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Topological Transitions in Mitochondrial Membranes controlled by Apoptotic Proteins GHEE HWEE LAI, LORI K. SANDERS, ABHIJIT MISHRA, NATHAN W. SCHMIDT, University of Illinois at Urbana-Champaign, GERARD C.L. WONG, University of California, Los Angeles, OLENA IVASHYNA, PAUL H. SCHLESINGER, Washington University School of Medicine — The Bcl-2 family comprises pro-apoptotic proteins, capable of permeabilizing the mitochondrial membrane, and anti-apoptotic members interacting in an antagonistic fashion to regulate programmed cell death (apoptosis). They offer potential therapeutic targets to re-engage cellular suicide in tumor cells but the extensive network of implicated protein-protein interactions has impeded full understanding of the decision pathway. We show, using synchrotron x-ray diffraction, that pro-apoptotic proteins interact with mitochondrial-like model membranes to generate saddle-splay (negative Gaussian) curvature topologically required for pore formation, while anti-apoptotic proteins can deactivate curvature generation by molecules drastically different from Bcl-2 family members and offer evidence for membrane-curvature mediated interactions general enough to affect very disparate systems.

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