

CCR5 /CCL5 axis interaction promotes migratory and invasiveness of pancreatic cancer cells

Santosh Kumar Singh¹, Manoj K. Mishra², Isam-Eldin A. Eltoun³, Sejong Bae⁴, James W. Lillard Jr.¹, and Rajesh Singh^{1,*}

¹Morehouse School of Medicine, Department of Microbiology, Biochemistry and Immunology, Atlanta, GA, 30310, USA

²Cancer Biology Research and Training Program, Department of Biological Sciences, Alabama State University, Montgomery, AL, 36101, USA

³Department of Pathology, University of Alabama at Birmingham, Birmingham, AL, 35294, USA

⁴Department of Medicine, University of Alabama at Birmingham, Birmingham, AL, 35294, USA

Send correspondence to: *Rajesh Singh, Morehouse School of Medicine, Department of Microbiology, Biochemistry and Immunology, 720 Westview Drive SW, Atlanta GA 30310, Tel: 404-756-6661, Fax: 404-752-1179, E-mail: rsingh@msm.edu

Supplementary Figure 2B

Supplementary Figure 2B: Expression of CCR5 by pancreatic cancer cells (A) Western blot expression of CCR5 by pancreatic cancer cells (AsPC-1, BxPC-3 and MIA PaCa-2) was resolved by the SDS-PAGE gel. (B) Expression of β -actin was detected by western blot analysis with anti- β -actin antibodies. β -actin was used as a loading control for all the three samples.

CCR5 Expression

(A)



(B) β -Actin Expression

