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Death receptor mediates apoptosis activation by extracellular matrix molecule

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A 26 kDa fragment from α1 chain of type IV collagen, arresten is generated during proteolytic degradation of vascular basement membrane (VBM). Arresten functions as an anti-angiogenic molecule by inhibiting endothelial cell proliferation, migration, and tube formation. We previously reported antiangiogenic activity of arresten partly mediating by induction of apoptosis in endothelial cells. However, active site of arresten responsible for its pro-apoptotic activities is not known yet. In the present study, both the N- and C-terminal subunits of arresten were cloned in the pET22b (+) expression vector and expressed in E. Coli. Expressed both arresten subunits were purified using different affinity and size exclusion chromatographies with simultaneous renaturation and studied their antiangiogenic and pro-apoptotic properties. Our preliminary in-vitro results with endothelial cells have shown inhibition of VEGF and bFGF induced proliferation, migration and tube formation by both the subunits. In addition, both N- and C-terminal subunits of arresten promoted FasL mediated apoptosis by releasing cytochrome-c from mitochondria and activating caspase-3 and PARP both in-vitro and in-vivo.

Biography

Dr. Raj Kumar Verma completed his Ph.D in the field of Medical Microbiology from CSM Medical University (formerly known as King George's Medical University), an accredited institution of higher education in India. Dr. Verma is currently working as a postdoctoral research fellow in Retinal and Tumor Angiogenesis Laboratory at Boys Town National Research Hospital, Omaha, NE USA. He also did postdoctoral training from University of Florida, Albany Medical Center and Creighton University and worked on periodontal disease, cell biology and cancer Research and asthma. He has published more than 10 papers in reputed journals and serving as a reviewer in several esteemed journals