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THE SARM-DEMIC: SOCIAL MEDIA ALGORITHMS, E-COMMERCE LOOPHOLES, AND THE NORMALIZATION OF EXPERIMENTAL PHARMACOLOGY IN RECREATIONAL USERS

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ABSTRACT

The epidemiology of performance- and image-enhancing drug (PIED) use has shifted toward non-athlete recreational users who primarily seek cosmetic physical changes. This review evaluates the unregulated distribution of Selective Androgen Receptor Modulators (SARMs). We analyze this trend at the intersection of experimental pharmacology, social media algorithms, and e-commerce loopholes. We comprehensively synthesized the literature to contrast the clinical intent of investigational SARMs with the toxicological reality of digital markets. Phase II trials demonstrated that tissue-selective compounds like enobosarm increase lean body mass at controlled micro-doses ranging from 0.1 to 3 mg daily. However, the online market promotes supraphysiological dosing regimens exceeding 20 mg daily. Chemical analyses reveal widespread mislabeling and cross-contamination with unapproved experimental compounds, including PPAR- δ agonists and growth hormone secretagogues. Recreational dosing bypasses tissue selectivity. Case reports document severe lipid dysregulation, hypogonadotropic hypogonadism requiring unmonitored post-cycle therapy, and idiosyncratic drug-induced liver injury (DILI). Histologically, these hepatic injuries predominantly show bland centrilobular canalicular cholestasis, though instances of significant hepatocellular injury have also been documented. SARM misuse is exacerbated by social media, where young adults enter algorithmic echo chambers that amplify muscle dysmorphia and minimize pharmacological risks. Addressing SARM misuse requires an update to traditional anti-doping frameworks, with public health strategies focusing on e-commerce regulation, algorithmic transparency, and targeted diagnostic training for clinicians.

KEYWORDS

Selective Androgen Receptor Modulators, Drug-Induced Liver Injury, Muscle Dysmorphia, Experimental Pharmacology, Social Media Algorithms, E-Commerce

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1. Introduction

1.1. Epidemiological Shifts in PIED Consumption

While gym culture historically associated performance- and image-enhancing drugs (PIEDs) with elite athletics and competitive bodybuilding, the past two decades have seen a demographic shift toward recreational lifters (Pope et al., 2014). Driven by baseline body dissatisfaction and modern aesthetic standards, these users predominantly seek physique improvements rather than athletic achievement. This epidemiological change has been further accelerated by the transition from localized, illicit distribution to global e-commerce platforms. By drastically increasing drug accessibility, online markets have expanded the consumer base to include adolescents and young adults (Brennan et al., 2013), for whom Selective Androgen Receptor Modulators (SARMs) have emerged as particularly sought-after compounds.

1.2. Clinical Development and Mechanism of Action

Pharmaceutical companies developed SARMs in the late 1990s as alternatives to classic androgen therapy (Narayanan et al., 2008; Yin et al., 2003), specifically aiming to counter muscle-wasting conditions such as cancer cachexia (Fearon et al., 2011), age-related sarcopenia, and chronic obstructive pulmonary disease (COPD). While classic anabolic-androgenic steroids (AAS) and testosterone replacement therapies effectively stimulate muscle protein synthesis, their long-term clinical utility is limited by severe side effects. This lack of tissue selectivity in traditional AAS leads to prostate hypertrophy, virilization in women, and cardiovascular events secondary to polycythemia and dyslipidemia (Basaria et al., 2010).

Traditional steroidal androgens possess a rigid four-ring tetracyclic cyclopentanoperhydrophenanthrene structure that makes them susceptible to enzymatic conversion in various tissues. For instance, aromatase (CYP19A1) in adipose tissue converts testosterone to estradiol, leading to gynecomastia. Concurrently, the 5-

alpha reductase enzyme (specifically type 1 in the skin and type 2 in the prostate) converts testosterone into dihydrotestosterone (DHT), which accelerates alopecia and prostatic hyperplasia. Ultimately, these metabolic conversions severely limit the safety profile of classical androgens.

To overcome these limitations, SARMs were engineered to target the androgen receptor (AR) selectively. The AR is a nuclear steroid receptor consisting of an N-terminal domain (NTD) containing the activation function-1 (AF-1), a DNA-binding domain (DBD), and a ligand-binding domain (LBD) containing the activation function-2 (AF-2). SARMs bind directly to the LBD, inducing specific conformational changes in the receptor. Unlike testosterone, which promotes a uniform recruitment of coactivators like Steroid Receptor Coactivator-1 (SRC-1), SARMs induce a distinct folding of the AF-2 region, leading to differential recruitment of coregulators. While SARMs recruit coactivators to initiate transcription in skeletal muscle, they fail to do so in the prostate, where they actively recruit corepressors like Nuclear Receptor Corepressor (NCoR) (Bhasin & Jasuja, 2009; Gao et al., 2005). This mechanism effectively promotes anabolic processes in skeletal muscle and bone while limiting androgenic stimulation in the prostate and sebaceous glands.

