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USERS VS. NON-USERS OF ANABOLIC STEROIDS: IS THE DIFFERENCE ONLY STEROID USE?

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ABSTRACT

Research on anabolic–androgenic steroids (AAS) often categorizes individuals simply as users or non-users, assuming that exposure itself is the only meaningful difference between the groups. Such a dichotomy overlooks important baseline and behavioral factors that shape health outcomes and risks generating misleading interpretations. People who engage in AAS use frequently present distinct psychosocial and medical histories, including psychiatric conditions, body-image concerns, and social vulnerabilities, which may influence both the decision to initiate use and the probability of developing adverse outcomes. In addition, AAS use almost never occurs in isolation: polypharmacy with hormones, stimulants, and ancillary drugs, as well as concomitant consumption of alcohol, tobacco, and illicit substances, introduces further complexity by modifying risks in ways that cannot be separated from the effects of steroids alone. When these heterogeneous profiles are collapsed into a binary classification, observational studies risk inflating or misplacing harm as directly attributable to AAS. This article highlights why the distinction between users and non-users cannot be considered scientifically rigorous without attention to these confounding factors and argues for the need of more robust methodologies, including multivariable analyses and prospective cohort designs, to achieve more accurate and clinically meaningful conclusions.

KEYWORDS: Anabolic–androgenic steroids (AAS), Steroid, polypharmacy.

INTRODUCTION

The use of anabolic–androgenic steroids (AAS) has become a relevant issue in both clinical and public health domains, given their association with adverse outcomes across cardiovascular, psychiatric, and metabolic systems.[1–3] Despite the frequency with which these risks are reported, much of the evidence relies on observational designs that separate

populations simply into “users” and “non-users.” This binary approach assumes that AAS use itself is the only relevant difference between the groups, while ignoring important methodological challenges.[4,5]

Such simplification may obscure the role of baseline differences that precede AAS initiation. Evidence indicates that users often present distinct psychosocial and medical histories, higher prevalence of polypharmacy, and frequent concomitant use of licit and illicit substances.[6–9] These factors may independently contribute to the outcomes under investigation, thereby undermining causal inference. We therefore examine why the common ‘users vs. non-users’ dichotomy is not methodologically adequate, focusing on baseline psychosocial/medical differences, polypharmacy, and concomitant licit/illicit substances.

Why the groups are not comparable:

I - Medical and psychosocial backgrounds

Research on the motivations for anabolic–androgenic steroid (AAS) use shows that this behavior is embedded within broader psychosocial and clinical vulnerabilities.[9] A systematic review reported higher prevalence among AAS users of anorexia, muscle dysmorphia, low self-esteem, negative body image, psychiatric disorders, drug use, and traumatic events such as bullying, rape, and divorce, indicating that a profile of risk often precedes AAS initiation.[9]

Survey data reinforce this pattern. In the Anabolic 500 study, 6.1% of participants reported a history of sexual abuse and 10% reported physical abuse.[7] Similarly, an investigation of 75 female weightlifters found that 13% had experienced rape, which contributed to compulsive training behaviors and subsequent adoption of ergogenic substances such as AAS and clenbuterol; notably, 70% reported using these substances to alter body image and enhance performance.[10]

Further evidence highlights the broader psychosocial context of use. Adolescent initiation has been linked more strongly to clusters of behavioral disorders than to athletic motivations alone.[11] Consistently, a Norwegian study of 1,351 students identified alcohol use, anxiety and depressive symptoms, and illicit drug consumption as major predictors of AAS initiation [12], while a Swedish study of 1,353 students reported associations with immigrant status, poor school performance, low self-esteem, and the use of alcohol, sedatives, and tranquilizers.[13] Together, these findings indicate that AAS users differ from non-users well before exposure, complicating any direct attribution of outcomes solely to steroid use (**Table 1**).

Table 1: Baseline differences, more frequent patterns, between AAS users and non-users.

