

RESEARCH MONOGRAPH . COMPOUND PROFILE

GT20029

A first-in-class topical PROTAC androgen receptor degrader developed by Kintor Pharmaceutical for androgenetic alopecia and acne vulgaris, distinguished from conventional antiandrogen therapies by catalytic, substoichiometric receptor protein elimination through the ubiquitin-proteasome system with negligible systemic exposure.

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AUTHOR	Kodi <i>ac</i> biolabs Research
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ABSTRACT

GT20029 is a small-molecule proteolysis-targeting chimera (PROTAC) designed for topical application to skin and scalp, representing the first PROTAC compound to enter and complete clinical trials via the topical route of administration. Developed by Suzhou Kintor Pharmaceutical Ltd. on a proprietary PROTAC platform employing a novel cereblon (CRBN) E3 ubiquitin ligase recruiting element (the TD-106 scaffold), GT20029 functions as a bifunctional molecular bridge: one pharmacophore engages the androgen receptor (AR) ligand-binding domain, a chemical linker spans the two recruiting elements, and a second pharmacophore recruits the CRBN-containing Cullin-RING E3 ubiquitin ligase complex, resulting in polyubiquitination and proteasomal degradation of the AR protein [1, 2]. The catalytic mechanism permits each GT20029 molecule to cycle through multiple rounds of AR degradation, enabling pharmacologically effective AR elimination at low topical concentrations. In preclinical dihydrotestosterone (DHT)-induced mouse models, GT20029 significantly promoted hair regrowth and reversed follicular miniaturization; in testosterone propionate-induced hamster flank organ models, it significantly inhibited sebaceous gland enlargement, supporting the dual indication development for androgenetic alopecia (AGA) and acne vulgaris [3].

Phase I clinical trials were completed in both China (92 subjects) and the United States (123 subjects) as randomized, double-blind, placebo-controlled studies with single ascending dose (SAD) and multiple ascending dose (MAD) cohorts. GT20029 demonstrated excellent safety and tolerability across all dose levels, with no

systemic exposure above the lower limit of quantitation (0.003 ng/mL) in the SAD cohort and maximal plasma concentrations not exceeding 0.015 ng/mL after 14 days of continuous topical application in the MAD cohort, confirming that the compound acts locally with negligible systemic absorption [3]. The Phase II trial for AGA enrolled 180 male patients with Hamilton-Norwood grade IIIv to V pattern hair loss across 12 centers in China in a multicenter, randomized, double-blind, placebo-controlled design. Patients received GT20029 solution at 0.5% or 1.0% concentration, applied once daily (QD) or twice weekly (BIW), for 12 weeks. The primary endpoint was the change from baseline in non-vellus target area hair count (TAHC). The 0.5% QD group demonstrated an increase of 16.80 hairs per square centimeter, 6.69 hairs per square centimeter greater than placebo (P less than 0.05); the 1.0% BIW group demonstrated an increase of 11.94 hairs per square centimeter, 7.36 hairs per square centimeter greater than placebo (P less than 0.05) [4, 5]. Target area hair width also improved significantly in the 1.0% BIW group versus placebo. No adverse sexual events were observed in any treatment group, distinguishing GT20029 from systemic 5-alpha-reductase inhibitors. The Phase II trial for acne vulgaris, conducted across 10 centers in China, also met its primary endpoint for total lesion count reduction (excluding nodules) at the 0.5% QD dose (P = 0.01 versus placebo), with both 0.5% and 1.0% QD groups achieving highly significant reductions in inflammatory lesion counts (P less than 0.01) [6].

On the basis of these results, Kintor has advanced GT20029 into Phase III clinical trials for male AGA in China and has initiated Phase II trials in the United States. The compound is not yet registered in any jurisdiction. The chemical structure of GT20029 has not been publicly disclosed; the molecular formula, molecular

weight, and Chemical Abstracts Service registry number are not available in the public domain. This monograph reviews the available pharmacology, mechanism of action, preclinical and clinical evidence, pharmacokinetics, safety profile, handling considerations, and a comparative assessment of GT20029 against five alternative approaches to androgen-mediated dermatological conditions.

1. Compound Identification

1.1 Names, synonyms, and registry identifiers

Development code: GT20029 (Kintor Pharmaceutical Ltd.; also rendered GT-20029 in some publications).

Alternative designations: AR-PROTAC (generic class descriptor used in early Kintor communications).

Sponsor: Suzhou Kintor Pharmaceutical Ltd. (also referred to as Kintor Pharma), headquartered in Suzhou, China, with clinical operations in China and the United States.

Chemical Abstracts Service registry number: Not publicly disclosed as of the date of this monograph. The compound has not been assigned an International Nonproprietary Name (INN) or a United States Adopted Name (USAN).

International Union of Pure and Applied Chemistry name: Not publicly disclosed. The chemical structure of GT20029 has not been released in the public domain, in peer-reviewed publications, or in accessible patent disclosures at sufficient specificity to permit independent structural characterization [7].

1.2 Structural class and chemistry

GT20029 belongs to the proteolysis-targeting chimera (PROTAC) class of heterobifunctional small molecules. PROTACs are composed of three covalently linked modules: (i) a ligand that binds the target protein of interest (in this case, the androgen receptor ligand-binding domain); (ii) a ligand that recruits an E3 ubiquitin ligase; and (iii) a chemical linker connecting modules (i) and (ii) [1, 2]. The resulting ternary complex (target protein, PROTAC, E3 ligase) positions the target protein for polyubiquitination by the E3 ligase, marking it for degradation by the 26S proteasome.

Kintor's PROTAC platform for GT20029 employs the TD-106 scaffold as the cereblon (CRBN) E3 ubiquitin ligase recruiting element, a proprietary derivative distinct from the thalidomide, lenalidomide, and pomalidomide scaffolds used in earlier-generation CRBN-recruiting PROTACs [8]. Published patent analysis indicates

that the linker attachment at the 6-position of the TD-106 scaffold is critical for efficient AR degradation, with compounds attached at the 5- and 7-positions showing reduced degradation efficiency [8]. The AR-binding pharmacophore is derived from known AR ligand scaffolds, though the specific warhead has not been publicly disclosed.

Molecular formula: Not publicly disclosed.

Molecular weight: Not publicly disclosed. PROTAC molecules typically range from 700 to 1200 daltons owing to the bifunctional architecture and linker requirements. GT20029 is expected to fall within this range based on the structural class, though this has not been confirmed.

Physical properties: GT20029 is formulated as a topical solution (for AGA trials) and as a topical gel (for acne trials). The physical form of the active pharmaceutical ingredient (crystalline, amorphous, or salt form) has not been disclosed. Solubility characteristics have not been publicly reported beyond the formulation vehicles used in clinical trials.

Stereochemistry: Not publicly disclosed.

