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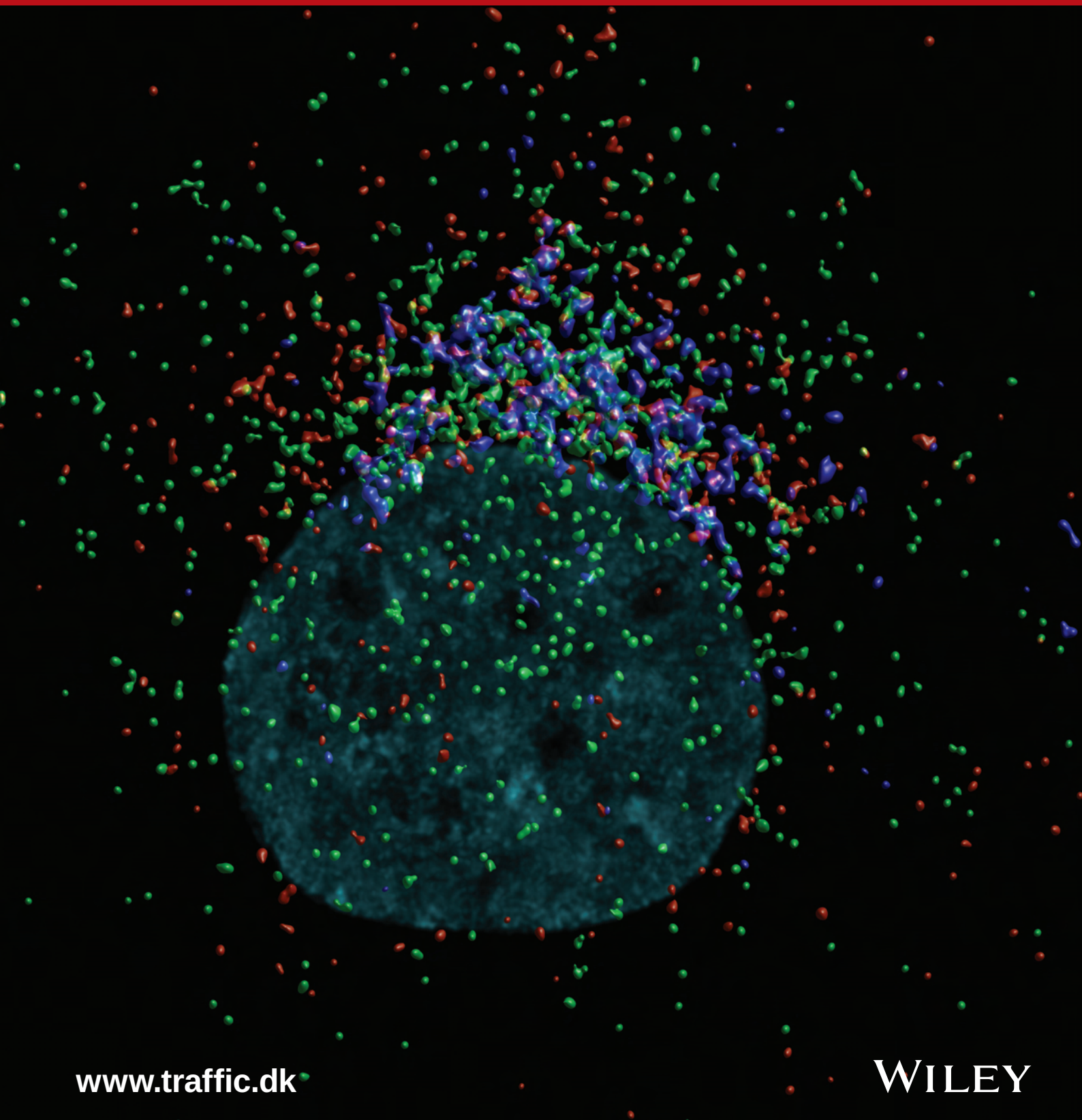

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Cover legend: EGFR (red) colocalize with Transferrin (blue) and Rab11 (green) at perinuclear recycling endosomes. Recycling arrest in HeLa cells treated with propranolol to trigger a signaling pathway that involves phosphatidic acid-mediated down regulation of cAMP levels and PKA activity. Confocal image acquired with a Leica TCS-SP8 microscope at Nyquist oversampling parameters, deconvoluted and three-dimensional reconstructed by surface rendering with Huygens software. See Metz, Traffic 22(10):345–361.

Read the full article on doi:10.1111/tra.12812

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Aim and Scope

Traffic encourages and facilitates the publication of papers in any field relating to intracellular transport in health and disease. Traffic papers span disciplines such as developmental biology, neuroscience, innate and adaptive immunity, epithelial cell biology, intracellular pathogens and host-pathogen interactions, among others using any eukaryotic model system. Areas of particular interest include protein, nucleic acid and lipid traffic, molecular motors, intracellular pathogens, intracellular proteolysis, nuclear import and export, cytokinesis and the cell cycle, the interface between signaling and trafficking or localization, protein translocation, the cell biology of adaptive an innate immunity, organelle biogenesis, metabolism, cell polarity and organization, and organelle movement.

All aspects of the structural, molecular biology, biochemistry, genetics, morphology, intracellular signaling and relationship to hereditary or infectious diseases will be covered. Manuscripts must provide a clear conceptual or mechanistic advance. The editors will reject papers that require major changes, including addition of significant experimental data or other significant revision.

Traffic will consider manuscripts of any length, but encourages authors to limit their papers to 16 typeset pages or less.