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Autodesk 3ds Max 2019 with Softimage, 978K1.. Autodesk 3ds Max 2019 with Softimage, 978K1. autodesk 3ds max 2019, x-force 2019, autocad 2019 with softimage, autodesk 2020, 2020 autocad with softimageSelective inhibition of inducible nitric oxide synthase prevents tacrolimus-induced cholestasis in a rat model of liver transplantation. The immunosuppressant tacrolimus (FK506) causes a number of side effects in liver transplant recipients that can result in graft loss. Recent data indicate that nitric oxide (NO) is involved in the pathogenesis of FK506-induced cholestasis, a common complication of liver transplantation. Since inducible nitric oxide synthase (iNOS) is stimulated in the liver in response to FK506, the authors used a novel inhibitor of iNOS, S-methylisothiourea sulfate (SMT), to determine if selective inhibition of iNOS could protect the liver against FK506-induced cholestasis and to assess the mechanism of cholestasis. Male SD rats received a combined liver-kidney transplant. FK506 was administered either systemically alone or in combination with SMT. Liver function was assessed by measuring aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, and bile salt excretion in urine. Protein expression of nitric oxide synthase (NOS) and hepatic cytokine production were examined in liver biopsy specimens. Liver function was impaired by FK506, evidenced by increased AST, ALT, total bilirubin, and bile salt excretion. SMT pretreatment prevented FK506-induced cholestasis. Inhibition of iNOS by SMT significantly suppressed hepatic iNOS expression, and production of macrophage migration inhibitory factor (MIF) and tumor necrosis factor alpha (TNFalpha). Inhibition of iNOS by SMT attenuated liver injury caused by FK506 and resulted in normalization of bile formation and bile acid excretion in the FK506-treated rats. Selective inhibition of iNOS is a novel approach for the prevention and treatment of FK506-induced cholestasis. iNOS inhibition supp 595f342e71