1.3 Synthesis

The synthetic route for GT20029 has not been published in the peer-reviewed literature or disclosed in accessible patent filings at sufficient detail for independent replication. Based on the general PROTAC synthesis paradigm and the disclosed use of the TD-106 CRBN-binding scaffold, the synthesis is expected to proceed through: (i) preparation or sourcing of the AR-binding warhead; (ii) preparation of the TD-106-derived CRBN ligand with a linker attachment point at the 6-position; (iii) conjugation of the AR warhead and the CRBN ligand through the chemical linker, typically via amide coupling, click chemistry, or nucleophilic substitution depending on the linker design; and (iv) purification by preparative high-performance liquid chromatography or recrystallization [1, 8]. Kintor has stated that the compound is manufactured under current Good Manufacturing Practice (cGMP) conditions for clinical supply.

2. Abstract

GT20029 is a first-in-class topical proteolysis-targeting chimera (PROTAC) that induces the degradation of androgen receptor (AR) protein through recruitment of the cereblon-containing E3 ubiquitin ligase complex and subsequent ubiquitin-proteasome-mediated proteolysis [1, 2]. The compound was developed by Suzhou Kintor Pharmaceutical Ltd. for the treatment of androgenetic alopecia (AGA) and acne vulgaris, two dermatological conditions in which AR signaling in the hair follicle dermal papilla and the pilosebaceous unit, respectively, drives pathological changes in response to circulating androgens, principally dihydrotestosterone (DHT) and testosterone [9, 10].

The PROTAC mechanism of GT20029 is fundamentally distinct from the two established pharmacological approaches to androgen-mediated hair loss: 5-alpha-reductase inhibition (finasteride, dutasteride), which reduces circulating and tissue DHT concentrations without affecting AR protein levels; and AR competitive antagonism (pyrilutamide/KX-826, topical bicalutamide analogs), which blocks ligand binding to AR but paradoxically stabilizes and upregulates the receptor protein [11, 12]. By eliminating the AR protein itself, GT20029 removes the signaling node through which androgens act on target tissues, and the catalytic nature of PROTAC-mediated degradation permits each drug molecule to cycle through multiple rounds of target elimination, enabling efficacy at low tissue concentrations [1].

Preclinical pharmacology in DHT-induced mouse alopecia models demonstrated significant hair regrowth promotion with topical GT20029, and in testosterone propionate-induced hamster flank organ models, the compound significantly inhibited sebaceous gland hypertrophy [3]. Phase I trials in China (92 subjects) and the United States (123 subjects) established safety, tolerability, and the absence of meaningful systemic exposure (maximal plasma concentration below 0.015 ng/mL after 14 days of continuous topical dosing), confirming that the pharmacological activity is confined to the site of application [3].

The Phase II AGA trial (180 patients, 12 centers, China) met its primary endpoint: the 0.5% QD group achieved a 16.80 hairs per square centimeter increase from baseline (6.69 hairs per square centimeter greater than placebo, P less than 0.05), and the 1.0% BIW group achieved an 11.94 hairs per square centimeter increase

(7.36 hairs per square centimeter greater than placebo, P less than 0.05) [4, 5]. The Phase II acne trial (10 centers, China) also met its primary endpoint, with significant reductions in total lesion counts and inflammatory lesion counts at both the 0.5% and 1.0% QD doses [6]. No sexual adverse events were reported in any trial, a clinically meaningful differentiation from systemic finasteride and dutasteride.

GT20029 is currently in Phase III clinical trials for male AGA in China and Phase II trials in the United States. The compound is not registered in any jurisdiction. It is the first topical PROTAC to complete Phase I and Phase II clinical trials globally. This monograph documents the available pharmacology, mechanism, preclinical and clinical evidence, pharmacokinetics, safety, and comparative positioning of GT20029 within the androgen-mediated dermatological therapy landscape.

3. Discovery and Development History

3.1 Origin and PROTAC platform

GT20029 emerged from Kintor Pharmaceutical's proprietary PROTAC drug discovery platform, which was established to develop targeted protein degraders for androgen receptor-driven conditions. Kintor, founded in 2009 and headquartered in Suzhou, China, had previously advanced the topical AR antagonist pyrilutamide (KX-826) into clinical development for AGA and acne vulgaris. The development of GT20029 represented a mechanistic escalation from AR antagonism (competitive inhibition) to AR degradation (protein elimination), leveraging the PROTAC platform to address limitations of conventional AR-targeting pharmacology, including receptor stabilization and upregulation associated with competitive antagonists [11, 12].

The PROTAC technology underlying GT20029 builds on foundational work by Sakamoto et al. (2001) and Crews and colleagues, who demonstrated that bifunctional molecules linking a target-protein ligand to an E3 ubiquitin ligase recruiting element could redirect the cellular ubiquitin-proteasome system to degrade selected proteins [13]. The Kintor platform specifically employed the TD-106 scaffold, a proprietary cereblon (CRBN) E3 ligase binder identified in the company's internal medicinal chemistry program, which offered advantages in molecular weight, drug-like properties, and degradation efficiency relative to the thalidomide-derived CRBN binders used in earlier PROTAC programs [8].

3.2 Preclinical development

Preclinical pharmacology and toxicology studies for GT20029 were conducted in mouse and hamster models. In the DHT-induced alopecia mouse model, topical GT20029 significantly promoted hair regrowth compared to vehicle control, with histological evidence of reversal of follicular miniaturization. In the testosterone propionate-induced hamster flank organ model, GT20029 significantly inhibited sebaceous gland enlargement, supporting the dual indication of AGA and acne [3]. The preclinical pharmacodynamic studies also included a representative degrader compound from the Kintor series (designated 33c or TD-802), which demonstrated a

half-maximal degradation concentration (DC50) of 12.5 nanomolar and a maximum degradation (Dmax) of 93% for AR protein in LNCaP prostate cancer cells, indicating potent and near-complete AR elimination in vitro [8].

3.3 Regulatory milestones and clinical timeline

The Investigational New Drug (IND) application for GT20029 was accepted by China's National Medical Products Administration (NMPA) in February 2021 for the indications of androgenetic alopecia and acne vulgaris [14]. The United States Food and Drug Administration (FDA) cleared the IND application in July 2021 [15].

The China Phase I clinical trial enrolled 92 subjects and was completed in August 2022, with top-line results announced in November 2022 [16]. The United States Phase I clinical trial enrolled 123 subjects, with completion of enrollment and dosing announced in October 2022 and positive top-line results announced in February 2023 [3, 17].

The China Phase II trial for male AGA enrolled 180 patients across 12 centers between April 2023 and April 2024, with primary endpoint results announced in April 2024 [4, 5]. The Phase II trial for acne vulgaris was conducted across 10 centers in China; primary endpoint results were announced in August 2025 [6].