Chemically, first-generation SARMs utilize nonsteroidal templates, including aryl-propionamides, quinolinones, and bicyclic hydantoin. Unlike the rigid steroidal framework, nonsteroidal SARMs utilize flexible linkages, such as ether or thioether bonds, which permit unique binding conformations. Furthermore, bulky substitutions at specific functional groups physically prevent the molecule from serving as a substrate for 5-alpha reductase or aromatase. Consequently, these molecules cannot undergo the enzymatic amplification or aromatization required to stimulate prostate tissue or induce gynecomastia directly.

Phase II clinical trials confirmed this tissue selectivity. For instance, Dalton et al. (2011) conducted a 12-week double-blind, placebo-controlled trial, evaluating the SARM GTx-024 (enobosarm) in 120 healthy elderly men and postmenopausal women (mean age 63.3 years; BMI <30). Subjects received daily oral doses of 0.1, 0.3, 1, and 3 mg of GTx-024 or a placebo. The primary endpoint, total lean body mass assessed by dual-energy X-ray absorptiometry (DXA), showed a dose-dependent increase. Notably, the 3-mg dose yielded a statistically significant average increase of 1.3 kg of lean mass compared to placebo, alongside a simultaneous average decrease of 0.6 kg in total fat mass. Subjects also showed significant improvements in physical function, quantified by increased power during a standardized Stair Climb Test (calculated using subject weight, stair rise, and transit time over 12 steps).

1.3. Regulatory Status and Digital Normalization

To date, no SARM has received marketing authorization from either the US Food and Drug Administration (FDA) or the European Medicines Agency (EMA), with the FDA issuing explicit clinical warnings regarding the risks of hepatotoxicity and cardiovascular events associated with these substances (U.S. Food and Drug Administration, 2023). Furthermore, SARMs are prohibited at all times by the World Anti-Doping Agency (2024) under class S1.2, requiring advanced mass spectrometry protocols for doping control analysis (Thevis & Schänzer, 2018).

Despite regulations, e-commerce vendors exploit legal loopholes to distribute SARMs. Often marketed as "research chemicals" and labeled "not for human consumption", these products evade regulatory oversight and consumer safety laws (Van Wagoner et al., 2017). Furthermore, this illicit network exploits psychological vulnerabilities. While earlier psychiatric studies linked muscle dysmorphia primarily with classical AAS (Pope et al., 2014), modern video-sharing platforms have normalized SARM consumption. Recommendation algorithms guide users into fitness-centric echo chambers (Cinelli et al., 2021), where uncredentialed content creators minimize medical risks. By presenting investigational drugs as safe alternatives to traditional steroids, they promote supraphysiological dosing among young users (Machek et al., 2020).

2. Methodology

2.1. Search Strategy and Information Sources

This review employed a search strategy guided by the PICO (Population, Intervention, Comparison, Outcome) framework. An electronic search was executed across PubMed/MEDLINE, Scopus, and the Web of Science Core Collection. The search window spanned from January 2000 through May 2026 to capture SARM clinical development and toxicological reports.

Search syntax relied on Boolean operators to combine Medical Subject Headings (MeSH terms) and free-text keywords across two distinct conceptual domains. The pharmacological domain utilized the string: ("Selective Androgen Receptor Modulators" OR "SARMs" OR "enobosarm" OR "ostarine" OR "RAD-140" OR "LGD-4033") AND ("pharmacodynamics" OR "drug-induced liver injury" OR "DILI" OR "hepatotoxicity" OR "hypogonadism" OR "dyslipidemia"), whereas the socio-technological domain incorporated the string: ("SARMs" OR "PIEDs") AND ("muscle dysmorphia" OR "social media" OR "algorithms" OR "echo chambers" OR "e-commerce"). Finally, these electronic queries were supplemented by manual backward and forward citation tracking of consensus statements and clinical trials.

2.2. Selection Criteria and Risk of Bias Assessment

To minimize selection bias, a two-stage screening process was employed, beginning with an independent review of titles and abstracts by reviewers, followed by a full-text assessment of eligible records. Studies were included if they met three core criteria: publication in peer-reviewed scientific journals; a primary focus on nonsteroidal SARM purity, pharmacodynamics, or clinical outcomes; and adherence to established methodological standards. These standards encompassed randomized controlled trials (Dalton et al., 2011), validated preclinical animal models (Vasilev et al., 2024; Vasilev et al., 2025), regulated analytical chemistry protocols (Gadaj et al., 2019; Gaudiano et al., 2024; Van Wagoner et al., 2017), and comprehensive clinical case reports containing metabolic, endocrine, or histological panels. Conversely, grey literature, internet forum anecdotes, and commercial marketing materials were strictly excluded.

