

Reply to comment on: “Pattern hair loss: Assessment of microinflammation in miniaturized and terminal hair follicles through horizontal histologic sections”



To the Editor: We thank Merlotto et al for sharing their experience and for providing further evidence to elucidate the role of microinflammation in pattern hair loss. Three main observations are worth highlighting: (1) inflammatory infiltrate was more pronounced in miniaturized follicles, (2) microinflammation was positively correlated with follicle apoptosis index, and (3) infiltrate surrounding miniaturized hair follicles was predominantly CD4⁺.

Most inflammatory infiltrates show CD4 predominance over CD8 in a 2:1 to 3:1 ratio,¹ and one may speculate on the role that cytokines such as interleukin (IL) 16 may play in the pathogenesis of pattern hair loss. Mature IL-16 is secreted by cells as a ligand for CD4. Originally, this was called *lymphocyte chemo-attractant factor*; however, currently, it is known that eosinophils, mast cells, monocytes, and B lymphocytes are responsive to IL-16,² thereby orchestrating a complex immune response. In addition, it has been suggested that IL-16 could regulate caspase-3, leading to apoptosis³; both series of molecular events could correlate with Merlotto et al's observations. Clarification is needed regarding the CD4:CD8 ratio, how many inflammatory cells expressed CD9, the presence of inflammatory cells expressing CD20, and

differences in single nucleotide polymorphisms in the IL-16 gene locus between affected patients and control individuals.

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