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Cellular Dewetting: Opening of Macroapertures in Endothelial Cells¹ DAVID GONZALEZ-RODRIGUEZ, Institut Curie, MADHAVI MAD-DUGODA, CAROLINE STEFANI, Universite de Nice, SEBASTIEN JANEL, FRANK LAFONT, Institut de Biologie de Lille, DAMIEN CUVELIER, Institut Curie, EMMANUEL LEMICHEZ, Universite de Nice, FRANCOISE BROCHARD-WYART, Institut Curie — Pathogenic bacteria can cross from blood vessels to host tissues by opening transendothelial cell macroapertures. Here we model the opening of macroapertures as a new form of dewetting, driven by the cell's membrane tension. While liquid dewetting is irreversible, we show that cellular dewetting is transient. Our model predicts the minimum radius for hole nucleation, the maximum hole size, and the dynamics of opening, in good agreement with the experiments. The physical model is then coupled with biological experimental data to reveal that a certain curvature-sensing protein controls the line tension at the rim of the hole and opposes its opening.

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