Risk of bias in included case reports was assessed using the CARE guidelines. Cases lacking exact dosing timelines were omitted. Reports of suspected hepatotoxicity were also excluded if they lacked serological viral exclusion panels or fractionated bilirubin measurements. Data regarding compound class, dosage protocols, chemical composition, and pathophysiological indices were extracted.

3. Results

3.1. Clinical Efficacy and Safety Benchmarks: The Phase II Trial Baseline

Evaluating the toxicity of recreational SARM misuse requires establishing the pharmacological baseline intended during clinical development. To illustrate this, Dalton et al. (2011) conducted a 12-week Phase II clinical trial evaluating GTx-024 (enobosarm) in a cohort of 120 healthy elderly men and postmenopausal women. Stringent inclusion criteria demanded specific baseline safety parameters, including a white blood cell count greater than or equal to 3,000 per cubic millimeter and a platelet count greater than or equal to 100,000 per cubic millimeter. Hepatic and renal function were strictly monitored; subjects were excluded if serum alanine aminotransferase (ALT) or aspartate aminotransferase (AST) equaled or exceeded 1.25 times the upper limit of normal, or if serum creatinine exceeded 1.5 mg/dL. Furthermore, male participants had to present a normal prostate specific antigen (PSA) of 4.0 ng/mL or less prior to receiving daily doses ranging from 0.1 to 3 mg, or a placebo, over 86 days.

Efficacy results indicated that the 3-mg dose significantly improved lean mass and physical function, while GTx-024 overall proved to be well tolerated. No serious adverse events required study discontinuation in the main cohorts, with the most common adverse events being limited to headache and back pain. Crucially, the compound maintained its intended tissue selectivity at these micro-doses. This was evidenced by the complete absence of clinically apparent androgenic adverse effects when evaluating hair growth in females (using a modified 4-point Ferriman-Gallwey Scale across six anatomical sites) and sebum production (measured via a DermaLab dual tape reader).

3.2. Pharmacokinetics and Bioavailability

Nonsteroidal SARMs were developed specifically to achieve high oral bioavailability, overcoming the pharmacokinetic hurdles associated with traditional androgen therapy. When administered orally, unmodified testosterone undergoes extensive first-pass hepatic metabolism, necessitating either parenteral administration via intramuscular injection or chemical alkylation, which invariably induces severe hepatotoxicity (Bhasin & Jasuja, 2009; Pope et al., 2014). Unlike classical androgens, molecules like enobosarm and LGD-4033 possess extended terminal half-lives that allow for convenient once-daily oral dosing. Specifically, enobosarm exhibits a terminal half-life of approximately 24 hours (Narayanan et al., 2008), whereas LGD-4033 demonstrates a prolonged half-life ranging from 24 to 36 hours (Machek et al., 2020). This extended pharmacokinetic profile results in stable, steady-state serum concentrations after several days of continuous administration.

Following systemic circulation and metabolic biotransformation, SARMs and their diagnostic metabolites are excreted in urine, which forms the basis for their detection in doping control analyses (Thevis & Schänzer, 2018). While pharmacokinetic stability is advantageous for clinical applications, it contributes to sustained toxicological exposure during recreational megadosing. At supraphysiological doses, SARMs exert compound- and dose-dependent suppressive effects on the hypothalamic-pituitary-gonadal (HPG) axis, potentially by suppressing upstream gonadotropin-releasing hormone (GnRH) or antagonizing luteinizing hormone receptors (Machek et al., 2020).

3.3. E-commerce Product Integrity and Analytical Chemistry Findings

Analytical evaluations of online SARM markets reveal extensive mislabeling and chemical adulteration, necessitating high-resolution techniques like liquid chromatography-tandem mass spectrometry (LC-MS/MS), the gold standard in doping control analysis (Thevis & Schänzer, 2018). Utilizing these protocols, Van Wagoner et al. (2017) assessed 44 products marketed online as SARMs, revealing severe quantitative discrepancies. Specifically, only 18 of the products (41%) contained an active compound matching the label's exact dosage. Among the remaining samples, 11 (25%) contained the listed SARM at significantly different concentrations, eight (18%) substituted the listed SARM with unlisted experimental compounds, and three (7%) combined the listed SARM with undisclosed adulterants. Furthermore, 9% of the products contained no active substance. Independent LC-MS/MS analysis within the European market confirmed these quantitative inaccuracies. Gaudiano et al. (2024) demonstrated that actual quantitative content in illicit products ranged from merely 30% to 90% of the label claim, with over 60% of samples exhibiting severe cross-contamination. Furthermore, the illicit distribution of these compounds is so extensive that advanced ultra-high performance liquid chromatography-tandem mass spectrometry (UHPLC-MS/MS) protocols are now required not only for human doping control but also to monitor potential SARM contamination in agricultural food production (Gadaj et al., 2019).

