

Patterned cycle of a relatively long-lasting anagen



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Patterned hair loss is thought to be caused by both inherited and physiological backgrounds. Patterned hair loss gradually proceeds following a specific pattern in front and on top of the scalp. Among males, hair shedding often begins after puberty due to the effect of the male hormone, androgen, and is named male pattern baldness, also known as androgenetic alopecia (AGA). The major cause of AGA in human is thought to be as follows; the human hair follicle follows a cycle of a relatively long-lasting anagen phase and relatively short catagen and telogen phases. Each hair follicle is replaced with a revitalized newly generated hair, but when AGA occurs, the anagen phase becomes shorter and the hair follicle does not fully grow and enter the next hair cycle, which results in increasing amounts of short and thin hairs (miniaturized hairs), and eventually the temporal or forehead scalp surface skin becomes visible (Sakamoto et al., 2017). Androgens are known to cause cell regression and balding of the scalp in AGA individuals.

Testosterone and dihydrotestosterone (DHT), which is formed by action of 5 α -reductase (5 α R), are two major androgens and DHT are considered more potent to trigger hair loss. Androgens also affect the dermal papilla (DP) of hair follicle, which produces paracrine signals that stimulate or inhibit the growth of follicular epithelium. These include insulin-like growth factor-1 (IGF-1) and vascular endothelial growth factor (VEGF).

Other growth factors are also found to be involved in the hair growth regulation such as keratinocyte growth factor (KGF) and hepatocyte growth factor (HGF) have a stimulatory effect on hair follicle growth, while epidermal growth factor (EGF) and transforming growth factor- β (TGF- β) have an inhibitory effect on hair follicle growth (Roh et al., 2002). 1.

1 TREATMENT OF HAIR LOSS 1.1.1 Approved drugs for hair loss

At present there are few drugs used to treat androgenic alopecia (AGA). The first drug approved for enhancing scalp hair growth was minoxidil (Rogaine®). It causes vasodilation (widening of blood vessels), thus increasing circulation.

In about a third of the people who try it, minoxidil improves hair growth, causing scalp follicles to enlarge and lengthening the growth cycle. For many, however, the hair growth is meager. Minoxidil does not help people who already are bald (Tortora & Derrickson, 2009). Minoxidil was first used as a vasodilator to treat cardiovascular disorders, but the unexpected side effect of hirsutism led to its topical use as a hair-growth stimulator. The mechanisms involved in AGA treatment are still unclear. It seems to open potassium channels and increase the proliferation and differentiation of epithelial cells in the hair shaft. However, local irritation, itching, dryness and erythema may occur when minoxidil is topically used, as well as systemic side effects such as dizziness and tachycardia.

Serious side effects, such as an increase in left ventricular end-diastolic volume, cardiac output, and left ventricular mass, have been reported with the use of 2% minoxidil solution. Unfortunately, another potential drawback of minoxidil therapy is the loss of newly grown hair within one to three months after discontinuation of the medicine (Kumar, Rungseevijitprapa, Narkkhong, Suttajit, & Chaiyasut, 2012). A low strength of finasteride is also licensed in treatment of AGA. It is a specific 5 α -reductase inhibitor which metabolizes testosterone into a more potent androgen, dihydrotestosterone (DHT). The active metabolite of dihydrotestosterone

(DHT) will binds to androgenicreceptors in the hair follicle and then activates the genes responsible forhair follicle regression (Herman & Herman, 2016) . But thesedrugs may possess certain side effects include impotence, decreased libido, ejaculation disorders and breast tenderness and enlargement (Committee, 2013).