



Review Article

Acne Relapse Under Persistent Exogenous Androgen Exposure: A Practical Management Algorithm

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Abstract

Introduction: Androgens play a central role in acne pathogenesis through androgen receptor-mediated stimulation of sebaceous gland activity. Long-term androgen administration is associated with a high risk of acne recurrence, even after successful treatment. This work proposes practical recommendations for the prevention and management of acne relapse in patients receiving long-term exogenous androgens, including female-to-male transgender individuals (transmen) and recreational athletes using anabolic-androgenic steroids. **Methods:** A narrative literature review of seven articles published between 2000 and 2026 was conducted using PubMed and Google Scholar. Search terms combined acne, androgen exposure, transgender individuals, bodybuilding, relapse, prevention, and management. Clinical characteristics, therapeutic approaches, and expert experience were synthesized to develop management recommendations. **Results:** The literature provides very limited data on acne relapse, its prevention, and management in individuals receiving long-term exogenous androgens, highlighting a significant knowledge gap. In patients who cannot or do not wish to discontinue androgen therapy, clinicians should consider the persistent androgen-driven stimulation of the pilosebaceous unit and its effects on sebaceous activity, follicular physiology, and the skin microbiome. This ongoing pathogenic stimulus may influence therapeutic choices and the long-term use of treatments such as spironolactone and isotretinoin. **Conclusion:** Exogenous androgen-driven acne relapse represents a distinct clinical entity requiring individualized, multidisciplinary management. Effective prevention and treatment strategies should address the persistent hormonal environment while considering both clinical and psychosocial consequences.

Keywords: Acne; Anabolic drugs; Steroids; Hormones; Testosterone; Oestrogen; Transgender.

Introduction

Androgens play a central role in the development of acne vulgaris (acne). Testosterone and its more potent metabolite, dihydrotestosterone (DHT), bind to androgen receptors in sebocytes [1,2]. They activate sebaceous gland activity, inducing a significant increase of sebum production with a modification of the composition of sebum [3]. Sebaceous gland hyperplasia, under androgenic control and via the activation of lipogenic pathways (SREBP-1, PPAR γ), leads to hyperseborrhoea associated with a qualitative alteration of sebum (increased triglycerides, relative decrease in linoleic acid, oxidation of squalene) [4,5]. This lipid alteration disrupts keratinocytes differentiation in the follicular canal by promoting hyperkeratinisation and impairment of the epithelial barrier (tight junctions, abnormal cornification) and in addition proliferation of keratinocytes of the epithelial barrier of the sebaceous gland. This ultimately results in an obstruction

of the sebaceous gland canal and the development of retentional lesions. The follicular microenvironment becomes more anaerobic and rich in lipid substrates, promoting follicular dysbiosis and the production of bacterial lipases, which generate pro-inflammatory free fatty acids. Concurrently, oxidized lipids (peroxidized squalene) activate keratinocytes and immune cells via TLR2 and the inflammasome (NLRP3), amplifying the local inflammatory response [6]. These androgen-induced changes in sebum production contribute to follicular dysbiosis, characterized by a reduction in the diversity of the six major *Cutibacterium acnes* (C. acnes) phylotypes and the predominance of phylotype IA1. Alterations in sebum composition also favour the emergence of a more virulent C. acnes phenotype, associated with the release of pro-inflammatory antimicrobial peptides and extracellular vesicles. This virulent phenotype appears to be driven, at least in part, by bacteriophage-mediated acquisition of virulence-associated genes in phylotype IA1. Together, these microbial and host interactions promote persistent activation of innate immunity and contribute to the chronic inflammatory process characteristic of acne [7,8].

Figure 1 summarises the role of androgens in the physiopathology of acne.