On the basis of the Phase II AGA results, Kintor initiated Phase III clinical trials for male AGA in China and Phase II trials in the United States. As of the date of this monograph (May 2026), Phase III enrollment in China is ongoing and Phase II enrollment in the United States has commenced [18, 19].

3.4 Significance in the PROTAC field

GT20029 is the first PROTAC compound developed for topical administration that has entered and completed human clinical trials. All prior PROTAC candidates in clinical development, including bavdegalutamide (ARV-110, Arvinas) and ARV-471 (Arvinas) for oncology indications, were designed for oral systemic administration [20, 21]. The topical approach addresses a fundamental challenge of the PROTAC class: the high molecular weight of bifunctional degraders (typically 700 to 1200 daltons) violates Lipinski's rule of five and limits oral bioavailability. By formulating GT20029 for topical application to the scalp or skin, Kintor circumvented the oral absorption barrier and simultaneously achieved the clinical objective of local AR degradation without systemic exposure [3].

4. Mechanism of Action

4.1 The ubiquitin-proteasome system and PROTAC technology

The ubiquitin-proteasome system (UPS) is the principal intracellular protein degradation pathway in eukaryotic cells. Target proteins are marked for destruction by covalent attachment of polyubiquitin chains through a three-enzyme cascade: E1 (ubiquitin-activating enzyme), E2 (ubiquitin-conjugating enzyme), and E3 (ubiquitin ligase). The polyubiquitinated protein is then recognized and degraded by the 26S proteasome, a large multisubunit protease complex [22].

PROTAC molecules co-opt this endogenous degradation machinery by simultaneously binding the target protein and an E3 ubiquitin ligase, forming a ternary complex (target protein:PROTAC:E3 ligase) that positions the target for ubiquitination. After ubiquitination and release of the target protein to the proteasome, the PROTAC molecule dissociates and is free to engage another target protein molecule, enabling catalytic, substoichiometric degradation [1, 13].

4.2 GT20029 target engagement and AR degradation

GT20029 binds the ligand-binding domain of the androgen receptor through its AR-recruiting pharmacophore and simultaneously recruits the cereblon (CRBN)-containing Cullin 4-RING E3 ubiquitin ligase complex (CRL4-CRBN) through its TD-106-derived CRBN ligand [2, 8]. The ternary complex (AR:GT20029:CRL4-CRBN) positions the AR for Lys48-linked polyubiquitination, the canonical proteasomal degradation signal. The polyubiquitinated AR is subsequently recognized and degraded by the 26S proteasome.

The catalytic nature of this mechanism is a defining pharmacological property. Unlike competitive AR antagonists (enzalutamide, bicalutamide, pyrilutamide), which require continuous stoichiometric occupancy of the AR ligand-binding pocket and which stabilize the AR protein and can promote compensatory receptor upregulation, GT20029 eliminates the AR protein from the cell [1, 11]. Each GT20029

molecule can cycle through multiple rounds of AR engagement, ubiquitination, and release, enabling effective AR depletion at drug concentrations substantially below those required for conventional receptor occupancy-driven pharmacology.

4.3 AR signaling in androgenetic alopecia

Androgenetic alopecia is driven by androgen action on the dermal papilla cells of genetically susceptible hair follicles, predominantly in the frontal and vertex scalp regions. Circulating testosterone is converted to the more potent androgen DHT by the enzyme 5-alpha-reductase (types I and II) within dermal papilla cells. DHT binds the intracellular AR with approximately five-fold higher affinity than testosterone, inducing AR nuclear translocation, dimerization, and transcriptional activation of androgen-responsive genes that promote follicular miniaturization: progressive shortening of the anagen (growth) phase, reduction in hair shaft diameter, and eventual conversion of terminal hairs to vellus hairs [9, 10].

GT20029, applied topically to the affected scalp, degrades AR protein within the dermal papilla and surrounding follicular structures, thereby eliminating the intracellular receptor through which DHT and testosterone exert their miniaturizing effects. This mechanism addresses the disease at the level of the signaling receptor rather than upstream (5-alpha-reductase inhibition, which reduces DHT but does not eliminate AR sensitivity to residual androgens) or at the level of ligand-receptor binding (competitive antagonism, which occupies the receptor but does not remove it) [11, 12].

4.4 AR signaling in acne vulgaris

In the pilosebaceous unit, AR activation by circulating androgens stimulates sebocyte proliferation and sebum production, contributing to the follicular occlusion, comedogenesis, and inflammatory cascade that characterize acne vulgaris [23]. AR expression in sebaceous glands is high, and androgen sensitivity of the pilosebaceous unit is a principal determinant of acne severity in both sexes. GT20029 applied topically to acne-affected skin degrades AR in sebocytes and surrounding dermal cells, reducing androgen-driven sebum production and sebaceous gland hypertrophy without affecting systemic androgen levels or hypothalamic-pituitary-gonadal axis function [6].

4.5 Selectivity and off-target considerations

The selectivity of GT20029 for the androgen receptor relative to other nuclear hormone receptors (estrogen receptor alpha and beta, progesterone receptor, glucocorticoid receptor, mineralocorticoid receptor) has not been reported in public-domain publications. PROTAC selectivity is determined both by the binding selectivity of the target-recruiting warhead and by the geometry of the induced ternary complex, which must be compatible with productive ubiquitination; off-target degradation requires both binding and a permissive ternary complex geometry [1]. The negligible systemic exposure observed in Phase I trials (plasma concentrations below 0.015 ng/mL) provides an additional margin of selectivity by confining pharmacological activity to the site of topical application [3].

The CRBN E3 ligase recruited by GT20029 is a constitutively expressed, broadly distributed E3 ligase that participates in endogenous protein homeostasis. CRBN-recruiting PROTACs do not inhibit CRBN function; they transiently redirect its ubiquitin ligase activity toward the intended target. The thalidomide-class immunomodulatory drugs (lenalidomide, pomalidomide) act through CRBN to degrade neosubstrates (Ikaros, Aiolos); however, PROTAC-mediated CRBN engagement does not produce neosubstrate degradation because the PROTAC occupies the substrate-binding pocket of CRBN and directs activity exclusively toward the tethered target protein [22].

5. Pharmacokinetics and Routes of Administration

5.1 Route of administration

GT20029 is administered topically. The AGA formulation is a solution applied to the affected scalp area; the acne formulation is a gel applied to affected facial or truncal skin. No oral, parenteral, or other systemic routes of administration have been studied or are planned.

5.2 Absorption and systemic exposure

The Phase I clinical trials in both China and the United States established that GT20029 produces negligible systemic absorption after topical application [3]. In the single ascending dose (SAD) cohort of healthy volunteers, no measurable systemic exposure was detected; plasma concentrations of GT20029 remained below the lower limit of quantitation of 0.003 ng/mL at all time points. In the multiple ascending dose (MAD) cohort of AGA and acne patients receiving 14 days of continuous topical application, the maximal plasma concentration (C_{max}) did not exceed 0.015 ng/mL, a level approximately three to five orders of magnitude below the concentrations at which systemic AR degradation would be expected based on in vitro degradation potency data [3, 8].

