Plasminogen Activation–Induced Pericellular Fibronectin Proteolysis Promotes Fibroblast Apoptosis

Jeffrey C. Horowitz, David S. Rogers, Richard H. Simon, Thomas H. Sisson, and Victor J. Thannickal

Division of Pulmonary and Critical Care Medicine, Department of Medicine, University of Michigan Medical Center, Ann Arbor, Michigan

Online Data Supplement

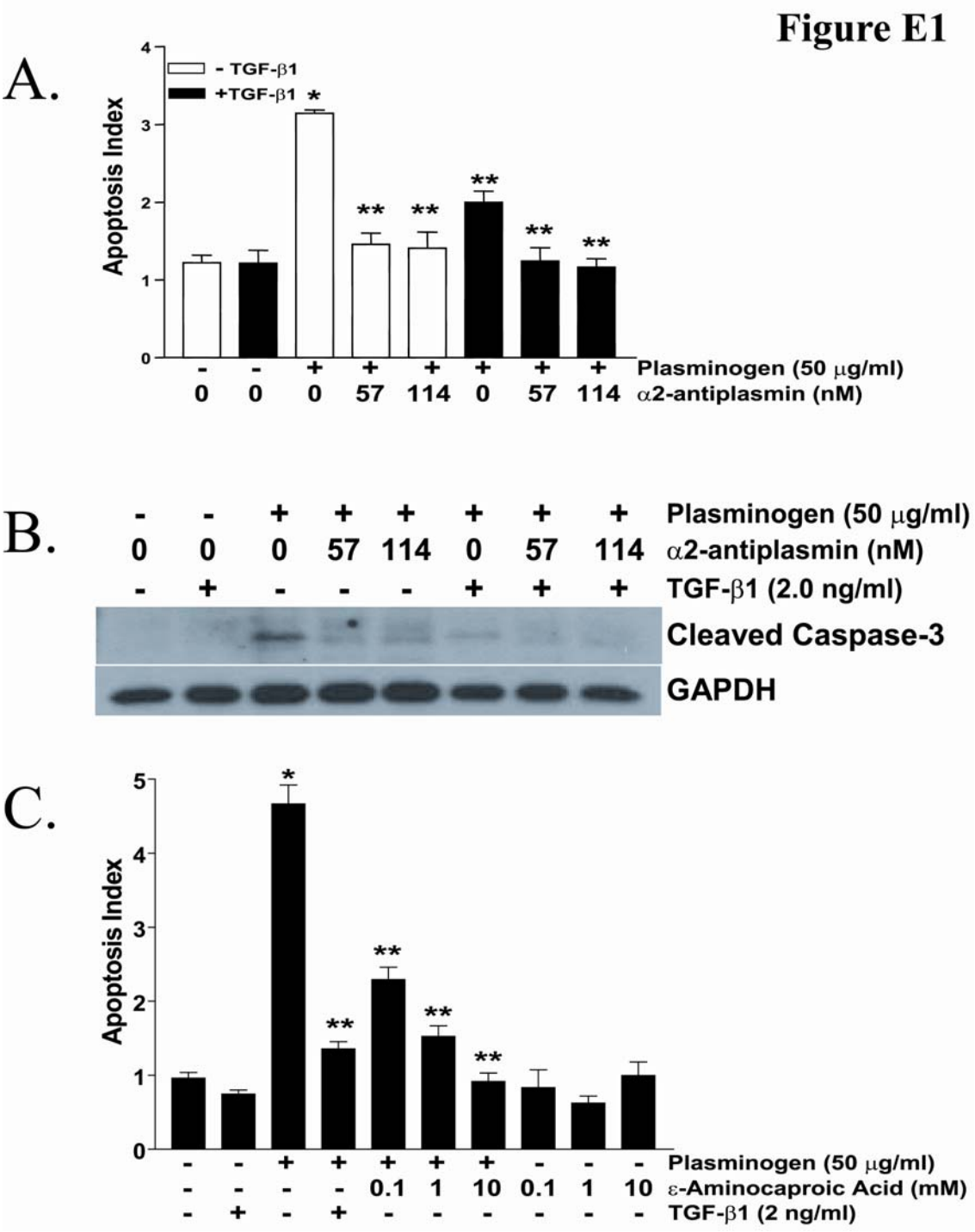

Figure E1: Inhibition of plasminogen activation or plasmin activity blocks plasminogen-mediated fibroblast apoptosis. ***(A and B)*** IMR-90 fibroblasts were treated with/without plasminogen (50 μg/ml) ± TGF-β1 (2 ng/ml) and/or the indicated concentrations of a2-antiplasmin for 18 hours. Apoptosis was assessed by *(A)* ELISA for ssDNA (n = 4; * $p < 0.001$ vs. control, ** $p < 0.001$ vs. plasminogen alone) and ***(B)*** Western immunoblotting for cleaved caspase-3. **(C)** IMR-90 fibroblasts were treated with/without plasminogen (50 μg/ml) in the presence/absence of the indicated doses of the lysine analog, ε-aminocaproic acid or TGF-β1 (2 ng/ml) for 18 hours, and apoptosis was assessed by ELISA for ssDNA. n = 4, * $p < 0.001$ vs. control, ** $p < 0.001$ vs. plasminogen treatment alone.