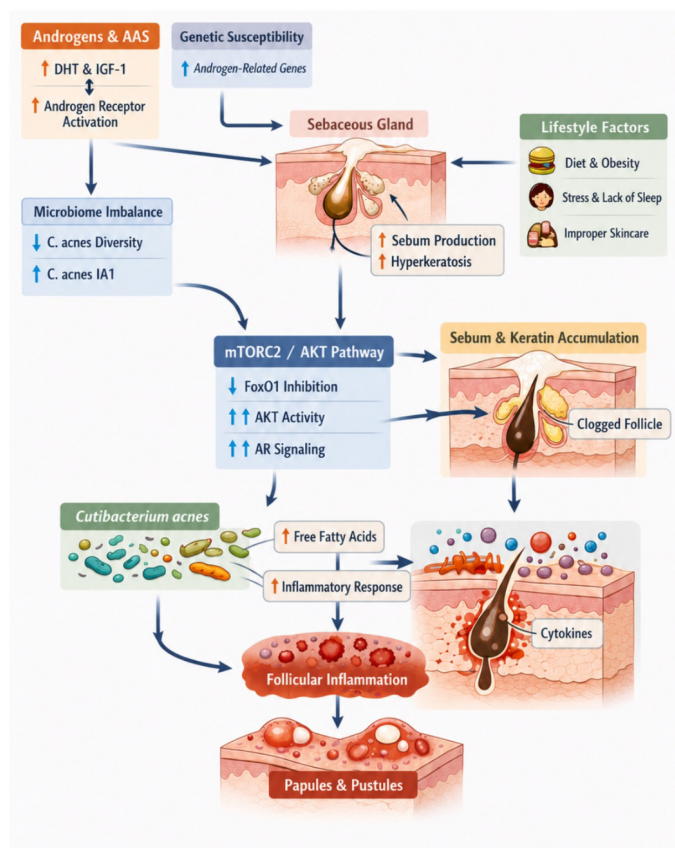


Figure 1: Physiopathology of acne with a focus on increased androgen levels.

In contrast to androgens, oestrogens generally exert anti-acne effects by suppressing ovarian androgen production and increasing hepatic synthesis of sex hormone-binding globulin (SHBG), thereby reducing circulating free testosterone [9,10].

The interplay between androgens, oestrogens, and progesterone underscores the importance of hormonal regulation in acne and provides the rationale for endocrine-based therapeutic strategies. On a genetic level, although 60% of acne patients have a familial factor. Certain androgen-related genes were characterised as being risk factors of acne, especially for severe acne, although support for these associations is not unanimous [11].

Other internal and external factors such as obesity, high-sugar, high-fat, or dairy-rich diets, improper skincare, comedogenic makeup, sun exposure, stress, and lack of sleep may trigger or exacerbate the condition [12]. Moreover, sweat, pressure, friction, and skin occlusion may contribute to acne exacerbation especially on the trunk [13]. Over recent years, dermatologists have increasingly encountered acne patients who, for therapeutic or personal reasons, cannot or do not wish to discontinue androgen therapy. These individuals are at high risk of acne relapse following treatment discontinuation. Key populations include transgender men receiving gender-affirming testosterone therapy and such as bodybuilders using androgenic-anabolic steroids (AAS) to enhance muscle mass and athletic performance. In both groups, continued androgen exposure complicates the long-term management of acne.

This article aims to provide treatment suggestions for maintaining therapeutic outcomes (relapse prevention or relapse treatment) in patients requiring long-term androgen therapy, based on the literature and the experience of a group of experts in the field of acne.

Material and Methods

In a first step, the authors performed a narrative literature

research of articles published between 2000 and 2026 to identify articles proposing therapeutic management strategies for long-term patients on androgens, limiting this research to transmen (women-to-men) and individuals (bodybuilders) performing intensive athletic and recreational performance training. Search key words included acne (main key word) combined with: long-term hormonal treatment, long-term hormonal supplementation, testosterone, anabolic steroids, progesterone, hormone therapy-induced cutaneous side effects, transgender, athletes, bodybuilding, dehydroepiandrosterone (DHEA), anabolic-androgenic steroids combined with relapse and prevention of relapse. Literature sources included reviews, clinical studies and case reports.

In a 2nd step, based on the results from the identified literature, the experts selected the information contained in 27 articles and analysed data from seven publications pertinent to the aim of this manuscript. They further discussed the best adapted global preventive and management approach for acne relapse in these two populations. The experts considered the specific therapeutic constraints: impossibility to stop testosterone therapy in transmen and potential non-willingness of individuals performing intensive athletic and recreational performance training and modulated the results of the analysis with their own experience.

