

# Treatments explored for moderate-to-severe alopecia areata

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(HealthDay)—For patients with alopecia areata (AA), treatment with an

inhibitor of janus kinase (JAK)3 and the tyrosine kinase expressed in the hepatocellular carcinoma kinase family, ritlecitinib, and an inhibitor of tyrosine kinase 2/JAK1, brepocitinib, is associated with improvements in the lesional scalp transcriptome, according to a study published online Dec. 1 in the *Journal of Allergy and Clinical Immunology*.

Emma Guttman-Yassky, M.D., Ph.D., from the Icahn School of Medicine at Mount Sinai in New York City, and colleagues conducted a biopsy substudy during the first 24 weeks of a phase 2a, randomized, placebo-controlled clinical trial examining the efficacy and safety of ritlecitinib and brepocitinib in the treatment of AA. Forty-six patients from the ritlecitinib, brepocitinib, and placebo groups (18, 16, and 12, respectively) were included. The change in biomarkers in lesional scalp biopsies between baseline and weeks 12 and 24 was examined as an exploratory end point. Correlations of biomarkers with hair regrowth were evaluated.

The researchers observed improvement with both ritlecitinib and brepocitinib exceeding 100 percent in the lesional scalp transcriptome toward a nonlesional profile at week 24. The improvements in scalp tissue were greater with brepocitinib than ritlecitinib at week 24; however, greater improvements were seen with ritlecitinib at week 24. There was a [positive association](#) seen for improvement in the Severity of Alopecia Tool scores with expression of Th1 markers and a negative association with expression of hair keratins for both ritlecitinib and brepocitinib.

"Since changes in molecular biomarkers occurred between weeks 12 and 24, it would be of interest to assess whether additional changes occur after 24 weeks," the authors write.

Several authors disclosed financial ties to [pharmaceutical companies](#), including Pfizer, which manufactures ritlecitinib and brepocitinib and

funded the study.

**More information:** [Abstract/Full Text](#)

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