We present a toolkit for the reliable, fast, multiplex, and super-resolution detection of phosphoinositides in fixed cells and tissue, based on recombinant biosensors with self-labeling SNAP tags. These are highly specific and reliably visualize the subcellular distributions of phosphoinositides across scales, from 2D or 3D cell culture to *Drosophila* tissue. Further, these probes enable super-resolution approaches, and using STED microscopy, we reveal the nanoscale organization of PI(3)P on endosomes and PI(4)P on the Golgi. Finally, multiplex staining reveals an unexpected presence of PI(3,5)P_2-positive membranes in swollen lysosomes following PIKfyve inhibition.

This approach enables the versatile, high-resolution visualization of multiple phosphoinositide species in an unprecedented manner.

ORIGINAL PAPER

Maib, H., P. Adarska, R. Hunton, J.H. Vines, D. Strutt, F. Bottanelli, and D.H. Murray. 2024. Recombinant biosensors for multiplex and super-resolution imaging of phosphoinositides. *J. Cell Biol.* 223 (6): e202310095. https://doi.org/10.1083/jcb.202310095

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V-ATPASE–ATG16L1 RECRUITS LRRK2 TO MAINTAIN LYSOSOME HOMEOSTASIS

Leucine-rich repeat kinase 2 (LRRK2), a Rab kinase associated with Parkinson's disease and several inflammatory diseases, has been shown to localize to stressed lysosomes and get activated to regulate lysosomal homeostasis. However, the mechanisms of LRRK2 recruitment and activation have not been well understood.

We found that the ATG8 conjugation system regulates the recruitment of LRRK2 as well as LC3 onto single membranes of stressed lysosomes/phagosomes. This recruitment did not require the FIP200-containing autophagy initiation complex, nor did it occur on double-membrane autophagosomes, suggesting independence from canonical autophagy. Consistently, LRRK2 recruitment was regulated by the V-ATPase–ATG16L1 axis, which requires the WD40 domain of ATG16L1 and specifically mediates ATG8 lipidation on single membranes. This mechanism was also responsible for the lysosomal stress-induced activation of LRRK2 and the resultant regulation of lysosomal secretion and enlargement.

These results indicate that the V-ATPase–ATG16L1 axis serves a novel non-autophagic role in the maintenance of lysosomal homeostasis by recruiting LRRK2.

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ORIGINAL PAPER
Eguchi, T., M. Sakurai, Y. Wang, C. Saito, G. Yoshii, T. Wileman, N. Mizushima, T. Kuwahara, and T. Iwatsubo. 2024. The V-ATPase–ATG16L1 axis recruits LRRK2 to facilitate the lysosomal stress response. *J. Cell Biol.* 223 (3): e202302067. https://doi.org/10.1083/jcb.202302067

LYSOSOME DAMAGE INDUCES DIRECT ATG8 CONJUGATION

Cells harness multiple pathways to maintain lysosome integrity, a central homeostatic process. Damaged lysosomes can be repaired or targeted for degradation by lysophagy, a selective autophagy process involving ATG8/LC3. We describe a parallel ATG8/LC3 response to lysosome damage, mechanistically distinct from lysophagy.

Using a comprehensive series of biochemical, pharmacological, and genetic approaches, we show that lysosome damage induces non-canonical autophagy and Conjugation of ATG8s to Single Membranes (CASM). Following damage, ATG8s are rapidly and directly conjugated onto lysosome membranes, independently of ATG13/WIPI2, lipidating to PS (and PE), a molecular hallmark of CASM. Lysosome damage drives V-ATPase V0-V1 association, direct recruitment of ATG16L1 via its WD40-domain/K490A, and is sensitive to *Salmonella* SopF. Lysosome damage-induced CASM is associated with formation of dynamic, LC3A-positive tubules, and promotes robust LC3A engagement with ATG2, a lipid transfer protein central to lysosome repair.

Together, our data identify direct ATG8 conjugation as a rapid response to lysosome damage with important links to lipid transfer and dynamics.

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ORIGINAL PAPER
Cross, J., J. Durgan, D.G. McEwan, M. Tayler, K.M. Ryan, and O. Florey. 2023. Lysosome damage triggers direct ATG8 conjugation and ATG2 engagement via non-canonical autophagy. *J. Cell Biol.* 222 (12): e202303078. https://doi.org/10.1083/jcb.202303078

JCB Journal of Cell Biology

N-CADHERIN DYNAMICALLY REGULATES GLIOMA CELL MIGRATION

Pediatric high-grade gliomas are highly invasive and essentially incurable. Glioma cells migrate between neurons and glia, along axon tracts, and through extracellular matrix surrounding blood vessels and underlying the pia. Mechanisms that allow adaptation to such complex environments are poorly understood.

N-cadherin is highly expressed in pediatric gliomas and associated with shorter survival. We found that intercellular homotypic N-cadherin interactions differentially regulate glioma migration according to the microenvironment, stimulating migration on cultured neurons or astrocytes but inhibiting invasion into reconstituted or astrocyte-deposited extracellular matrix. N-cadherin localizes to filamentous connections between migrating leader cells but to epithelial-like junctions between followers. Leader cells have more surface and recycling N-cadherin, increased YAP1/TAZ signaling, and increased proliferation relative to followers. YAP1/TAZ signaling is dynamically regulated as leaders and followers change position, leading to altered N-cadherin levels and organization.

Together, the results suggest that pediatric glioma cells adapt to different microenvironments by regulating N-cadherin dynamics and cell–cell contacts.