In a last step, the experts developed recommendations for prevention of acne relapse management in these patients receiving exogenous androgens.

Results

From the seven identified articles, six were about testosterone treatment in transmen and one about AAS administration in athletes and bodybuilders. Table 1 provides a tabulated summary of these publications. None of these seven publications provided specific data about acne relapse.

Authors	Study design, number of subjects and treatment period and follow-up	Acne incidence and severity after initiation of testosterone or AAS	Acne relapse
Testosterone-induced acne			
Wierckx et al.[16]	prospective interventional study in 20 hormone naive transmen and a cross-sectional study in 50 transmen with an average of 10 years on T therapy	At baseline, 35% of the subjects had facial acne and 15% had acne on the back and/or chest. After 6 months of T administration, facial acne was present in 82.4% of subjects and in 88.2% of subjects on the back and/or chest. After 12 months of androgen therapy, facial acne was still present in 55% of participants and 50% of participants on the back and/or chest. The presence of acne as well as the severity increased during T administration. The majority of trans men had mild acne lesions, 20% had moderate acne lesions, and none had severe or very severe acne lesions.	None reported
Cocchetti et al. [17]	multicenter prospective study. 291 transmen were evaluated at baseline (T0), 6 (T1) and 12 months (T2)	Significant increase acne severity during T treatment from baseline after 6 months, with no significant increase after 12 months. After six months, 4.2% and 18.3% of participants reported, respectively, severe and moderate acne.	None reported
Chu et al. [18]	Retrospective patient record analysis of 60 transmen, 12 months and 12 months follow-up	Of 60 patients, 46 (77%) did not have baseline acne. 25 of those 46 patients developed acne (54%) within 1-year of T initiation. Overall incidence proportion was 70% at 2-years; patients who used progestin prior to or during follow up were more likely to develop acne than non-users (92 % vs 33%, $p < 0.001$).	None reported
Park et al. [19]	Systematic medical chart record of 69 transmen treated for up to 24 years follow-up	Acne developed in 9% of transmen after 3 months and increased to 18% after 6 months. After 24 months 38% of the patients had developed acne.	None reported
Motosko et al. [20]	retrospective and prospective cross-sectional analysis of testosterone on acne on the chest and abdomens of 90 transmen, treatment duration not indicated	Exogenous testosterone showed significant effects on the incidence of acne in transgender men. Acne was present on the chest of 52% of transmen taking testosterone	None reported
Smith et al. [21]	retrospective matched cohort study of electronic health record data of 11234 transmen with up to 5 years of follow-up. Individuals had no baseline acne	Transmen had a fourfold higher risk of acne than cisgender men and a 1.5-fold higher risk than cisgender women, with the risk peaking during the first year following testosterone initiation and remaining elevated throughout the subsequent 5 years.	None reported
AAS-induced acne			
Smit [23]	12 months prospective observational study with a systematic approach in 111 men having AAS administrated	Observed increase of acne compared to baseline was 28% after 12 months	None reported

Table 1: Summary table of analysed studies conducted in transmen and athletes receiving AAS; AAS: Anabolic-Androgenic Steroids; T: Testosterone.

• Transmen

Testosterone therapy in transmen may worsen existing acne or cause acne relapse due to its androgenic effects on the skin [14,15].

Three different studies observed a significant rise in acne prevalence after initiating testosterone therapy, with reported rates ranging from about 30% to over 60%, depending on study design and follow-up duration [16-18]. In a cross-sectional study in 50 transmen with an average of 10 years of testosterone therapy, persistent acne symptoms were reported in 70% of participants [19]. Another

recent study including 55 transmen receiving testosterone therapy for at least two years found a 38% incidence of new-onset acne. The occurrence of acne was associated with serum testosterone levels exceeding 630 ng/dL. Acne on the back was frequent (52%) and tended to be more severe than facial acne, reflecting the typical distribution pattern observed in transmen [20]. A recent retrospective study involving 11234 transmen showed that these subjects had a 4 times higher risk of acne than cisgender men and a 1.5-fold higher risk than cisgender women, with the risk peaking during the first year following testosterone initiation and remaining elevated throughout the subsequent 5 years. However, the article did not provide any information about acne relapse [21].

