Public Abstract First Name:Jing Middle Name: Last Name:Chen Adviser's First Name:Bimal Adviser's Last Name:Ray Co-Adviser's First Name: Co-Adviser's Last Name: Graduation Term:FS 2006 Department:Veterinary Pathobiology Degree:MS Title:MECHANISM OF INDUCTION OF VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) IN OSTEOARTHRITIS

Osetoarhtritis (OA) is an autoimmune disease defined as bone damage in joints. Angiogenesis (new blood vessel formation) is involved in the pathogenesis of OA. A factor called VEGF promotes new blood vessel formation. Specific mechanism controlling induction of VEGF in OA is not clear. In the pathogenic condition of OA, a factor called SAF-1 is abundantly present in the diseased joint tissues. This study uses several molecular techniques to determine if SAF-1 regulates VEGF expression and the mechanisms by which inflammatory cytokines mediate joint damage. The results showed that SAF-1 regulate VEGF expression in a dose dependent manner. SAF-1 plays an important role in VEGF expression induced by inflammatory cytokines. Dysfunction of SAF-1 completely abolishes the expression of VEGF induced by inflammatory cytokines. The function of SAF-1 is also regulated by an other factor. Together these results indicate that the abundance of SAF-1 induced by inflammatory cytokines promote VEGF expression, leading to the damage of joint tissues. Therefore, SAF-1 could be a potential therapeutic target to design novel drug for the treatment of OA patients.