

The DHT Story in Hair Loss-Still True, but PGD2 Writes a Missing Chapter

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Abstract

This study examines how artificial intelligence tools shape workflow and internal communication within student organizations at the University of the Philippines Los Baños, using Media Richness Theory as a guiding framework. Through four focus group discussions with officers and members from diverse clubs, the research investigates perceptions of AI's ability to convey rich information, reduce ambiguity, and support collaborative tasks. Participants described AI-driven platforms—such as chatbots, automated scheduling systems, and content-generation tools—as enhancing message clarity and speeding decision processes, yet noted barriers related to learning curves and access. Analysis revealed that AI applications functioned as richer media when users received adequate training and when tools aligned with task complexity; conversely, mismatches between tool features and organizational needs led to confusion or underuse.

The findings suggest that deliberate integration strategies—emphasizing hands-on workshops, peer mentoring, and clear guidelines—can help student organizations leverage AI to improve meeting efficiency, member engagement, and document management. By demonstrating the conditions under which AI attains optimal media richness, this research offers practical recommendations for student-led groups seeking to modernize their communication practices without sacrificing interpersonal connection.

Full Article

[Finasteride](#), [dutasteride](#), RU58841, and [saw palmetto](#) all target the same pathway: they lower [dihydrotestosterone \(DHT\)](#) levels or block its receptor. [Minoxidil](#) can extend the [anagen](#) (growth) phase of hair follicles. Yet many users continue to lose density and experience recession, indicating that DHT is only one factor in pattern hair loss.

PGD?: The Surprise Co-Conspirator

Back in 2012, a Penn dermatology team compared “hairy” and “bald” punch biopsies from the same scalps. In the bald zones they found **triple the level of [prostaglandin D? \(PGD?\)](#)** and a surge in its maker enzyme, PTGDS. When they drenched cultured follicles—or even shaved mice—in PGD?, growth simply stopped. The more PGD?, the shorter the hair. Chemically, PGD? slots into the **CRTH2/DP2 receptor** on follicle cells, flicking on an inflammatory circuit that hustles hairs out of their growth phase. Think of DHT as the arsonist and PGD? as the accelerant—it doesn't start the fire, but it makes sure the building burns.

Big Pharma's Airball on Inhibiting PGD?

Asthma companies already had **DP2 antagonists** on the shelf, so repurposing looked easy money. Oral DP2 antagonists—setipiprant, fevipiprant, and timapiprant—were repurposed from asthma pipelines but never delivered clinical gains. Setipiprant failed a 24-week, 169-subject trial, showing hair counts no better than placebo. Fevipiprant and timapiprant never even started scalp studies after lukewarm asthma data and looming patent expirations. The common lesson: oral dosing keeps blood levels safe for the lungs but too low to saturate follicles, and pushing the dose high enough would raise systemic-safety flags.

Drug (route)	What happened in AGA?	Post-mortem
Setipiprant (oral)	169 men, 24 weeks— no better than placebo	Blood levels safe for lungs never reached follicles oaicite:1
Fevipiprant, Timapiprant (oral)	Never reached a scalp trial	Ho-hum asthma data, patents expiring oaicite:2

Moral: a systemic pill can't fix a problem parked inside an oil-gland funnel unless the dose goes hair-raisingly high, which regulators (and livers) dislike.

Over-The-Counter Underdog: Topical Cetirizine Front and Center

Cetirizine—the familiar non-drowsy allergy pill—can be compounded into a 1 % scalp solution that **down-regulates PTGDS, lowers PGD?, and nudges “pro-growth” PGE? upward**. In a 16-week single-blind pilot of 40 men, topical cetirizine raised total and anagen hair counts to about the same level achieved by 5 % minoxidil ([PubMed](#)). A 24-week double-blind study in 66 women then paired cetirizine with minoxidil and reported thicker shafts, denser vertex coverage, and higher self-ratings than minoxidil plus placebo ([PubMed](#)).

Why Topical Cetirizine Succeeds Where Oral DP2 Blockers Falter

Why might cetirizine show traction when oral DP2 antagonists stalled? Three plausible reasons:

1. **Local delivery, higher follicular exposure.** A 1 % solution can sit in sebum and diffuse directly into the follicle sheath, achieving concentrations oral drugs never reach without systemic-safety trade-offs.
2. **Dual prostaglandin shift.** Cetirizine not only dampens PGD? by suppressing PTGDS but also modestly increases PGE?, a prostaglandin linked to anagen maintenance. The oral DP2 blockers only blocked the receptor, leaving the overall PG balance unchanged.
3. **Extra anti-inflammatory benefit.** As an H? antihistamine, cetirizine calms mast-cell activity and scalp itch—secondary effects that may further reduce micro-inflammation around miniaturising follicles.

Put together, cetirizine delivers more drug exactly where it matters, nudges the prostaglandin balance in two directions, and adds a layer of anti-inflammatory support—advantages the first-generation oral antagonists simply didn't have.

Cetirizine's Off-Label Role in Custom Hair Treatments

Because cetirizine is already FDA-approved for allergies, pharmacies may legally compound it, and tele-derm services—[Roots by GA](#) among them—let physicians slot the molecule into personalised cocktails alongside minoxidil, finasteride, or latanoprost ?[Roots](#). That doesn't make cetirizine `a certified hair-loss cure—large, head-to-head studies are still missing—but the early data highlight a simple principle: **when you shut PGD? down right at the follicle, the follicle listens**.

Synergistic Treatment: Hormones, Growth, Inflammation

A credible protocol might stack:

1. **Hormone control** – dutasteride, finasteride, alongside anti-androgen treatments.
2. **Growth push** – minoxidil, microneedling, exosome cocktails.

3. Inflammation brake – cetirizine now; bespoke DP2 or PTGDS inhibitors later.

Addressing all three fronts is likely to produce better, longer-lasting results than focusing on DHT alone. DHT is still central to androgenetic alopecia, but it does not act in isolation. Elevated PGD? levels add an inflammatory signal that keeps follicles cycling out of growth even when DHT is suppressed. Early setbacks with oral DP2 antagonists showed that systemic delivery isn't enough; treatment has to reach the follicle. Topical cetirizine gives the first proof of concept, and next-generation DP2 or PTGDS inhibitors may refine the approach further.

Citations

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