- **Bodybuilders receiving AAS**

AAS refer to male sex anabolic steroid hormones including testosterone, their synthetic derivatives, and their biologically active metabolites. Although these compounds have established therapeutic indications, they are widely recognized for their non-therapeutic misuse among healthy individuals [22].

The use of AAS as performance-enhancing agents has become highly prevalent by bodybuilders. Its non-medical use is increasing, especially among adolescents and women and has emerged as a significant cause of acne and other drug-induced (iatrogenic) health complications [23].

The risk and severity of acne relapse are AAS dose-dependent and correlate with the androgenic potency of the compound used [24]. Supraphysiologic doses, frequently reported in non-medical AAS use, significantly increase the likelihood of severe acne onset and relapse and may also lead to scarring and severe ulcerated acne lesions [25-28].

Expert opinion on strategies for preventing and managing acne relapse in patients receiving long-term exogenous androgen therapy

- **Patient questioning**

Prior to initiating treatment, dermatologists should obtain a detailed history of exogenous androgen use, including the duration of therapy and whether androgen administration is expected to continue long term (at least two years). The number of acne

relapses following treatment discontinuation, the interval between treatment withdrawal and relapse, the duration and localization of acne flares, and all previously received acne treatments should also be systematically assessed.

If the patient does not report any previous acne, current treatment recommendations for transmen and acne may be considered [29-31]. Should the patient report previous acne episodes, the experts recommend the following pathway.

- **Severity assessment**

Prior to any treatment, the experts suggest to assess the severity of acne relapse using currently available grading tools such as the GEA (Global evaluation of Acne) scale or AFAST (Adult Female Acne Severity Tool) for transmen and TRASS (Truncal Acne Severity Scale) in patients with truncal acne relapse should always be used [32-34].

- **Proposed prevention and relapse management recommendations**

Only very sparse treatment and prevention of relapse recommendations for exogenous androgen-induced acne relapse exist for transmen patients and no specific treatment recommendations at all for AAS-driven acne exist [29]. For these reasons and due to their urgent need, the experts propose the following preventive and therapeutic management recommendations for this type of acne relapse.

- **Relapse phenotypes**

Treatment of exogenous androgen-driven acne relapse should always be dynamically adjusted according to the type and intensity of relapse, with the aim of restoring follicular homeostasis while minimizing cumulative toxicity and avoiding microbiome disruption. Three main relapse phenotypes can be distinguished, retentional, inflammatory, and mixed relapse, each reflecting distinct pathophysiological imbalances within the pilosebaceous unit.

To this end, the experts propose an algorithm presented in Table 2 that provides a treatment algorithm focusing on exogenous androgen-driven acne relapse.

	Acne severity Spironolactone ***		
Phenotype	Mild <i>(few scattered lesions, no scarring)</i>	Moderate <i>(numerous lesions, possibility of scars)</i>	Severe <i>(numerous lesions, nodules and scars)</i>
Initial relapse therapy			
Retentional acne (open and closed comedones, microcysts)	<ul style="list-style-type: none">topical retinoid	<ul style="list-style-type: none">topical retinoid orclascoterone*+ keratolytics ± low-dose isotretinoin**	<ul style="list-style-type: none">systemic isotretinoin** + hormonal re-assessment
Inflammatory acne (Papules, pustules, nodules)	<ul style="list-style-type: none">fixed combination: topical retinoid + BPO	<ul style="list-style-type: none">topical retinoid orclascoterone* + keratolytics ± low-dose isotretinoin**Spironolactone ***	<ul style="list-style-type: none">systemic isotretinoin** + administration of oral antibiotics (<3 months)
Mixed acne (Comedones, microcysts, papules, pustules, nodules)	<ul style="list-style-type: none">fixed combination: topical retinoid + BPO	<ul style="list-style-type: none">fixed combination orclascoterone*+ phenotype-driven systemic therapySpironolactone ***	<ul style="list-style-type: none">systemic isotretinoin** + endocrine re-assessment
Relapse prevention therapy	Long-term administration of topical retinoids		
Adjuvant measures	<ul style="list-style-type: none">Control of hormonal factors (AAS exposure, testosterone therapy), diet (high glycaemic load, IGF-1, supplements), psychological stress and environment (occlusion, sweating, UV, pollution)Choice of acne-specific dermatocosmetic care to restore damaged skin barrierAvoidance of comedogenic skin care products		
Follow-up	Re-assessment of acne severity every 3–6 months; adjustment of therapy according to relapse pattern		