Category	AAS Users	Non-users
Psychological/psychiatric	Anxiety, depression, muscle dysmorphia, low self-esteem, history of physical/sexual abuse	Lower prevalence of psychiatric and psychological disorders
Motivation	Body image modification, compulsive training, performance enhancement	Predominantly recreational or athletic motivation
Social history	Bullying, divorce, higher social vulnerability, immigrant status, poor academic performance	Lower prevalence of social vulnerabilities
Previous drug use	Higher likelihood of prior alcohol, cannabis, and other substance use	Less frequent and less diverse substance use

II - Polypharmacy

Polypharmacy is highly prevalent among individuals who use anabolic–androgenic steroids (AAS) and represents a central reason why comparisons with non-users are methodologically fragile.[6] In practice, AAS are almost never used in isolation. Users commonly layer other agents on top of steroid regimens, a pattern that can independently shape cardiovascular, hepatic, endocrine, and neuropsychiatric outcomes. When statistical analyses do not explicitly measure and separate these co-exposures, effect estimates are vulnerable to bias, and there is a substantial risk of assigning causal weight solely and improperly to AAS.[6]

A systematic review synthesizing fifty studies documented routine concomitant use of multiple drug classes during AAS cycles, including analgesics, anti-inflammatory agents, opioids, central nervous system stimulants and depressants, diuretics, dermatologic or "cosmetic" medications, cardiovascular drugs, recreational substances, and additional hormones. The authors underscored that this constellation of exposures is an underrecognized risk factor that clinicians, researchers, and policy makers must account for when interpreting outcomes.[6] Complementing these findings, survey data mapped the ancillary pharmacology typically combined with AAS into four broad categories: accessory anabolic hormones such as growth hormone, insulin, and IGF-1; stimulants such as ephedrine, amphetamine, thyroid hormones, yohimbine, and dinitrophenol; diverse agents including diuretics, muscle relaxants, and analgesics; and drugs used to mitigate side effects, including clomiphene citrate, aromatase inhibitors, tamoxifen, and human chorionic gonadotropin.[14]

Concomitant use is also evident among women. In a Brazilian cohort, 18.7% reported diuretics, 2.1% ephedrine, and 2.1% clenbuterol alongside AAS.[16] Historical data indicate escalation over time in the uptake of potent ancillary hormones. Among male users in 1997, reported use was 12% for growth hormone, 2% for insulin, and 2% for thyroid hormones; by 2006, these figures rose to 25, 25, and 45%, respectively.[17,14] Several adjuncts, especially when taken without medical supervision, can precipitate acute clinical emergencies independent of AAS exposure. Insulin, thyroxine, diuretics, and stimulants are notable examples, each carrying potential for hypoglycemia, arrhythmias, psychiatric disturbances, and serious cardiovascular events.[15,3]

Corroborating this line of reasoning, Piatkowski and colleagues compared men consuming AAS plus clenbuterol with men consuming AAS alone (N = 1,146) and observed significantly greater odds of several adverse effects in the clenbuterol group: negative impact on the heart (adjusted odds ratio [aOR] 2.76; $p < .001$), rapid mood fluctuation (aOR 1.73; $p = .010$), and irrational excitability (aOR 1.61; $p = .032$).[18] These findings reinforce that co-exposures can independently worsen outcomes, and that analyses failing to measure and model adjunct compounds risk attributing causality solely and improperly to AAS.

Adverse effects that arise during AAS use do not routinely lead to cessation. Many users adopt self-medication strategies with additional agents to control side effects rather than reducing or discontinuing AAS, compounding exposure and further confounding attempts to attribute outcomes to a single agent.[14,6] In sum, because AAS use almost never occurs alone, analyses that compare "users" with "non-users" without measuring and properly modeling co-exposures are at high risk of biased inference, including the erroneous attribution of causality entirely to AAS.[6,14,16]

The data observed, with the potential influence of polypharmacy as a confounding factor for causality, are presented in the following table (**Table 2**).