The negligible systemic exposure is attributable to a combination of factors: the high molecular weight of the PROTAC molecule (expected to exceed 700 daltons), limiting passive transdermal diffusion into the systemic circulation; the topical formulation design, which is optimized for penetration into the epidermis and upper dermis (where the hair follicle bulge, dermal papilla, and sebaceous gland reside) rather than transdermal absorption; and the likely rapid local metabolism or binding of the compound within the dermal compartment.

5.3 Distribution

Systemic distribution of GT20029 has not been characterized because plasma concentrations are at or below the limit of quantitation after topical application. The compound is expected to distribute locally within the epidermis, dermis, and hair follicle structures at the site of application. Dermal pharmacokinetic studies (tape stripping, skin biopsy quantitation) have not been reported in the public domain.

5.4 Metabolism

The metabolic fate of GT20029 has not been reported in public-domain publications. Based on the general properties of PROTAC molecules and the negligible systemic exposure, hepatic metabolism is not expected to be a significant determinant of drug disposition. Local cutaneous metabolism (esterases, amidases, cytochrome P450 enzymes expressed in skin) may contribute to compound clearance from the dermal compartment, but this has not been characterized.

5.5 Elimination

Elimination pathways for GT20029 have not been reported. Given the absence of meaningful systemic absorption, renal and hepatic elimination are expected to be minimal. Local clearance from the application site is likely dominated by surface removal (washing, desquamation) and local metabolism.

5.6 Special populations

Pediatric, geriatric, and organ-impairment pharmacokinetic studies have not been reported. The Phase I and Phase II trials enrolled adult male patients (for AGA) and adult patients of both sexes (for acne); age ranges and demographic characteristics have not been fully published. The negligible systemic exposure profile suggests that hepatic and renal impairment are unlikely to affect GT20029 pharmacokinetics, but this has not been formally studied.

6. Clinical Evidence Base

6.1 Phase I clinical trials

6.1.1 China Phase I trial

A randomized, double-blind, placebo-controlled Phase I clinical trial was conducted in China, enrolling 92 subjects in SAD (healthy volunteers) and MAD (AGA and acne patients) cohorts. The trial was completed in August 2022 with top-line results announced in November 2022 [16]. GT20029 was safe and well tolerated at all dose levels in all cohorts. No treatment-emergent adverse events (TEAEs) related to GT20029 were reported in the SAD stage. In the MAD stage, mild local application-site reactions (dryness, itching, burning, pain) were the most common TEAEs [16].

6.1.2 United States Phase I trial

A randomized, double-blind, placebo-controlled Phase I clinical trial was conducted in the United States, enrolling 123 subjects in SAD (healthy volunteers) and MAD (AGA and acne patients) cohorts [3, 17]. Enrollment and dosing were completed in October 2022, with positive top-line results announced in February 2023. Results were consistent with the China Phase I trial: GT20029 was safe and well tolerated at all dose levels, with no serious adverse events, no Grade 3 or higher adverse events, and no treatment-related study withdrawals. Systemic exposure was negligible (SAD: below 0.003 ng/mL at all time points; MAD: C_{max} not exceeding 0.015 ng/mL after 14 days) [3].

6.2 Phase II clinical trial for androgenetic alopecia

6.2.1 Study design

A multicenter, randomized, double-blind, placebo-controlled Phase II trial was conducted across 12 clinical research centers in China under the direction of Professor Yang Qiping at Fudan University Huashan Hospital [4, 5]. The trial enrolled 180 male patients with Hamilton-Norwood grade IIIv to V AGA between April 2023 and April 2024. Patients were randomized equally into six groups: GT20029 0.5% QD

(once daily), GT20029 1.0% QD, GT20029 0.5% BIW (twice weekly), GT20029 1.0% BIW, and corresponding placebo groups for each dosing frequency. Treatment duration was 12 weeks.

6.2.2 Primary endpoint

The primary endpoint was the average change from baseline in non-vellus target area hair count (TAHC) at 12 weeks compared to placebo [4, 5].

6.2.3 Efficacy results

All four GT20029-treated groups showed increases in TAHC from baseline. The primary endpoint was met in two dosing groups:

The 0.5% QD group achieved an increase of 16.80 hairs per square centimeter from baseline, representing a treatment difference of 6.69 hairs per square centimeter versus placebo (P less than 0.05) [4, 5].

The 1.0% BIW group achieved an increase of 11.94 hairs per square centimeter from baseline, representing a treatment difference of 7.36 hairs per square centimeter versus placebo (P less than 0.05) [4, 5].

A dose-response relationship was observed across the treatment groups. Target area hair width (TAHW) also improved significantly in the 1.0% BIW group versus placebo, indicating reversal of follicular miniaturization at the structural level [5].

6.2.4 Safety

The incidence of treatment-emergent adverse events was comparable between GT20029-treated groups and placebo. No sexual adverse events (erectile dysfunction, decreased libido, ejaculatory disorders) were reported in any treatment group [4, 5]. This finding is clinically significant given that sexual adverse events are the principal tolerability limitation of systemic 5-alpha-reductase inhibitors (finasteride, dutasteride) in the treatment of AGA [24].

6.2.5 Recommended Phase III dose

Based on the efficacy, safety, and convenience profile, the 1.0% BIW dosing regimen was selected as the recommended dose for Phase III clinical trials in China [5].

6.3 Phase II clinical trial for acne vulgaris

A multicenter, randomized, double-blind, placebo-controlled Phase II trial for acne vulgaris was conducted across 10 clinical research centers in China under the direction of Professor Xiang Leihong at Fudan University Huashan Hospital [6]. Two GT20029 gel treatment groups (0.5% QD and 1.0% QD) were compared to placebo.

6.3.1 Primary endpoint

The primary endpoint was the change from baseline in total lesion counts (excluding nodules) at the end of treatment [6].

6.3.2 Efficacy results

The primary endpoint was met in the 0.5% QD group ($P = 0.01$ versus placebo); the 1.0% QD group approached significance ($P = 0.05$) [6]. For inflammatory lesion counts, both the 0.5% QD and 1.0% QD groups achieved P less than 0.01 versus placebo [6]. For non-inflammatory lesion counts, neither group reached statistical significance (0.5%: $P = 0.14$; 1.0%: $P = 0.09$) [6]. The Investigator's Global Assessment (IGA) success rate was significant in the 0.5% QD group ($P = 0.03$ versus placebo) and non-significant in the 1.0% QD group ($P = 0.15$) [6].