Table 2: Treatment recommendations for exogenous androgen-driven acne relapse and its prevention; Topical retinoids: adapalene, tretinoin, trifarotene), fixed combination: adapalene + benzoyl peroxide, *: where available, **in transmen in combination with reliable contraception (3rd or 4th generation contraceptive pills),***Spironolactone may be given off-labelling to transmen with moderate acne.

• Retentional acne relapse (microcomedonal/comedonal predominance)

Retentional acne relapse primarily reflects defective keratinocyte differentiation and impaired desquamation within the follicular infundibulum, often in a context of persistent sebaceous activity driven by hormonal stimulation. The cornerstone of management is the optimization of topical retinoid therapy, with escalation in frequency or potency (e.g., adapalene to tretinoin, or trifarotene in selected cases) [30]. Topical retinoids restore the follicular epithelial turnover, reduce microcomedone formation, and exert indirect anti-inflammatory effects.

Adjunctive use of keratolytic agents such as salicylic acid or low-concentration alpha-hydroxy acids may be considered in mild cases to enhance corneocyte shedding, although their use should remain controlled to avoid barrier disruption.

For mild retentional relapse, topical optimization alone is generally sufficient. In moderate cases, short courses of oral isotretinoin at low dose may be considered to re-establish long-term control, particularly in patients with recurrent microcomedonal cycling if no contraindication [30].

Dermocosmetic care should always be considered as a therapeutic component rather than an adjunct [35]. Daily hygiene must rely on mild cleansing agents that preserve the skin barrier and maintain physiological pH, thereby avoiding disruption of the resident microbiota. Overly aggressive cleansing or inappropriate topical products may induce barrier dysfunction and promote compensatory sebaceous activity. The use of non-comedogenic emollients is critical to maintain epidermal integrity and ensure tolerance to long-term topical treatments.

- **Inflammatory acne relapse (papulopustular or nodular predominance)**

Inflammatory acne relapse reflects activation of innate immune pathways, often associated with microbial imbalance and increased production of pro-inflammatory lipids (e.g., oxidized squalene, free fatty acids). First-line management relies on topical anti-inflammatory and antimicrobial agents, notably benzoyl peroxide (BPO), either alone or in combination with a topical retinoid. BPO provides rapid reduction in *Cutibacterium acnes* density without inducing bacterial resistance and contributes to limiting biofilm formation [30]. The use of topical antibiotics should not be considered.

Moreover, in patients with mild inflammatory acne relapse, clascoterone (where available), systemic zinc (especially during summer) or topical azelaic acid may be prescribed [36].

In patients under AAS administration, inflammatory relapse may indicate instability in sebaceous lipid composition rather than quantity alone, reinforcing the rationale for combining anti-inflammatory and comedolytic approaches. For more severe or nodular relapse, escalation to systemic isotretinoin (low to intermediate cumulative dosing strategies) may be necessary, even in maintenance settings, particularly when inflammatory lesions are recurrent or scarring (always if no contraindication) [30].

- **Mixed acne relapse (both retentional and inflammatory lesions)**

Mixed acne relapse represents the most common clinical type of acne relapse and reflects the coexistence of altered keratinization, sebaceous dysregulation, and immune activation. Management requires combination therapy targeting all pathogenic components simultaneously. A fixed regimen combining a topical retinoid with benzoyl peroxide constitutes the backbone of treatment. This dual approach addresses both microcomedone formation and inflammatory amplification [30].

In moderate forms of relapse, systemic antibiotics, generally cyclins may be introduced depending on the predominant component. Low-dose isotretinoin should be given for significant retentional burden or recurrent disease while short courses of oral

antibiotics should be prescribed for inflammatory flares, with strict duration control.