Table 2: Polypharmacy associated with AAS use.

Drug category	Common examples	Effects/risks independent of AAS
Accessory anabolic hormones	Growth hormone, insulin, IGF-1, thyroid hormones (T3)	Hypoglycemia, arrhythmias, psychiatric disturbances
Stimulants/thermogenics	Ephedrine, amphetamines, yohimbine, dinitrophenol (DNP)	Arrhythmias, hypertension, psychosis, sudden death
Other ergogenic agents	Diuretics, muscle relaxants, analgesics	Dehydration, renal failure, dependence
Agents to mitigate side effects	Clomiphene, aromatase inhibitors, tamoxifen, hCG	Independent endocrine effects, thrombotic risk

III - Concomitant licit and illicit substances

Concomitant use of licit and illicit substances is widely documented among individuals who use anabolic–androgenic steroids (AAS), and AAS use, again, almost never occurs in isolation.[6] Across cohorts, the most frequently reported substances include alcohol, tobacco, cannabis, cocaine, hallucinogens, heroin, inhalants, LSD, and methamphetamine, indicating clustered risk behaviors rather than a single pharmacologic exposure.[6] In the Anabolic 500 survey, past-year concomitant use rates among AAS users were 47.2% for alcohol, 22.9% for tobacco, 30.6% for cannabis, 3.0% for heroin, and 11.3% for cocaine.[7] Among women, parallel patterns were observed: 29.2% reported alcohol and 10.4% tobacco alongside AAS.[16] These exposures have independent cardiometabolic and neuropsychiatric effects and therefore confound any attempt to attribute subsequent outcomes solely to AAS.[6,7,16]

Multiple studies also suggest that psychoactive substance use often precedes AAS initiation, reinforcing the role of baseline vulnerabilities. Prior consumption of alcohol and cannabis is frequently reported, alongside opioids and heroin, before the onset of AAS use.[6,19,20,15,8] Moreover, AAS users commonly combine numerous performance-enhancing drugs (PEDs): in the Anabolic 500 study, participants reported a mean of 11.1 different substances (maximum 29) when considering AAS plus other PEDs. Even excluding AAS, users reported a mean of 8.9 substances (maximum 28), a number significantly higher than that of non-users.[7]

Taken together, these quantitative patterns show that co-exposures are the norm. When statistical analyses do not measure and appropriately model alcohol, tobacco, illicit drugs, and other PEDs, effect estimates are highly vulnerable to confounding, with a substantial risk of misattributing causality entirely and improperly to AAS.[6,7,8,15,16,19,20] The prevalence of concomitant use of licit and illicit substances among individuals who use AAS, as reported in survey studies, is summarized in the table below (**Table 3**).

Table 3: Prevalence of concomitant use of licit and illicit substances among AAS users.

Substance	Prevalence among AAS users (survey data)
Alcohol	29.2 – 47.2%
Tobacco	10.4 – 22.9%
Cannabis	30.6%
Cocaine	11.3%
Heroin	3.0%

A conceptual flowchart summarizing the interrelated factors that challenge comparability between AAS users and non-users is presented in **Figure 1.** and illustrates the conceptual cycle from psychosocial and social vulnerabilities, through the decision to initiate anabolic–androgenic steroid (AAS) use, to patterns of polypharmacy and co-exposures,

culminating in clinical outcomes. It also highlights the common misinterpretation in observational research, where outcomes are attributed solely to AAS without accounting for these underlying and concomitant factors (**Figure 1**).

