

Evaluation of the Expression of Genes Associated with Inflammation and Apoptosis in Androgenetic Alopecia by Targeted RNA-Seq

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Keywords

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Abstract

Androgenetic alopecia (AGA) or male pattern baldness is the most common form of hair loss in humans. Despite being a very frequent dermatological entity, molecular pathophysiology remains unclear. Several authors relate the presentation of AGA with a premature apoptotic process during the anagen phase and with an inflammatory microenvironment in the hair follicle. We evaluated a panel of 30 genes associated with inflammation and apoptosis in 5 AGA patients by targeted RNA-Seq. *WNT7A* gene was highly expressed in patients in stages 3V to 5 on the Hamilton-Norwood scale compared to patients with 5A stage. *CASP7* and *TNF* genes were overexpressed in stages 3V and 4 compared to stages 5 and 5A. Overexpression of these genes detected only at early stages of AGA proves the role of WNT pathway, apoptosis, and inflammation in the development of this disorder.

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Introduction

Androgenetic alopecia (AGA), or male baldness pattern androgenetic alopecia, is the most common form of hair loss in humans and affects 80% of Caucasian men and 40–50% of Caucasian women [1]. In AGA, there is an alteration of the hair growth cycle [2]. AGA is characterized by miniaturization of the hair follicle, a phenomenon that occurs due to a rapid change from the anagen phase to the catagen/telogen phase, which probably reflects an ongoing follicle apoptotic process [3]. During the hair growth cycle, there is an important interaction between growth factors such as cytokines, hormones, and neurotransmitters and their receptors [4]. However, it is still not clear if AGA results from disruption of proliferation or an increase in apoptosis in the hair follicle [5]. Despite this, one of the main targets of topical and systemic antioxidant therapies for AGA is apoptosis inhibition [6]. In fact, studies of drugs such as finasteride have shown decreased levels of caspases after 6 months of treatment in patients with AGA compared with healthy subjects [7]. Most studies evaluating caspases in patients with AGA