Beyond inaccurate SARM dosages, this unregulated market introduces severe polysubstance exposure risks, with Van Wagoner et al. (2017) finding unapproved investigational drugs in 39% of the tested products. These chemical adulterants present independent toxicological threats:

- **GW501516 (Cardarine):** Frequently detected in counterfeit SARM products, this compound is a peroxisome proliferator-activated receptor-delta (PPAR- δ) agonist targeting fatty acid oxidation pathways. Clinical development of GW501516 was terminated due to severe preclinical toxicities, prompting an explicit global health warning regarding its cancerogenic potential (World Anti-Doping Agency, 2013).
- **Classical steroid hormones:** SARM products are often adulterated with classical androgens and estrogens. For instance, in a recent case of severe hypogonadism initially attributed to SARM use, laboratory analysis of the ingested capsules revealed undisclosed concentrations of exogenous testosterone and estradiol, highlighting how mislabeling obscures the true pharmacological drivers of adverse events (Chong et al., 2024).
- **Ibutamoren (MK-677):** A non-peptide spiropiperidine that mimics the action of ghrelin, MK-677 stimulates the growth hormone (GH)/IGF-1 axis. In a two-month clinical trial utilizing a 25-mg daily dose, Svensson et al. (1998) demonstrated a significant, 40% increase in serum IGF-1 levels among obese males. While potentially effective for specific growth hormone deficiencies, chronic use by healthy adults impairs glucose homeostasis and reduces insulin sensitivity, increasing the risk of early-onset type 2 diabetes mellitus. Furthermore, MK-677 administration has been associated with transient elevations in serum transaminases, which in one case required study discontinuation due to a three-fold increase in hepatic enzymes (Svensson et al., 1998).

3.4. SARM-Associated Hepatotoxicity and DILI Pathophysiology

While clinical trials of enobosarm demonstrated a stable safety profile at a 3-mg daily dose, recreational users frequently self-administer quantities that are typically four to ten times higher than those evaluated in clinical settings (Vignali et al., 2023). Supraphysiological dosing overwhelms hepatic clearance mechanisms, inducing idiosyncratic drug-induced liver injury (DILI). Mechanistically, the hepatic metabolism of these compounds is hypothesized to disrupt normal metabolic pathways, generating reactive oxygen species (ROS) that deplete intracellular glutathione and induce mitochondrial oxidative stress. Consequently, this oxidative burden disrupts biliary transporters, such as the bile salt export pump (BSEP), leading to intracellular bile acid accumulation and subsequent hepatocyte apoptosis (Habebe et al., 2025; Machek et al., 2020).

SARM-induced DILI typically presents after a latency period of 2 to 16 weeks of continuous exposure. For instance, Habebe et al. (2025) documented the case of a 27-year-old male who developed progressively worsening jaundice and dark-colored urine following one year of daily RAD-140 consumption. Initial laboratory analyses revealed pronounced hepatocellular injury, with an alanine aminotransferase (ALT) peak of 469 U/L and an aspartate aminotransferase (AST) of 122 U/L. As the injury evolved, the patient's total serum bilirubin surged from 9.6 mg/dL to a delayed peak of 27 mg/dL, while the R-factor plummeted to 0.8, marking a clear transition to severe cholestasis.

A similar pathophysiological trajectory was described by Labban et al. (2024) in a 52-year-old male who presented with pruritic jaundice and a rapid 15-pound weight loss after a three-month regimen of LGD-4033. Upon admission, the patient exhibited significantly elevated transaminases (AST 107 U/L; ALT 207 U/L) and a peak total bilirubin of 17 mg/dL, with a direct fraction of 10 mg/dL. Alkaline phosphatase (ALP) peaked at 196 U/L. A comprehensive diagnostic workup rigorously excluded acute viral hepatitis and autoimmune liver diseases. Furthermore, a computed tomography (CT) scan of the abdomen and pelvis ruled out extrahepatic biliary obstruction, confirming the intrahepatic origin of the toxicity. This rapid onset of toxicity is further corroborated by Weinblatt and Roy (2022), who reported significant hepatic dysfunction, characterized by dark urine and generalized pruritus, after only two weeks of enobosarm use.

Epidemiological analyses document the adverse effects of unregulated SARM consumption. In a comprehensive review of 20 documented adverse events, Leciejewska et al. (2024) identified ostarine, ligandrol, and RAD-140 as the most frequently implicated compounds. While drug-induced liver injury emerged as the most prevalent complication, the analysis also exposed severe cardiovascular and neuropsychiatric risks, detailing instances of acute myocarditis, cerebrovascular accidents, and psychiatric disturbances including delusions, paranoia, and altered mood.

Histologically, liver biopsies from affected patients predominantly reveal bland centrilobular canalicular cholestasis, characterized by bile plugs within dilated canaliculi, varying degrees of ductopenia, and portal fibrosis, often without significant hepatocellular necrosis or massive inflammatory infiltration (Leciejewska et al., 2024; Vignali et al., 2023). Because this histological pattern closely mimics classical C-17 alpha-alkylated anabolic steroid toxicity, clinical resolution is notoriously prolonged. Consequently, patients frequently require months of supportive therapy utilizing choleretic agents, such as ursodeoxycholic acid (UDCA), before hepatic panels and physiological function fully normalize (Perananthan & George, 2024; Vignali et al., 2023).