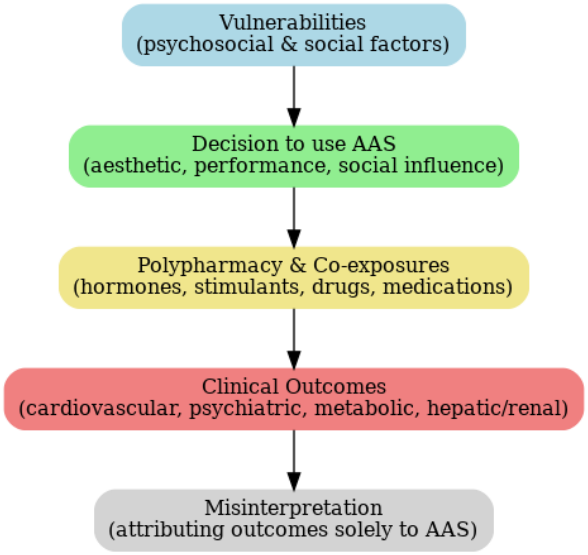

Figure 1: Conceptual cycle of vulnerabilities, AAS initiation, polypharmacy, and clinical outcomes.

DISCUSSION

Comparisons between "users" and "non-users" of anabolic–androgenic steroids (AAS) are methodologically fragile because baseline differences and co-exposures are both frequent and substantial.[6,7,9] Many individuals who turn to AAS already present distinct psychosocial and clinical profiles before the first exposure. Concerns with body image, a history of psychiatric disorders, or broader patterns of behavioral risk are often part of this trajectory and can independently influence outcomes. If these aspects are not properly measured and accounted for, they inevitably bias the interpretation of causality.[9,12,13]

Another central point is that AAS use rarely occurs in isolation. Most users combine steroids with a wide range of additional substances, including other performance-enhancing drugs, prescription or over-the-counter medications, and both licit and illicit drugs with independent cardiometabolic and psychiatric effects.[6,7,14,16,19,20] This layered pharmacology makes it very unlikely that a single indicator of "use" can capture the complexity of exposure. Data from surveys illustrate this well: alcohol, tobacco, cannabis, cocaine, and heroin are all consumed at high rates among AAS users, and many report the use of more than ten different substances during their trajectories.[7,16] Studies also show that adjunct compounds can worsen outcomes, as in the case of clenbuterol combined with AAS, which is associated with higher odds of cardiac symptoms and mood instability.[18] These findings reinforce the risk of attributing causal weight exclusively to steroids when other exposures remain unmeasured.

When observational data are interpreted without attention to these elements, the result is often an exaggeration or misplacement of harm, which has consequences for both clinical practice and public health communication.[1,3] Moving forward, studies in this field need to take these realities into account. At the very least, researchers should measure and report co-exposures, describe substance-use profiles alongside AAS status, and discuss the possibility of residual confounding whenever full adjustment is not feasible.[6,7,14,16,18,20] Methodologically, stronger designs are also required. Multivariable analyses that adjust for polypharmacy and comorbidities, as well as prospective cohorts that monitor

vulnerabilities, exposures, and outcomes over time, are strategies that can help overcome the current limitations. Only by moving beyond the oversimplified dichotomy of “user” versus “non-user” will it be possible to reach conclusions that are both accurate and clinically meaningful.

CONCLUSION

Users and non-users of AAS do not differ only by steroid exposure. Baseline psychosocial and clinical profiles, frequent polypharmacy, and the common concomitant use of licit and illicit substances create systematic incomparability that challenges causal interpretation in observational research. Because AAS are almost never used in isolation, analyses that fail to measure and model co-exposures are at high risk of biased inference and erroneous attribution of harm solely to AAS. Therefore, the binary simplification between users and non-users should be replaced by models that reflect the complex reality of substance use, otherwise research will continue to generate biased inferences of limited clinical value.

Future work should not only adopt cautious causal language but also systematically explore alternative methodological approaches, including prospective cohorts with longitudinal monitoring, case–control studies with careful matching of psychosocial and behavioral factors, and mixed-methods research integrating quantitative and qualitative data. Multicenter registries capturing real-world patterns of polydrug use may also provide valuable insights. By embedding these strategies into study design, the field can move toward a more accurate and clinically meaningful understanding of the health risks associated with AAS use.

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