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Toll-Like receptor 7 regulates pancreatic carcinogenesis

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We found that TLR7 expression is upregulated in both epithelial and stromal compartments in human and murine pancreatic cancer. We postulated that carcinogenesis is dependent on TLR7 signaling. TLR7 ligation vigorously accelerated tumor progression and induced altered expression of PTEN, p21, p27, p53, p16, c-myc, SHPTP1, STAT3, TGF-b, and PPAR-g in Kras-transformed pancreata. Conversely, blockade of TLR7 completely protected against carcinogenesis. Since pancreatic tumorigenesis requires stromal expansion, we proposed that TLR7 ligation modulates pancreatic cancer by driving stromal inflammation. Accordingly, we found that TLR7-/- bone-marrow-chimeric p48Cre; KrasG12D mice were protected from neoplasia. Further, to affect peritumoral inflammation, TLR7 signaling interfaced with Notch as well as classic NF-kB and MAP kinase pathways but down regulated expression of Notch target genes. These data suggest that targeting TLR7 holds promise for treatment of human pancreatic cancer.

Biography

Adeel Ur Rehman has completed his M.D at the age of 24 years from Shifa College of Medicine, Pakistan and postdoctoral research fellowship from New York University School of Medicine. He is currently a general surgery resident at University of Texas Health Science Center at San Antonio.

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