

Inhibition of mTOR restores corticosteroid sensitivity in COPD

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restored corticosteroid sensitivity. CSE stimulated mTOR activity and c-Jun expression in U937 cells; rapamycin pretreatment inhibited both and reversed corticosteroid insensitivity induced by CSE.

"mTOR inhibition by rapamycin restores corticosteroid sensitivity via inhibition of c-Jun expression, and thus mTOR is a potential novel therapeutic target for COPD," the authors write.

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(HealthDay)—Inhibition of mammalian target of rapamycin (mTOR) by rapamycin restores corticosteroid sensitivity in patients with chronic obstructive pulmonary disease (COPD), according to a study published in the Jan. 15 issue of the *American Journal of Respiratory and Critical Care Medicine*.

Akihisa Mitani, M.D., Ph.D., from Imperial College London, and colleagues examined the role of mTOR in corticosteroid sensitivity in COPD. Corticosteroid sensitivity was assessed in peripheral blood mononuclear cells obtained from patients with COPD, smokers, and nonsmoking controls, as well as in human monocytic U937 cells exposed to cigarette smoke extract (CSE), in the presence and absence of the mTOR inhibitor rapamycin.

The researchers observed increased mTOR activity in peripheral blood mononuclear cells from patients with COPD, which was inhibited by rapamycin treatment. Rapamycin treatment also

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