Mohammed Yousif Merza, J Clin Cell Immunol 2015, 6:5
http://dx.doi.org/10.4172/2155-9899.C1.024

OMICS International
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4th International Conference and Exhibition on
Immunology
September 28-30, 2015 Crowne Plaza Houston River Oaks, Houston, TX, USA

# Role of CXCL4 in neutrophil accumulation and tissue injury in severe acute pancreatitis

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Leukocyte infiltration and acinar cell necrosis are hallmarks of severe acute pancreatitis (AP) but the signaling pathways regulating inflammation and organ injury in the pancreas remain elusive. In the present study, we investigated the role of CXCL4 in AP. Male C57BL/6 mice were treated with an anti CXCL4 antibody (20 μ/kg) prior to induction of pancreatitis by infusion of taurocholate into the pancreatic duct. Pretreatment with anti CXCL4 Ab reduced blood amylase levels, pancreatic neutrophil recruitment and hemorrhage and edema formation in taurocholate-evoked pancreatitis. Moreover, administration of anti CXCL4 Ab decreased the taurocholate-induced increase of myeloperoxidase activity in the pancreas and lung. Treatment with anti CXCL4 Ab markedly reduced levels of CXCL2 in the pancreas and IL-6 in the plasma in response to taurocholate challenge. Notably, inhibition of CXCL4 had no direct effect on secretagogue-induced activation of trypsinogen in pancreatic acinar cells *in vitro*. A significant role of CXCL4 was confirmed in an alternate model of AP induced by L-arginine challenge. Our findings show that CXCL4 regulates neutrophil accumulation and tissue damage via CXCL2 formation in AP. Thus, these results reveal new signaling mechanisms in pancreatitis and indicate that targeting CXCL4 might be an effective way to ameliorate severe AP.

## Biography

Mohammed Yousif Merza is a PhD student and permanent staff at Hawler Medical University, Kurdistan. He has graduated from Salahadin University, Hawler in 2003 and completed a Master's degree at Glasgow University, UK in 2008. He has published 5 papers in reputed journals.

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