

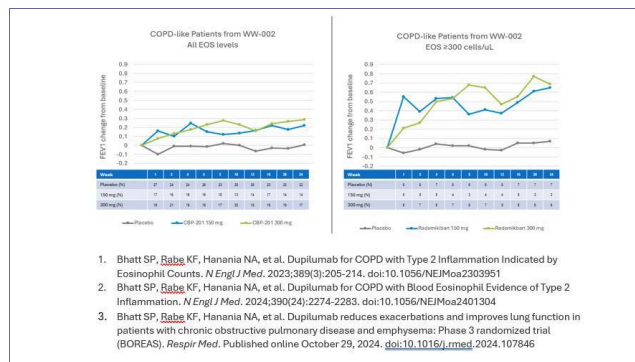
[Print this Page for Your Records](#)[Close Window](#)**Control/Tracking Number:** 2025-S-13157-ATS**Activity:** Scientific Abstract**Current Date/Time:** 2/25/2025 2:17:52 PM**Efficacy Of Rademikibart In COPD-like Patients: Sub-analyses From The Phase 2b Trial In Patients With Moderate-to-Severe Asthma****Author Block:** R. Collazo, J. Wang, B. Quart;
Connect Biopharma, San Diego, CA, United States.**Abstract:**

RATIONALE: More than 26 million Americans are burdened with asthma symptoms and another 15.7 million have been diagnosed with chronic obstructive pulmonary disease (COPD). Rademikibart is an IL-4R α -blocker that blocks both interleukin-4 and interleukin-13 signaling. In a previously reported Phase 2b asthma trial, rademikibart demonstrated significant improvements in lung function by Week 1 that were sustained through 24-weeks of treatment. Recent evidence suggests that rademikibart, as an IL-4R α -blocker, may also work well in eosinophilic driven COPD¹⁻³. We assessed rademikibart's efficacy in a subset of COPD-like patients from the Phase 2b trial in patients with uncontrolled moderate-to-severe asthma.

METHODS: The parent Phase 2b trial (NCT04773678) was a global, placebo-controlled study with 322 patients randomized 1:1:1 to rademikibart 150 mg every two weeks (Q2W; n=106), 300 mg Q2W (n=108) each with a 600 mg loading dose or placebo (n=108). COPD-like patients were defined as having asthma onset age > 40 year and post-bronchodilator FEV₁/forced vital capacity < 0.7 at screening visit (n=18, n=23 and n=27, respectively). Additionally, COPD-like patients with Baseline eosinophils \geq 300 cells/ μ L were identified for analysis (n=6, n=8 and n=8, respectively). The *post hoc* end point of interest was absolute change from baseline (CFB) in pre-bronchodilator FEV₁ at Week 12. Additional analyses included pre-BD FEV₁ in COPD-like patients over time (Week 1-Week 24) and in COPD-like patients with a blood eosinophil count of \geq 300 cells/ μ L.

RESULTS: Pre-BD FEV₁ in COPD-like patients at Week 12 was improved over baseline with both the rademikibart 150 mg (Mean CFB 160 mL [95% CI -40-370]) and 300 mg (160 mL [0-320]) doses. Further CFB improvement was achieved in patients with eosinophil levels of \geq 300 cells/ μ L (370 mL [-40-1130] and 470 mL [40-910]). Improvements started in Week 1 (both doses) and were sustained through 24 weeks of treatment in both the overall COPD-like population (Week1 CFB 160 mL [-30-340 mL] and 80 mL [-50-200]; Week 24: 220 mL [30-420 mL] and 260 mL [90-420 mL], respectively) and in those with eosinophils \geq 300 cells/ μ L (Week 1: 550 mL [110-990 mL] and 210 mL [80-350 mL]; Week 24: 650 mL [40-127 mL] and 690 mL [-80-147 mL], respectively).

CONCLUSION: Rademikibart has previously shown efficacy in moderate-to-severe asthma. Though small numbers, this *post hoc* analysis of COPD-like patients from the rademikibart Phase 2b trial in asthma, combined with recent IL-4/COPD data, supports the further examination of rademikibart in the eosinophilic driven COPD patient population.



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