in patients with allergic asthma who are steroid naive with the Th2-hi phenotype. However, development of this drug for use in asthma has been discontinued.

Short-Term Treatment With Rosuvastatin Reduces Systemic Inflammation in Stable COPD Patients

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The use of short-term rosuvastatin to treat patients with stable chronic obstructive pulmonary disease (COPD) reduces systemic inflammation but has no effect on pulmonary function and improves endothelial function only in patients with baseline C-reactive protein (CRP) levels >1.7 mg/L.

Anke Neukamm, MD, Cardiothoracic Research Group, Akershus University Hospital, Lørenskog, Norway, presented results of the Effect of Rosuvastatin Treatment in Stable COPD study [RODEO; NCT00929734; Neukamm A et al. *Am J Respir Crit Care Med* 2014], a randomized, double-blind clinical trial conducted to test the hypothesis that the use of statin therapy in patients with stable COPD would be associated with improved endothelial and pulmonary function and reduced systemic inflammation.

The study included 99 patients with stable COPD who were randomly assigned to once-daily treatment with rosuvastatin 10 mg (n=49) or placebo (n=50) for 12 weeks. Patients were excluded from the study if they had histories of or active coronary artery diseases, other lung diseases except asthma bronchiale, uncontrolled arterial hypertension, histories of diabetes or hypercholesterolemia, or statin use within the 4 weeks prior to the onset of the study.

Of the 99 patients, 94 patients completed the study (47 in each treatment arm). Baseline characteristics were similar between the 2 groups for age (mean, 65 years), sex (48% women), history of hypertension (25%), and smoking habit (mean, 37 pack-years).

To evaluate the effect of rosuvastatin on endothelial and pulmonary function and systemic inflammation, the primary endpoint was the change in peripheral vasodilator function measured by peripheral arterial tonometry and expressed as the reactive hyperemia ratio (RHI). The study also included a prespecified subgroup analysis of the effect of rosuvastatin in patients with elevated CRP levels.

Secondary endpoints included change in change in pulmonary function as assessed by forced expiratory volume in 1 second (FEV_1) and the ratio of FEV_1 to forced volume capacity ratio (FVC), as well as change in the circulating proinflammatory markers CRP, interleukin-6 (IL 6), and high-sensitivity CRP (hsCRP).

In an intention-to-treat analysis, the study found no difference between rosuvastatin and placebo in the primary end point of the study, vasodilator function.

However, there was a significant difference in vasodilator function, as determined by the change in RHI, with rosuvastatin compared with placebo in the subgroup of patients with elevated baseline CRP levels (median >1.7 mg/L, p=0.026).

In terms of secondary endpoints, rosuvastatin was associated with a significant reduction in hsCRP and attenuated the rise in IL6 compared with placebo (Table 1). No significant differences were seen in pulmonary function in FEV_1 or FEV_1/FVC.

Based on these results, the investigators emphasize the need for long-term randomized trials with large cohorts to more accurately assess the benefits of statin therapy in patients with stable COPD.

Table 1. Results of Secondary Endpoints

	Rosuvastatin (n=47)		Placebo (n=47)		
Variable	**Baseline**	**Follow-Up**	**Baseline**	**Follow-Up**	**p[a]**
FEV_1, L[b]	1.34 ± 0.09	1.35 ± 0.08	1.47 ± 0.10	1.45 ± 0.94	.462
FEV_1/FVC, %[b]	46.9 ± 2.01	47.3 ± 1.72	48.8 ± 1.72	47.9 ± 1.79	.292
hsCRP, mg/L[c]	1.4 (0.8, 3.7)	1.2 (0.7, 2.6)	1.8 (1.0, 4.4)	2.2 (1.3, 3.6)[d]	.017
IL-6, pg/mL[c]	4.1 (2.9, 5.2)	4.1 (3.4, 5.5)	3.4 (2.7, 4.7)	4.4 (3.0, 6.7)[e]	.028

FEV_1=forced expiratory volume in 1 second; FVC=forced vital capacity; hsCRP=high-sensitivity C-reactive protein; IL-6=interleukin-6.
[a]Analysis of covariance.
[b]Mean ± SEM.
[c]Median (25th percentile, 75th percentile).
[d]p<0.05, paired *t* test.
[e]p<0.05, Student *t* test.