3.5. Endocrine Disruption and HPG Axis Suppression

Even at controlled therapeutic micro-doses, nonsteroidal SARMs significantly alter the endocrine profile. During the Phase II clinical trial evaluating GTx-024, a 3-mg daily dose administered over 86 days induced a notable reduction in sex hormone-binding globulin (SHBG). Specifically, male subjects experienced an average SHBG reduction of 15.8 nmol/L, which was accompanied by a corresponding 7.4 nmol/L decrease in serum total testosterone compared to placebo. Similarly, postmenopausal women exhibited a 36.4 nmol/L reduction in SHBG compared to placebo, alongside decreases in luteinizing hormone (LH) and follicle-stimulating hormone (FSH). However, because free testosterone levels remained unchanged across both cohorts, these suppressions were not considered clinically significant in the context of therapeutic micro-dosing (Dalton et al., 2011).

Conversely, when administered at supraphysiological recreational dosages, this endocrine suppression escalates to pathological levels. By overwhelming the inherent tissue-selectivity of the molecules, high SARM concentrations cross the blood-brain barrier and exert negative feedback on the hypothalamus. This mechanism downregulates gonadotropin-releasing hormone (GnRH) secretion, effectively halting the release of LH and FSH from the anterior pituitary (Machek et al., 2020; Pope et al., 2014). Deprived of LH stimulation, Leydig

cells in the testes cease endogenous testosterone production, culminating in severe hypogonadism and testicular atrophy.

Chong et al. (2024) documented axis disruption in a 40-year-old male who developed bilateral gynecomastia and hypogonadotropic hypogonadism following a six-month regimen of a supplement "stack" containing RAD-140, MK-677, and Cardarine. Laboratory evaluations revealed that the patient's total serum testosterone was severely suppressed to 89-107 ng/dL (3.1-3.7 nmol/L), accompanied by inappropriately low LH (0.6-1.0 IU/L) and FSH (1.3-1.6 IU/L) concentrations. Crucially, in vitro analysis of these black-market supplements revealed significant contamination with undisclosed exogenous hormones, including estradiol and testosterone (with 12.50 ng/dL of testosterone detected within the B-140 capsule, purported to contain RAD-140), directly contributing to the endocrine disruption (Chong et al., 2024). Consequently, abrupt withdrawal from such suppressive regimens leaves users in a state of endogenous testosterone deficiency, precipitating severe withdrawal symptoms including erectile dysfunction, loss of libido, and post-cycle depression (Bonnecaze et al., 2021; Pope et al., 2014).

3.6. Dyslipidemia and Alterations in Metabolic Function

Systemic administration of nonsteroidal SARMs exerts a distinctly deleterious effect on lipid profiles. During the Phase II clinical trial evaluating GTx-024, the 3-mg dose significantly decreased fasting blood glucose and improved overall insulin sensitivity, effectively lowering the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) score by 27.5% (Dalton et al., 2011). However, this metabolic improvement was accompanied by a simultaneous, dose-dependent reduction in cardioprotective high-density lipoprotein (HDL) cholesterol. Specifically, while the 1-mg cohort showed a 17% decrease in HDL, the 3-mg dose induced a 27% reduction, alongside concomitant decreases in total cholesterol and serum triglycerides (Dalton et al., 2011).

The biochemical mechanism of this lipid dysregulation is linked to hepatic androgen receptor activation. When SARMs bind to these hepatic receptors, they induce hepatic triglyceride lipase activity and alter apolipoprotein A1 synthesis. This enzymatic upregulation accelerates the lipolytic degradation and subsequent clearance of HDL particles, precipitating a rapid decline in serum HDL concentrations (Machek et al., 2020). Consequently, despite the favorable reduction in insulin resistance, this rapid drop in HDL represents a significant pharmacological reality of SARM administration that contributes to the long-term cardiovascular risks associated with unregulated use.

3.7. Preclinical Models of Dyslipidemia and Endurance Depletion

In the context of recreational megadosing, physiological disruption extends beyond organ toxicity to actively impair adaptations to exercise. Preclinical models utilizing male Wistar rats demonstrate that compounds like ostarine neutralize the beneficial effects of physical training. To evaluate this, Vasilev et al. (2024) subjected rats to an 8-week regimen of subcutaneous ostarine administration (0.4 mg/kg) combined with a graded treadmill training protocol. While endurance training predictably increased both maximal time to exhaustion (MTE) and grip strength, concurrent administration of ostarine negated these cardiovascular adaptations, inducing a statistically significant reduction in submaximal endurance.