ORIGINAL PAPER

Kim, D., J.M. Olson, and J.A. Cooper. 2024. N-cadherin dynamically regulates pediatric glioma cell migration in complex environments. *J. Cell Biol.* 223 (6): e202401057. https://doi.org/10.1083/jcb.202401057

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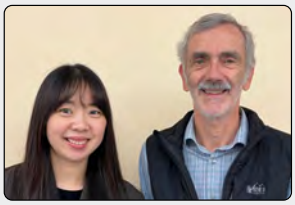

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THE BIOPHYSICAL MECHANISMS OF MYOSIN 2 ASSEMBLY IN CELLS

The ability to dynamically assemble contractile networks is required throughout cell physiology, yet direct biophysical mechanisms regulating non-muscle myosin 2 filament assembly in living cells are lacking.

We use a suite of dynamic, quantitative imaging approaches to identify deterministic factors that drive myosin filament appearance and amplification. We find that actin dynamics regulate myosin assembly, but that the static actin architecture plays a less clear role. Instead, remodeling of actin networks modulates the local myosin monomer levels and facilitates assembly through myosin:myosin-driven interactions. Using optogenetically controlled myosin, we demonstrate that locally concentrating myosin is sufficient to both form filaments and jump-start filament amplification and partitioning. By counting myosin monomers within filaments, we demonstrate a myosin-facilitated assembly process that establishes filament stacks prior to partitioning into clusters that feed higher-order networks.

Together, these findings establish the biophysical mechanisms regulating the assembly of non-muscle contractile structures that are ubiquitous throughout cell biology.

ORIGINAL PAPER

Quintanilla, M.A., H. Patel, H. Wu, K.A. Sochacki, S. Chandrasekar, M. Akamatsu, J.D. Rotty, F. Korobova, J.E. Bear, J.W. Taraska, P.W. Oakes, and J.R. Beach. Local monomer levels and established filaments potentiate non-muscle myosin 2 assembly. *J. Cell Biol.* 223 (4): e202305023. https://doi.org/10.1083/jcb.202305023

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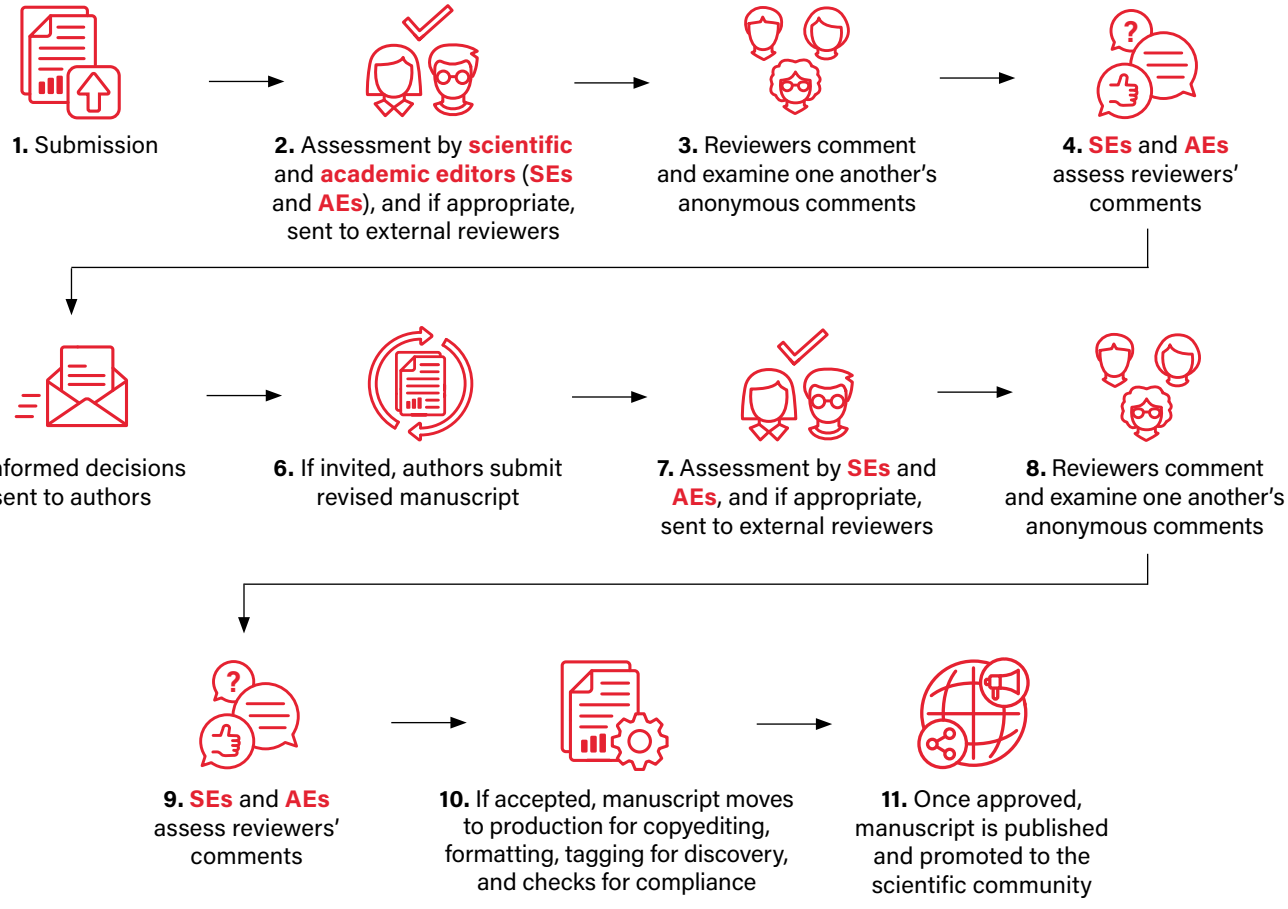

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