6.3.3 Safety

GT20029 gel demonstrated satisfactory safety and tolerability. The overall incidence of adverse events was low, and drug-related adverse events in the 0.5% and 1.0% groups were comparable and lower than in the placebo group, with mild severity [6]. The recommended dose for Phase III acne trials was determined to be 0.5% QD [6].

6.4 Ongoing and planned clinical trials

As of May 2026, the following trials are ongoing or planned:

Phase III clinical trial for male AGA in China (NCT06692465 and related registrations), with the 1.0% BIW dosing regimen as the primary treatment arm [18, 19].

Phase II clinical trial for male AGA in the United States, with enrollment commencing [18].

Phase III clinical trial for acne vulgaris in China is anticipated on the basis of the positive Phase II results but has not been publicly registered as of the monograph date [6].

7. Reported Research Dose Ranges

7.1 Preclinical doses

Preclinical DHT-induced mouse alopecia model: topical GT20029 at concentrations and application frequencies that have not been publicly specified in detail. The representative PROTAC compound TD-802 (33c) demonstrated a DC50 of 12.5 nanomolar for AR degradation in LNCaP cells in vitro [8].

Preclinical testosterone propionate-induced hamster flank organ model: topical GT20029 at concentrations not publicly specified [3].

7.2 Phase I doses

SAD stage (healthy volunteers): ascending single doses of GT20029 solution or gel at concentrations that have not been publicly specified in full detail [3].

MAD stage (AGA and acne patients): multiple ascending doses over 14 days at concentrations that have not been publicly specified in full detail [3].

7.3 Phase II doses

AGA trial: GT20029 solution at 0.5% and 1.0% concentrations, applied once daily (QD) or twice weekly (BIW) for 12 weeks [4, 5].

Acne trial: GT20029 gel at 0.5% and 1.0% concentrations, applied once daily (QD) for the treatment duration [6].

7.4 Recommended clinical doses

Male AGA (for Phase III): 1.0% BIW topical solution, applied to the affected scalp area [5].

Acne vulgaris (for Phase III): 0.5% QD topical gel, applied to affected skin [6].

Investigators acquiring GT20029 for research purposes should note that the compound is not available as a research-grade commercial preparation. Access is limited to clinical trial participation and to collaborations with Kintor Pharmaceutical.

8. Sourcing and Quality Verification

GT20029 is not commercially available as a pharmaceutical product or as a research-grade chemical compound. The compound is manufactured by Kintor Pharmaceutical Ltd. under cGMP conditions for clinical trial supply.

Commercial pharmaceutical sources: None. The compound is not registered in any jurisdiction and is not marketed under any brand name.

Research-grade chemical suppliers: GT20029 is not listed in the catalogs of major research chemical suppliers (Sigma-Aldrich, Tocris, Cayman Chemical, MedChemExpress, Selleckchem) as of the date of this monograph. The chemical structure has not been publicly disclosed, which precludes independent synthesis by third parties.

Access for research: Investigators wishing to study GT20029 must engage with Kintor Pharmaceutical through clinical trial participation, investigator-initiated study proposals, or material transfer agreements. Kintor has conducted trials at sites in China and the United States and may consider collaborative research requests.

Quality verification: For any GT20029 material obtained through collaboration, investigators should confirm:

- Identity by mass spectrometry or nuclear magnetic resonance spectroscopy (against a Kintor-provided reference standard)
- Purity by high-performance liquid chromatography (target greater than 95 percent)
- Formulation concentration by validated analytical method
- Stability under intended storage conditions

The absence of commercial availability and the undisclosed chemical structure distinguish GT20029 from the majority of compounds reviewed in this monograph series and impose practical limitations on independent research applications.

9. Reconstitution and Handling

GT20029 is supplied for clinical trials as a ready-to-use topical formulation (solution for AGA, gel for acne) and does not require reconstitution by the end user. The following handling considerations apply based on the available information:

Storage: Storage conditions for the clinical formulation have not been publicly disclosed. Standard practice for topical pharmaceutical formulations suggests storage at controlled room temperature (20 to 25 degrees Celsius) with protection from light and humidity. Refrigerated storage (2 to 8 degrees Celsius) may be specified for the active pharmaceutical ingredient.

Application: For the AGA indication, the GT20029 solution is applied directly to the affected scalp area using the applicator provided. For the acne indication, the GT20029 gel is applied to the affected facial or truncal skin. Application volumes and techniques have not been publicly specified in detail.

Stability: Stability data for the formulated product have not been published. PROTAC molecules are generally stable in the solid state and in anhydrous formulations but may be susceptible to hydrolysis of the linker or the E3 ligase recruiting element in aqueous solution at elevated temperatures or extreme pH values.

Handling precautions: As with all investigational compounds, GT20029 should be handled with appropriate personal protective equipment (gloves, laboratory coat). The compound is intended for topical application to human skin and is not expected to pose acute toxicity hazards by dermal contact, based on the clinical safety profile established in Phase I and Phase II trials.

10. Stack Interaction Considerations

GT20029 is a topically applied compound with negligible systemic absorption (plasma C_{max} below 0.015 ng/mL after 14 days of continuous application). The absence of meaningful systemic exposure substantially limits the potential for pharmacokinetic drug-drug interactions through hepatic cytochrome P450 enzymes, transporters, or plasma protein binding [3].

Topical co-application considerations:

Minoxidil: The most common topical treatment for AGA. No pharmacokinetic interaction is expected because the two compounds act through independent mechanisms (minoxidil through potassium channel opening and vasodilation, GT20029 through AR degradation). Co-application has not been formally studied. Investigators considering concurrent topical minoxidil and GT20029 should apply the two formulations at separate times (morning and evening) to avoid formulation incompatibility at the application site.

Topical retinoids (tretinoin, adapalene, tazarotene): Commonly used for acne. Topical retinoids alter epidermal barrier function and may theoretically increase dermal penetration of co-applied compounds. No interaction data are available. Sequential application with adequate drying time is advisable.

Topical corticosteroids: May be used for concurrent scalp or facial inflammatory conditions. No interaction data are available. AR and glucocorticoid receptor are structurally related nuclear hormone receptors; theoretical concerns about cross-reactivity of the GT20029 AR-recruiting warhead with the glucocorticoid receptor have not been addressed by published selectivity data.

Topical ketoconazole (antifungal shampoo): Commonly used as adjunctive therapy for AGA. No pharmacokinetic interaction is expected.

Systemic co-administration considerations:

5-alpha-reductase inhibitors (finasteride, dutasteride): The combination of systemic 5-alpha-reductase inhibition with topical AR degradation is mechanistically complementary (reduced DHT production plus reduced AR protein). The combination has not been studied. The theoretical benefit is additive efficacy; the theoretic

cal risk is excessive androgen pathway suppression at the target tissue level, though systemic androgen levels would be affected only by the 5-alpha-reductase inhibitor, not by GT20029.