To elucidate the molecular mechanisms driving this decline, genetic analysis of the skeletal muscle was performed. The results revealed that ostarine significantly increased the mRNA expression of myostatin (Mstn), a powerful inhibitor of muscle growth that exerts a catabolic effect on protein metabolism. Notably, ostarine achieved this myostatin upregulation without concurrently affecting the expression of vascular endothelial growth factor-A (VEGF-A) or insulin-like growth factor-1 (IGF-1), highlighting a specific, detrimental alteration in muscular adaptation to exercise (Vasilev et al., 2024).

These negative impacts on systemic health and exercise adaptation were further corroborated when Vasilev et al. (2025) evaluated Ligandrol (LGD-4033) utilizing a similar 8-week protocol. Consistent with previous findings, Ligandrol significantly decreased submaximal endurance and maximal oxygen consumption in the trained cohort. Furthermore, Ligandrol provoked severe alterations in the lipid profile, inducing statistically significant increases in total cholesterol and triglycerides, while high-density lipoprotein (HDL) levels remained unaffected. Endocrine analysis also revealed a severe suppression of serum testosterone and follicle-stimulating hormone (FSH) levels. Collectively, these findings provide definitive preclinical evidence that nonsteroidal SARMs negatively alter lipid metabolism and exert potent suppressive effects on both the HPG axis and cardiovascular endurance (Vasilev et al., 2024; Vasilev et al., 2025).

3.8. Tabular Synthesis of Pharmacological, Analytical, and Toxicological Findings

Table 1. Analytical evaluations of SARM products distributed via unregulated e-commerce networks.

Study / Reference	Sample Size	Authenticity (Presence of SARMs)	Contamination & Unlisted Compounds
Van Wagoner et al. (2017)	44 products	Only 52% contained 1 or more SARMs. 9% contained no active substance.	39% contained unapproved drugs (e.g., ibutamoren, GW501516). 25% contained unlisted substances.
Gaudio et al. (2024)	13 products	~70% contained the declared SARM. In 23%, the expected SARM was replaced by a different one. Quantitative content ranged from 30% to 90% of the label claim.	30% contained undeclared pharmaceutical substances (e.g., tamoxifen, testosterone). >60% showed copresence of >1 active substance (cross-contamination).

Table 2. Clinical characteristics and diagnostic parameters of SARM-induced systemic toxicity.

Reference	Implicated Compound & Duration	Peak Laboratory Findings	Histopathology / Final Diagnosis
Weinblatt & Roy (2022)	Enobosarm for 2 weeks	Markedly elevated transaminases (ALT 346 U/L); Normal total bilirubin (0.5 mg/dL)	Hepatocellular drug-induced liver injury (DILI). R-factor: 7.5; RUCAM score: 7 (Probable)
Labban et al. (2024)	LGD-4033 for 3 months	ALT: 207 U/L; AST: 107 U/L; Total bilirubin: 17 mg/dL; ALP: 196 U/L	Severe idiosyncratic DILI.
Habeb et al. (2025)	RAD-140 for 1 year	ALT peak: 469 U/L; AST: 122 U/L; Total bilirubin delayed peak: 27 mg/dL	Hepatocellular DILI transitioning to severe cholestasis. Initial R-factor: 9.8, dropped to 0.8. RUCAM score: 7 (Probable)
Chong et al. (2024)	RAD-140, MK-677, and Cardarine ("stack") for 6 months	Total testosterone: 89–107 ng/dL; LH: 0.6–1.0 IU/L; FSH: 1.3–1.6 IU/L	Severe biochemical hypogonadotropic hypogonadism and gynecomastia.

4. Discussion

4.1. The Pharmacological Disconnect: Clinical Design vs. Recreational Practice

The literature demonstrates a discrepancy between the clinical development of SARMs and their recreational application. In clinical settings, nonsteroidal SARMs were engineered to act precisely on muscle and bone. In controlled trials, a 3-mg daily dose of GTx-024 (enobosarm) improved physical function and lean mass without significant impact on androgenic tissues. However, even these carefully monitored micro-doses were not entirely free of side effects, as they still induced dose-dependent reductions in high-density lipoprotein (HDL) cholesterol and suppressed endogenous testosterone (Dalton et al., 2011).

The reality of the unregulated market bypasses these clinical safety limits entirely. Driven by modern physique standards, recreational users frequently self-administer doses that are up to ten times higher than those used in research (Vignali et al., 2023). At these supraphysiological levels, the fundamental advantage of SARMs—their tissue selectivity—is completely lost, as the body's androgen receptors become saturated (Machek et al., 2020). Consequently, the surplus of active, unbound chemicals "spills over" into the systemic circulation and penetrates non-target organs, most notably the liver (Machek et al., 2020; Pope et al., 2014).

