In pattern hair loss (PHL) the genetically predisposed hair follicles are susceptible to androgen-stimulated hair follicle miniaturization, leading to replacement of large and pigmented hairs by barely visible, depigmented hairs. The result is a progressive decline in visible scalp hair density that follows a defined, age- and sex-dependent pattern. Major advances have been achieved in understanding peculiarities of the androgen metabolism involved. Nevertheless, clinical practice has shown that simply blocking androgens has limited success. On histologic examination, the miniaturization of terminal hairs is frequently associated with perifollicular inflammatory phenomena and fibrosis. Therefore, sustained microscopic follicular inflammation with connective tissue remodeling, eventually resulting in permanent hair loss, is considered a possible cofactor in the etiology of PHL. There seems to exist a continuum of inflammatory phenomena and fibrosis in PHL reaching from microscopic follicular inflammation and fibrosis (Jaworsky et al, 1992) to localized phenomena in frontal fibrosing alopecia (Kossard, 1994) and a more generalized pattern in fibrosing alopecia in a pattern distribution (Zinkernagel and Trüeb, 2000). So far, the inflammatory component has not been included in treatment protocols for PHL. Besides androgens and genetic imbalance, additional pathogenic factors are now suspected, such as microbial flora, endogenous and exogenous stress, microinflammation, and possibly others. Dissecting the molecular controls of immune-mediated, physiological hair follicle degeneration by apoptosis-mediated organ deletion could provide insights into how progression of some forms of permanent alopecia might be halted, which can be suppressed with only limited success by current treatment modalities. This could also hold true for further studies in PHL with microscopic or clinical evidence of inflammatory phenomena and fibrosis.

Further Reading