Low-dose isotretinoin maintenance therapy (0.1-0.2 mg/kg) represents a particularly rational option in transmen with recurrent acne relapse despite optimized topical treatment. Beyond its well-established efficacy in reducing acne, isotretinoin directly targets several androgen-dependent pathogenic mechanisms, including sebaceous gland hypertrophy, excessive sebum production, follicular hyperkeratinisation, and the creation of a microenvironment favorable to *C. acnes*-pro inflammatory profile [37].

Although specific studies evaluating long-term low-dose isotretinoin in exogenous androgen-driven acne are currently lacking, the experts consider that continuous or intermittent low-dose isotretinoin could be a valuable maintenance strategy in patients with persistent androgen exposure, particularly in those with repeated relapses, truncal involvement, or a tendency toward scarring. In such situations, the objective is not necessarily to achieve definitive cure but rather to maintain long-term control of a chronic androgen-driven disease process while minimizing disease burden and preventing irreversible sequelae.

Mixed acne relapse frequently reflects partial endocrine control, necessitating reassessment of hormonal balance (dose, molecule, adherence), especially in the context of transmen or AAS exposure.

- **Stratification according to acne relapse intensity**

Management of mild acne relapse should consist in optimisation of topical therapy (retinoid ± BPO), reinforcement of dermatocosmetic as well as exposome measures. Moderate acne relapse should be managed in adding systemic agents such as antibiotics or low-dose isotretinoin, depending on severity of relapse. Severe or recurrent acne relapse should profit of a full-course of isotretinoin or the adjustment of the hormonal therapy in mandatory collaboration with the patient's endocrinologist as isotretinoin may impact serum lipid levels and liver enzymes [38].

- **Long-term holistic and integrative relapse preventing strategy**

The long-term management of exogenous androgen-driven acne to prevent relapse relies not only on lesions suppression but also on the stabilisation of the sebaceous gland-follicular-epithelium-microbiome axis.

To this end, maintenance therapy after a curative course should always ensure persistent normalisation of keratinocyte differentiation within the follicular canal and control of sebaceous gland activity. Topical retinoids remain the cornerstone of this approach due to their comedolytic and anti-inflammatory properties, as well as their ability to restore epithelial homeostasis.

Adjunctive agents such as azelaic acid may contribute to microbial regulation and modulation of subclinical inflammation without inducing microbial resistance.

In 2026; the EuroGuiDerm evidence-based guideline recommend in women the administration of off-labelling use of Spironolactone as an alternative to androgenic contraceptives [30]. Therefore, the experts consider that Spironolactone may be administered in selected transmen. However, when considered clinically appropriate by the dermatologist and after discussion with the patient, referral to an endocrinologist may be necessary, particularly in cases where acne severity outweighs concerns regarding the partial anti-androgenic effects associated with treatment. In addition the expert suggests to combine acne treatment with a holistic approach that specifically addresses the following factors. Nutritional factors play a critical role, particularly through modulation of insulin/IGF-1 signalling pathways and sebaceous lipogenesis. Diets characterized by high glycaemic load or excessive intake of certain amino acids (notably leucine-rich protein supplements). Nutritional supplements especially proteins containing leucines may trigger or worsen acne [39-41]. Leucine stimulates the production of IGF-1 [39-43].

Therefore, they should be carefully regulated. Conversely, dietary patterns enriched in omega-3 fatty acids may exert anti-inflammatory effects and contribute to improved disease stability [44].

Psychophysiological stress represents another key modulator, acting through neuroendocrine pathways that influence sebaceous gland function and cutaneous immune responses [45]. Chronic stress may exacerbate inflammatory signalling and alter the skin microbiome, justifying the integration of stress-reduction strategies within the long-term management plan.

Environmental and occupational exposures should always be considered as well [46]. Individuals engaged in outdoor activities or high-intensity physical training are subjected to mechanical occlusion, sweating, ultraviolet radiation, and pollution, all of which can alter follicular homeostasis and promote dysbiosis. Appropriate measures, including rapid post-exercise hygiene, use of non-occlusive protective formulations, and photoprotection, are essential to mitigate these effects. Moreover, the patient should be advised on lifestyle, diet, and quality of life improves adherence, psychological well-being and adapted management perspectives should be proposed and initiated and avoid chest binding [47].