Once inside the liver, this massive chemical influx creates a severe metabolic overload. The liver's detoxification system, primarily cytochrome P450 enzymes, struggles to process the excess compounds, generating toxic reactive metabolites. This process quickly depletes the cells' protective antioxidants (glutathione) and triggers severe mitochondrial oxidative stress (Machek et al., 2020). As a result, critical pumps responsible for removing bile from the liver—such as the bile salt export pump (BSEP)—stop functioning properly. Bile acids accumulate inside the liver cells, causing them to die and resulting in severe drug-induced liver injury (DILI) (Habeib et al., 2025; Machek et al., 2020).

While metabolic overload contributes to hepatotoxicity, the clinical presentation of SARM-induced liver injury varies. The latency period and injury severity do not consistently correlate with the administered dose, indicating that SARM-induced hepatotoxicity is an idiosyncratic reaction (European Association for the Study of the Liver, 2019).

4.2. Algorithmic Architecture, Muscle Dysmorphia, and Neurocognitive Toxicity

Recreational SARM consumption is associated with social media platforms, where algorithms promote digitally altered physiques. Continuous exposure to these unrealistic standards during adolescence correlates with rising rates of muscle dysmorphia, a condition classified under the DSM-5-TR as a specifier of Body Dysmorphic Disorder (American Psychiatric Association, 2022; Nagata et al., 2023). This pathology is characterized by severe dietary rigidity, compulsive resistance training, and the continuation of PIED use despite documented physical harm. The architecture of these platforms exacerbates the issue by favoring the formation of echo chambers; recommendation systems enhance homophily, drawing users into closed networks that filter out dissenting medical information and reinforce extreme positions (Cinelli et al., 2021). Consequently, these isolated communities increasingly rely on anecdotal, unverified dosing protocols distributed by uncredentialed influencers (Gaudio et al., 2024).

Network analyses of male weightlifters demonstrate how these specific behavioral symptoms interlock to sustain substance abuse. Utilizing the Expected Influence (EI) metric to quantify node centrality—which measures the cumulative strength of a symptom's connections within a network—Scarath et al. (2023) identified that exercise dependence acts as the most central node among AAS users. This symptom exhibited the highest EI score, forming strong pathological connections to diet and size/symmetry concerns. This indicates that obsessive-compulsive training and dietary rigidity are the primary behavioral drivers maintaining the substance abuse network. Addressing these high-centrality nodes through targeted cognitive-behavioral interventions is therefore critical to dismantling the psychological dependency loop (Scarath et al., 2023).

Furthermore, the supraphysiological stimulation of central nervous system androgen receptors raises concerns regarding neurocognitive toxicity. While earlier research established that young AAS users develop significant deficits in visuospatial memory and executive function, recent evaluations utilizing the Cambridge Neuropsychological Test Automated Battery (CANTAB) in middle-aged men revealed no significant cognitive differences between long-term users and non-users (Kaufman et al., 2024). These data suggest that androgen-induced cognitive impairments manifest during early exposure in youth and plateau, while non-users develop similar deficits later due to age-related cognitive decline (Kaufman et al., 2024). Since SARMs cross the blood-brain barrier to exert negative feedback on the hypothalamus, high-dose administration exposes the developing adolescent brain to these very same early neurotoxic risks. Impaired executive function may diminish the user's capacity to evaluate the risks of SARM consumption, reinforcing the dependency.

4.3. Regulatory Challenges and Supply-Side Vulnerabilities

E-commerce distribution networks currently operate with minimal oversight, allowing vendors to exploit legal loopholes by labeling unapproved substances as "research chemicals" to bypass pharmaceutical safety regulations (Van Wagoner et al., 2017). Consequently, this regulatory vacuum results in severe product instability, exposing recreational users to unlisted experimental compounds, severe cross-contamination, and highly variable dosages (Gaudiano et al., 2024; Van Wagoner et al., 2017). Complicating the clinical landscape further, consumers often perceive these potent agents as standard dietary or lifestyle supplements (U.S. Food and Drug Administration, 2023). Due to underlying stigma or fear of legal and medical judgment, these individuals rarely disclose their substance use to healthcare providers (Labban et al., 2024). Ultimately, this lack of clinical transparency significantly delays the accurate diagnosis of acute toxicities, such as idiosyncratic drug-induced liver injury, and heavily shifts the medical burden onto acute care facilities (Labban et al., 2024).

4.4. Clinical Implications and Diagnostic Recommendations

The increasing prevalence of SARM use among young adults requires physician awareness during diagnosis. As the lack of voluntary disclosure complicates early detection, clinicians must maintain a high index of suspicion when young, athletic individuals present with unexplained jaundice or lethargy, and should explicitly inquire about the consumption of online performance-enhancing supplements (Labban et al., 2024).

