

ERS: Mepolizumab is glucocorticoid-sparing in asthma

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(HealthDay)—For patients with eosinophilic asthma, mepolizumab has a glucocorticoid-sparing effect and reduces exacerbations when administered intravenously or subcutaneously, according to two studies published online Sept. 8 in the *New England Journal of Medicine* to coincide with the European Respiratory Society's International Congress, held Sept. 6 to 10 in Munich.

Elisabeth H. Bel, M.D., Ph.D., from the University of Amsterdam in the Netherlands, and colleagues compared the glucocorticoid-sparing effect of mepolizumab with that of <u>placebo</u> in 135 <u>patients</u> with severe eosinophilic asthma. The researchers found that the likelihood of a reduction in the glucocorticoid-dose stratum was significantly greater in the mepolizumab group than in the <u>placebo group</u> (odds ratio, 2.39). In the mepolizumab group, the median percentage reduction from baseline in the glucocorticoid dose was 50 percent, compared with no reduction



in the placebo group.

Hector G. Ortega, M.D., Sc.D., from GlaxoSmithKline in Research Triangle Park, N.C., and colleagues conducted a double-blind, double-dummy study involving 576 patients with recurrent asthma exacerbations and evidence of eosinophilic inflammation in spite of receiving high doses of inhaled glucocorticoids. Patients were randomized to mepolizumab as a 75-mg intravenous dose or a 100-mg subcutaneous dose or to placebo. The researchers found that compared with patients receiving placebo, those receiving intravenous mepolizumab and subcutaneous mepolizumab had reductions in the rate of exacerbations (47 and 53 percent, respectively).

"Anti-interleukin-5 therapy offers an important advance in our ability to care for patients with severe eosinophilic asthma," writes the author of an accompanying editorial.

Both studies were funded by GlaxoSmithKline. Authors of both studies and the editorial disclosed financial ties to GlaxoSmithKline and other pharmaceutical companies.

More information: <u>Abstract—Bel</u>

Full Text
Abstract—Ortega
Full Text
Editorial

More Information

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