Finally, microbiome preservation is a central objective. The prolonged use of topical or systemic antibiotics should be avoided whenever possible due to their impact on microbial diversity and the risk of selecting pro-inflammatory strains. Maintenance strategies should instead favour management approaches that support a balanced microbial ecosystem and limit inflammatory

amplification. Microbiome preservation may be obtained in using specifically adapted dermocosmetics [35].

Discussion

The present work addresses a clinically under recognised challenge: the management and prevention of acne relapse in patients who receive long-term exogenous androgen therapy and for whom discontinuation is either medically contraindicated or personally unacceptable. Our narrative literature review identified only seven relevant publications, none of which specifically reported on acne relapse as a primary endpoint [16-21,23]. This finding is itself significant. Unlike conventional post-isotretinoin relapse, which typically occurs after a finite course of treatment in the absence of ongoing hormonal drive, exogenous androgen-induced relapse reflects a continuous pathogenic stimulus that cannot be eliminated. This fundamental distinction justifies a separate therapeutic algorithm, as proposed in the present article.

The six studies conducted in transmen consistently demonstrated a significant increase in acne incidence following testosterone initiation, with rates ranging from 38% to over 80% depending on follow-up duration [16-21]. Truncal involvement was notably frequent, reflecting the androgenic distribution pattern typically observed in male-pattern acne. Importantly, none of these studies prospectively evaluated relapse after therapeutic response, confirming that the clinical management of recurrence in this population remains largely empirical. The only large-scale data available (Smith et al. n = 11,234) confirms that the risk remains elevated throughout at least five years of testosterone therapy, emphasising the need for long-term, adaptive management strategies rather than short-term interventions [23]. Evidence in bodybuilders and athletes using anabolic-androgenic steroids (AAS) is even more limited, with only one prospective observational study identified. This population presents additional challenges: AAS use is frequently illicit and undisclosed, doses are often supraphysiological, and combination regimens are common. Furthermore, the androgenic potency of compounds used varies widely across AAS classes, which directly influences the severity and pattern of acne. These factors make evidence-based recommendations particularly difficult to formulate, and the expert recommendations proposed herein should be considered provisional pending dedicated prospective studies in this population.

Several limitations of the present work must be acknowledged. First, the evidence base identified through our literature review is extremely limited. Only seven publications were deemed relevant to the clinical question, and none specifically addressed acne relapse as a primary outcome. The absence of randomised controlled trials in these two patient populations underlines the fact

that all therapeutic recommendations presented herein are based on expert opinion only and on indirect evidence from observational studies, corresponding to a low level of evidence (Grade C-D). Second, both populations are clinically and socially distinct and cannot be treated as a homogeneous group. Therapeutic contexts differ substantially: transgender men receive testosterone under medical supervision as part of a gender-affirming care pathway, whereas AAS use in bodybuilders is typically self-administered, frequently at supraphysiological doses, and may involve multiple compounds simultaneously. Finally, the psychosocial and gender-identity dimensions of acne management in transgender patients introduce additional therapeutic complexity that is currently not fully captured by conventional severity scales. Parameters such as quality of life, treatment acceptability relative to gender-affirming care, and multidisciplinary coordination represent important outcome dimensions that future prospective studies in this field should systematically integrate.

In conclusion, long-term management and prevention of exogenous androgen-driven acne relapse require an integrated, interdisciplinary approach that considers pharmacological, hormonal, metabolic, environmental, and behavioural factors. As persistent androgenic signalling and hormonal fluctuations can influence sebaceous activity and treatment response, management should focus on maintaining follicular homeostasis and preventing the re-establishment of a pro-inflammatory, dysbiotic microenvironment. Long-term relapse prevention should prioritize non-antibiotic strategies, sustained normalization of follicular biology, and preservation of microbial diversity to reduce recurrent disease and limit the emergence of resistant microbial communities.

Conflict of interest

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Data availability statement

The herewith presented data is available upon reasonable request from the corresponding author

Ethical statement

Not applicable

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