Diagnosis of hepatotoxicity relies heavily on standardized clinical algorithms, most notably the Roussel Uclaf Causality Assessment Method (RUCAM) updated by Danan and Teschke (2015). As the standard tool for evaluating suspected DILI, RUCAM assigns a numerical score ranging from -9 to +14 points across seven distinct domains. These domains evaluate the time to symptom onset, the clinical course following drug cessation, specific risk factors (such as age and alcohol use), concomitant medications, existing hepatotoxicity profiles, the rigorous exclusion of non-drug causes, and the response to unintentional re-exposure (Danan & Teschke, 2015). A final score of 9 or higher indicates a highly probable causal relationship, 6 to 8 indicates a probable relationship, and 3 to 5 categorizes the relationship as possible.

Concurrently, clinicians must calculate the R-factor—the ratio of serum ALT divided by its upper limit of normal (ULN), divided by serum ALP divided by its ULN—to classify the injury pattern. While an R-factor ≥ 5 indicates hepatocellular injury and ≤ 2 indicates cholestatic injury, SARM-induced DILI can initially present as hepatocellular injury before transitioning to cholestasis (European Association for the Study of the Liver, 2019; Habeb et al., 2025). To achieve a definitive RUCAM score, guidelines mandate the rigorous serological exclusion of competing etiologies, requiring testing for acute viral hepatitis (A, B, C, E), Cytomegalovirus, Epstein-Barr virus, and autoimmune markers prior to confirming SARM-induced toxicity (Danan & Teschke, 2015; European Association for the Study of the Liver, 2019).

Beyond hepatotoxicity, SARM misuse involves self-administered endocrine interventions that severely disrupt the HPG axis. As outlined in the harm reduction frameworks established by Bonnacaze, O'Connor, and Burns (2021), the cessation of exogenous androgens precipitates a hypogonadal state characterized by major depressive episodes and sexual dysfunction. To mitigate these withdrawal symptoms, users autonomously initiate unmonitored "Post-Cycle Therapy" (PCT) by sourcing Selective Estrogen Receptor Modulators (SERMs), such as clomiphene citrate (25 to 50 mg daily) or tamoxifen (10 to 20 mg daily). These agents act as estrogen receptor antagonists in the hypothalamus, blocking the negative feedback loop to stimulate the release of GnRH, LH, and FSH. Furthermore, users frequently stack SERMs with human chorionic gonadotropin (hCG), an exogenous LH analog that directly stimulates Leydig cells to maintain testicular volume and basal testosterone production (Bonnacaze et al., 2021).

The illicit sourcing of PCT agents from unregulated vendors introduces additional toxicological risks, as unmonitored SERM usage is associated with hypercoagulable states, deep vein thrombosis, and visual field disturbances. Consequently, clinicians must actively screen for concurrent SERM and hCG use when evaluating young males presenting with SARM-induced hepatotoxicity or endocrinopathy (Bonnacaze et al., 2021; Pope et al., 2014).

Management requires concurrent medical and psychiatric interventions. Addressing physiological symptoms is insufficient if the underlying psychological driver remains. Patients presenting with SARM-induced toxicities should be evaluated using DSM-5-TR criteria and referred for targeted cognitive-behavioral therapy to dismantle the psychological dependency loop and reduce the risk of relapse (American Psychiatric Association, 2022; Scarth et al., 2023).

4.5. Limitations of the Review

While this review synthesizes the current pharmacological and toxicological literature on SARM misuse, it has several limitations. The main limitation is our reliance on Level V evidence, namely retrospective case reports and case series, to describe human toxicity (Vignali et al., 2023). Since ethical constraints prevent conducting prospective, randomized controlled trials with the high doses typical of recreational use, our clinical understanding stems almost entirely from observational data and animal models. This reliance naturally introduces publication bias. The medical literature tends to capture only the most severe, acute adverse events that lead to hospital admissions, meaning milder or chronic side effects are likely underreported. In addition, the chemical instability and frequent contamination of SARM products sold online make it difficult to confidently link specific pathophysiological mechanisms to individual SARM compounds in human users (Gaudiano et al., 2024; Van Wagoner et al., 2017).

5. Conclusions

The recreational use of SARMS is a growing public health issue that goes beyond traditional sports doping. Early clinical trials indicate therapeutic potential for muscle-wasting conditions at controlled micro-doses, whereas recreational use involves unmonitored, high-dose protocols. Facilitated by social media, online markets distribute contaminated products and promote supraphysiological dosing protocols.

By taking supraphysiological doses, users override the intended tissue selectivity of SARMS, resulting in idiosyncratic drug-induced liver injury, hypogonadism, and atherogenic lipid profiles. To mitigate this problem, healthcare providers and regulators need to look past standard anti-doping policies. Recreational PIED use should be treated as a combined issue of digital health and psychiatric dependency. Addressing this trend will require stricter monitoring of online distributors, better transparency regarding social media algorithms, and updated medical education so physicians can better diagnose and manage SARM-induced toxicity. Looking forward, research should focus on longitudinal studies to determine if these toxicities are reversible over time, as well as finding effective psychiatric interventions to help users break the cycle of dependency.

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Ethics Approval: As this is a review article synthesizing previously published and publicly available literature, ethical approval from an institutional review board was not required.

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