Oral antiandrogens (spironolactone, cyproterone acetate): Used in female-pattern hair loss and acne. The combination with topical GT20029 has not been studied. Additive antiandrogen activity at the target tissue is expected.

Hormonal contraceptives: No interaction expected given the absence of systemic GT20029 exposure.

CYP450 inhibitors and inducers: No systemic interaction expected given the absence of meaningful systemic GT20029 exposure.

11. Adverse Events and Safety

11.1 Common adverse events

The adverse-event profile of GT20029 from Phase I and Phase II trials is characterized by mild, local application-site reactions [3, 4, 5, 6]:

- Dryness at the application site (frequency not quantified in public-domain reports; observed in MAD cohorts)
- Itching at the application site (mild, transient)
- Burning sensation at the application site (mild, transient)
- Pain at the application site (mild, infrequent)

These local reactions are consistent with the vehicle formulation effects commonly observed in topical dermatological trials and were not clearly dose-dependent in the reported data.

11.2 Absence of systemic adverse events

No systemic adverse events attributable to GT20029 were reported in Phase I or Phase II trials [3, 4, 5, 6]. Critically, no sexual adverse events (erectile dysfunction, decreased libido, ejaculatory disorders, gynecomastia) were observed in any treatment group in any trial. This finding is consistent with the negligible systemic exposure and local mechanism of action, and represents a clinically meaningful safety advantage relative to systemic 5-alpha-reductase inhibitors [24].

No serious adverse events were reported in any GT20029-treated subject. No Grade 3 or higher adverse events occurred. No subjects withdrew from studies due to treatment-related adverse events [3].

11.3 Laboratory and hormonal safety

Changes in systemic hormone levels (testosterone, DHT, luteinizing hormone, follicle-stimulating hormone) have not been reported in public-domain publications but are expected to be absent given the negligible systemic exposure. No hepatic

enzyme elevations, hematological abnormalities, or other laboratory safety signals have been reported [3].

11.4 Theoretical safety considerations

CRBN engagement: The theoretical concern that PROTAC-mediated CRBN engagement could produce immunomodulatory effects (analogous to thalidomide-class drugs that degrade CRBN neosubstrates Ikaros and Aiolos) is mitigated by two factors: (i) PROTAC molecules occupy the CRBN substrate-binding pocket and direct activity exclusively toward the tethered target (AR), precluding neosubstrate engagement; and (ii) the negligible systemic exposure limits any CRBN-mediated off-target activity to the local dermal compartment [22].

Long-term AR depletion in skin: The consequences of chronic AR protein depletion in dermal and follicular cells over years of continuous topical application have not been characterized. AR plays physiological roles in wound healing, sebum homeostasis, and hair follicle cycling beyond the pathological miniaturization response. Long-term safety data from Phase III trials and post-marketing surveillance will be necessary to characterize this risk.

11.5 Pregnancy and lactation

GT20029 has not been studied in pregnant or lactating women. The compound is under development for male AGA (Phase III) and for acne (both sexes). Given the local mechanism of action and negligible systemic exposure, teratogenic risk is theoretically low but has not been formally assessed in reproductive toxicology studies available in the public domain. Use in pregnancy and lactation cannot be recommended without additional safety data.

12. Comparable Compounds Assessment

This section presents a structured comparative assessment of GT20029 against five alternative approaches to androgen-mediated dermatological conditions, evaluated on five competency standards: novelty, effect size, promising potential (current clinical state and trajectory), side-effect profile (clinical), and overall validation. The comparators were selected to span the principal pharmacological classes relevant to AGA and acne treatment, from established therapies to emerging investigational compounds.

12.1 Finasteride (oral, 1 mg daily for AGA)

Mechanism: Selective inhibitor of type II 5-alpha-reductase. Reduces conversion of testosterone to DHT in target tissues and systemically. Does not affect AR protein levels; residual DHT and testosterone continue to activate AR. Oral administration with complete systemic bioavailability [24].

Novelty: Low. Finasteride has been FDA-approved for male AGA since 1997 (Propecia). The mechanism is well characterized and the compound is the reference standard for AGA pharmacotherapy.

Effect size: Moderate. In pivotal registration trials, finasteride 1 mg daily produced a mean increase of approximately 12 to 16 hairs per square centimeter from baseline at 12 months in the vertex, with approximately 48 percent of patients showing visible improvement on investigator assessment at 1 year and 66 percent at 2 years [24, 25]. The GT20029 Phase II data (16.80 hairs per square centimeter increase at 12 weeks with 0.5% QD) are comparable in magnitude to finasteride at a substantially earlier time point, though cross-trial comparisons are limited by differences in patient populations, scalp regions assessed, and measurement methodologies.

Promising potential: Established. Finasteride is the standard of care for male AGA and is approved in most jurisdictions globally. Generic formulations are widely available at low cost.

Side-effect profile: The principal tolerability limitation of finasteride is sexual adverse events. In registration trials, erectile dysfunction (1.3% versus 0.7% placebo), decreased libido (1.8% versus 1.3%), and ejaculatory disorders (1.2% versus 0.7%) were reported [24]. Post-marketing reports of persistent sexual dysfunction following finasteride discontinuation ("post-finasteride syndrome") have attracted substantial attention, though the incidence, causality, and mechanism remain subjects of debate [26]. GT20029 has not produced any sexual adverse events in clinical trials, representing a clear safety differentiation.

Overall validation: High. Finasteride has the strongest evidence base and longest clinical track record of any AGA pharmacotherapy. The compound is limited by the sexual adverse-event signal, which reduces patient acceptance and adherence.

Comparison to GT20029: GT20029 offers a mechanistically distinct approach (receptor degradation versus enzyme inhibition) with the critical advantage of local activity and absence of systemic exposure, eliminating the sexual adverse-event concern. Finasteride offers established long-term efficacy data, global availability, and low cost. The two compounds could be mechanistically complementary (reduced DHT plus reduced AR), though combination studies have not been conducted.

12.2 Dutasteride (oral, 0.5 mg daily for AGA)

Mechanism: Dual inhibitor of type I and type II 5- α -reductase. Produces more complete suppression of serum and tissue DHT (approximately 90% reduction versus approximately 70% with finasteride) [27]. Oral administration.

Novelty: Low. Dutasteride is approved for benign prostatic hyperplasia in most jurisdictions and is used off-label (or approved in some jurisdictions, notably South Korea and Japan) for male AGA.

Effect size: Moderate to high. Direct comparative trials have shown dutasteride 0.5 mg to be superior to finasteride 1 mg for AGA, with greater increases in hair count and hair width at 24 weeks [27]. The more complete DHT suppression translates to a modestly greater clinical effect, though the dose-response relationship on DHT suppression plateaus at approved doses.

Promising potential: Established, with ongoing adoption for AGA in more jurisdictions.

Side-effect profile: Similar to finasteride but with modestly higher rates of sexual adverse events (erectile dysfunction, decreased libido, ejaculatory disorders) consistent with the more complete androgen pathway suppression [27]. The same post-marketing concerns regarding persistent sexual dysfunction apply. Additionally, dutasteride has a substantially longer elimination half-life (approximately 5 weeks) than finasteride (approximately 6 hours), meaning that adverse effects persist longer after discontinuation.

Overall validation: High. The evidence base is slightly smaller than finasteride for AGA specifically but is supported by large benign prostatic hyperplasia trials and by direct comparative AGA data.

Comparison to GT20029: The comparison parallels finasteride. GT20029 offers the advantage of local activity, absence of systemic exposure, and no sexual adverse events. Dutasteride offers more complete DHT suppression than finasteride and may produce greater efficacy, but at the cost of a higher adverse-event burden and a very long half-life.

12.3 Pyrilutamide (KX-826, topical, for AGA)

Mechanism: Topical competitive androgen receptor antagonist. Binds the AR ligand-binding domain and competitively blocks DHT and testosterone binding. Does not degrade AR protein; may stabilize and upregulate AR expression through the well-characterized antagonist-induced receptor stabilization phenomenon [11, 12]. Developed by Kintor Pharmaceutical (the same sponsor as GT20029).

Novelty: Moderate. Pyrilutamide is the first topical AR antagonist to reach Phase III clinical trials for AGA. The topical route is novel for AR antagonists, though the mechanism (competitive AR antagonism) is well established from systemic antiandrogens used in prostate cancer.

Effect size: Mixed. The China Phase III trial for male AGA did not achieve statistically significant superiority over placebo on the primary endpoint of TAHC change, though a trend toward efficacy was observed [28]. More recent Phase III data (March 2026) reportedly showed improved results, and Kintor has announced plans to submit a New Drug Application (NDA) to NMPA in 2026 with anticipated commercialization in 2027 [28]. The Phase II AGA data for GT20029 showed statistically significant superiority over placebo at 12 weeks, a result that pyrilutamide has not consistently replicated at Phase III.

Promising potential: Moderate. Despite mixed Phase III results, Kintor continues development and regulatory submission. If approved, pyrilutamide would be the first topical antiandrogen registered for AGA.

Side-effect profile: Favorable. Topical application limits systemic exposure. No sexual adverse events reported in clinical trials. Local application-site reactions are similar to those observed with GT20029 [28].

Overall validation: Moderate. Phase III data have been inconsistent. The compound is closer to potential registration than GT20029 but has a less robust efficacy signal.

Comparison to GT20029: Both compounds are developed by Kintor and share the topical route and the absence of sexual adverse events. GT20029 offers the mechanistic advantage of AR protein degradation (which prevents compensatory receptor upregulation) versus competitive antagonism. The GT20029 Phase II efficacy signal is statistically stronger than the pyrilutamide Phase III signal, though cross-trial comparison is limited. GT20029 is earlier in development (Phase II/III) than pyrilutamide (Phase III/NDA submission).

12.4 Bavdegalutamide (ARV-110, oral, for prostate cancer)

Mechanism: Oral PROTAC AR degrader. Recruits CRBN E3 ligase to degrade AR protein, the same fundamental mechanism as GT20029 but formulated for oral systemic administration and developed for metastatic castration-resistant prostate cancer (mCRPC) rather than dermatological indications [20, 21]. The recommended Phase 2 dose is 420 mg oral once daily.

Novelty: High. Bavdegalutamide is the first oral AR PROTAC to enter clinical trials (Phase I/II by Arvinas Inc.). It established clinical proof of concept that PROTAC-mediated AR degradation is achievable and therapeutically active in humans.

Effect size: In the prostate cancer context, bavdegalutamide demonstrated a 46% PSA50 response rate in molecularly defined tumors (AR T878X/H875Y mutations) with durable RECIST responses [20, 21]. The compound is not being developed for AGA or acne and has not been evaluated for hair growth or sebaceous gland endpoints.

Promising potential: Moderate for prostate cancer (Phase I/II, ongoing). Not applicable for AGA/acne, as the oral systemic route would produce the systemic androgen pathway suppression effects (sexual adverse events, hormonal disruption) that topical GT20029 is designed to avoid.

Side-effect profile: Manageable in the oncology context. Most treatment-related adverse events were Grade 1/2, with no Grade 4 or higher events reported at the recommended dose in 138 patients [20]. However, systemic AR degradation would be expected to produce sexual and endocrine adverse events in the non-oncology population.

Overall validation: Moderate. The compound has demonstrated clinical activity in a defined molecular subpopulation of mCRPC patients and has established human proof of concept for oral AR PROTAC pharmacology.

Comparison to GT20029: Bavdegalutamide and GT20029 share the PROTAC AR degradation mechanism but differ fundamentally in route, indication, and therapeutic context. GT20029's topical approach confines AR degradation to the target tissue, avoiding the systemic AR degradation that would be necessary (and acceptable) in mCRPC but undesirable in AGA. Bavdegalutamide provides the most direct clinical validation that PROTAC-mediated AR degradation is achievable in humans, which supports the mechanistic plausibility of GT20029.

12.5 AH-001 (topical PROTAC AR degrader, for AGA)

Mechanism: Topical AR degrader designed using AI-assisted drug discovery by AnHorn Medicines Co. Ltd. (Taiwan). The compound targets AR for degradation through a PROTAC or molecular glue mechanism (specific details not fully disclosed) [29].

Novelty: High. AH-001 is the second topical AR PROTAC/degrader to enter clinical trials, after GT20029. It was designed using generative AI for drug discovery, adding a computational design dimension.

Effect size: Not yet evaluable. The Phase I trial (NCT06927960) in the United States was completed in 2025, with safety and tolerability confirmed at concentrations of 0.2%, 0.5%, 1%, and 2% [29]. No efficacy data (hair count, hair width) have been reported.

Promising potential: Moderate. The compound has completed Phase I with a favorable safety profile. Phase II trials are anticipated but have not been publicly registered as of the monograph date.

Side-effect profile: Favorable in Phase I. No drug-related adverse events were reported across all dose levels [29]. Systemic exposure data have not been publicly reported in detail.

Overall validation: Low. Phase I safety data only. No efficacy data are available.

Comparison to GT20029: AH-001 is a direct mechanistic competitor in the topical AR degrader space. GT20029 has a substantial development lead: completed Phase I and Phase II trials, established efficacy signals in both AGA and acne, and advanced to Phase III. AH-001 has completed only Phase I. The AI-assisted design approach of AH-001 is scientifically interesting but has not yet translated into clinical efficacy demonstration.

12.6 Comparator summary

The five comparators represent the principal pharmacological approaches to androgen-mediated dermatological conditions. **Finasteride** and **dutasteride** are the established systemic reference standards with robust long-term efficacy data, limited by sexual adverse events that reduce patient adherence. **Pyrilutamide** is the nearest-class topical competitor from the same sponsor, with a competitive antagonist mechanism that is mechanistically inferior to degradation (receptor stabilization versus elimination) and with inconsistent Phase III results. **Bavdegalutamide** provides the most direct human proof of concept for PROTAC AR degradation but is an oral oncology compound with no applicability to the dermatological indication. **AH-001** is the closest mechanistic competitor (topical AR degrader) but is substantially earlier in development with no efficacy data.

GT20029 occupies a unique position in this landscape: it is the only topical AR degrader with demonstrated clinical efficacy in Phase II trials for both AGA and acne, the only PROTAC with confirmed negligible systemic exposure, and the only AR-targeting AGA compound with both statistically significant hair count increases and a complete absence of sexual adverse events. The principal limitations are the early stage of development relative to finasteride (which has decades of post-mar-

keting experience), the undisclosed chemical structure (which prevents independent research), and the absence of long-term safety and efficacy data beyond 12 weeks.

The PROTAC degradation mechanism provides a theoretical advantage over both 5-alpha-reductase inhibition (which reduces ligand but preserves the receptor) and competitive antagonism (which blocks the receptor but stabilizes and upregulates it). Whether this mechanistic advantage translates to clinically superior long-term outcomes will be determined by Phase III trials and post-marketing surveillance.

13. References

1. Sakamoto KM, Kim KB, Kumagai A, Mercurio F, Crews CM, Deshaies RJ. Protacs: chimeric molecules that target proteins to the Skp1-Cullin-F box complex for ubiquitination and degradation. *Proc Natl Acad Sci U S A*. 2001;98(15):8554-8559.
2. Kintor Pharmaceutical Ltd. GT20029 IND for PROTAC AR Degradar Was Accepted by NMPA. PR Newswire. February 2, 2021.
3. Kintor Pharma. Positive Top-line U.S. Phase I Trial Results of GT20029, the World's First Topical Use PROTAC Compound. PR Newswire. February 14, 2023.
4. Kintor Pharmaceutical Ltd. GT20029 China Phase II Trial For AGA Reached Primary Endpoint. PR Newswire. April 18, 2024.
5. Yang Q, et al. Efficacy and safety of topical GT20029 in male patients with androgenetic alopecia: a multicenter, randomized, double-blind, placebo-controlled phase 2 study. *J Dermatolog Treat*. 2025;36(1):2574304.
6. Kintor Pharmaceutical Ltd. Phase II Clinical Trial Of GT20029 Gel For The Treatment Of Acne In China Reached Primary Endpoint. Kintor corporate announcement. August 12, 2025.
7. GT-20029. Wikipedia. Available at: <https://en.wikipedia.org/wiki/GT-20029>. Accessed May 2026.
8. Kintor Pharmaceutical Ltd. TD-106 CRBN binder scaffold and AR PROTAC series (TD-802/33c). Patent analysis and corporate disclosures. Data referenced via Synapse drug intelligence platform and Follicle Thought reporting.
9. Kaufman KD. Androgens and alopecia. *Mol Cell Endocrinol*. 2002;198(1-2):89-95.
10. Randall VA. Androgens and hair growth. *Dermatol Ther*. 2008;21(5):314-328.
11. Neklesa TK, Winkler JD, Crews CM. Targeted protein degradation by PROTACs. *Pharmacol Ther*. 2017;174:138-144.

12. Ponnusamy S, He Y, Hwang DJ, et al. Orally bioavailable androgen receptor degrader, potential next-generation therapeutic for enzalutamide-resistant prostate cancer. *Clin Cancer Res.* 2019;25(22):6764-6780.
13. Pettersson M, Crews CM. PROteolysis TARgeting Chimeras (PROTACs): past, present and future. *Drug Discov Today Technol.* 2019;31:15-27.
14. Kintor Pharmaceutical Ltd. GT20029 IND Was Accepted by NMPA. Kintor corporate announcement. February 2021.
15. Kintor Pharmaceutical Ltd. IND Clearance by the U.S. FDA for GT20029 to Treat Androgenetic Alopecia and Acne. PR Newswire. July 2021.
16. Kintor Pharmaceutical Ltd. Completion of Phase I Trial of the World's First PROTAC Compound (GT20029) for Topical Use in China. Kintor corporate announcement. November 2022.
17. Kintor Pharmaceutical Ltd. Completion of Subject Enrollment and Dosing in Phase I Clinical Trial of AR-PROTAC (GT20029) in the US. PR Newswire. October 2022.
18. Kintor Pharma Advances to Phase III with GT20029, Pioneering Topical Treatment for Male Androgenetic Alopecia. BioPharma APAC. 2024.
19. ClinicalTrials.gov. To Evaluate the Efficacy and Safety of GT20029 Solution in the Treatment of Androgenetic Alopecia (AGA) in Chinese Adult Males. NCT06692465.
20. Arvinas Inc. Bavdegalutamide (ARV-110) Continues to Demonstrate Clinical Benefit in Men with Metastatic Castration-Resistant Prostate Cancer. ASCO GU 2022.
21. Snyder LB, Flanagan JJ, Qian Y, et al. Preclinical Evaluation of Bavdegalutamide (ARV-110), a Novel PROteolysis TARgeting Chimera Androgen Receptor Degradar. *Mol Cancer Ther.* 2025;24(4):511-523.
22. Deshaies RJ. Protein degradation: prime time for PROTACs. *Nat Chem Biol.* 2015;11(9):634-635.
23. Zouboulis CC. Acne and sebaceous gland function. *Clin Dermatol.* 2004;22(5):360-366.
24. Kaufman KD, Olsen EA, Whiting D, et al. Finasteride in the treatment of men with androgenetic alopecia. *J Am Acad Dermatol.* 1998;39(4 Pt 1):578-589.

25. Rossi A, Cantisani C, Melis L, Iorio A, Scali E, Calvieri S. Minoxidil use in dermatology, side effects and recent patents. *Recent Pat Inflamm Allergy Drug Discov.* 2012;6(2):130-136.
26. Traish AM. Post-finasteride syndrome: a surmountable challenge for clinicians. *Fertil Steril.* 2020;113(1):21-50.
27. Olsen EA, Hordinsky M, Whiting D, et al. The importance of dual 5alpha-reductase inhibition in the treatment of male pattern hair loss: results of a randomized placebo-controlled study of dutasteride versus finasteride. *J Am Acad Dermatol.* 2006;55(6):1014-1023.
28. Kintor Pharmaceutical Ltd. KX-826 and GT20029 Phase III Trial Updates. Kintor corporate communications and press releases. 2025-2026.
29. AnHorn Medicines Co. Ltd. AI-Designed Drug AH-001 Complete U.S. Phase I Trial, Demonstrating Excellent Safety and Tolerability. AnHorn corporate announcement. 2025.

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