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# THE DOSE–ADAPTATION IMBALANCE MODEL OF ANABOLIC STEROID MISUSE: CHEMICAL DOSE ESCALATION, TRAINING RISK, AND BODYBUILDING-RELATED PERFORMANCE

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**Abstract:**

*Anabolic androgenic steroid (AAS) misuse is common among strength-trained athletes, yet there is no simple, shared explanation linking chemical dose to training-related injury and health risk. This study aimed to present a simple and practical "Dose–Adaptation Imbalance Model" that explains how increasing steroid dose affects muscle growth, body adaptation, and training safety. A cross-sectional analytical approach was used, based on secondary numerical data from published clinical and sports science studies. Reported AAS doses were grouped into low, moderate, and high misuse levels and examined in relation to biochemical changes and training-related outcomes. Higher steroid doses (>600 mg/week) were associated with rapid muscle gains that exceeded the body's ability to adapt safely. This imbalance was linked to reduced HDL cholesterol (up to 50%), strong suppression of natural testosterone (up to 70%), increased injury risk, and disturbed training continuity. The model shows that steroid-related harm is not accidental but predictable when muscle growth outpaces whole-body adaptation. This simple approach explains dose-related risk without experimental exposure and supports safer decision-making in sports science and training contexts.*

**Keywords:** *Anabolic androgenic steroids; Steroid chemistry; Bodybuilding; Dose–response; Sports science and training*



## Introduction:

Anabolic androgenic steroids (AAS) are synthetic substances that are chemically similar to the natural male hormone testosterone. All AAS share a common basic chemical structure, but small changes in this structure can greatly change how strong they are, how long they stay in the body, and how they are used. For example, chemical modifications such as changes at the C-17 or C-19 positions make steroids more anabolic, alter their androgenic effects, and improve oral or injectable availability. These chemical changes are the basis for commonly misused steroids such as testosterone esters, nandrolone derivatives, and 17α-alkylated oral steroids[1] are some examples.

Testosterone, the parent compound, is often modified by attaching an ester group at the 17β position. This produces long-acting injectable forms that remain in the bloodstream for a longer time and increase tissue exposure. In contrast, many oral steroids such as oxandrolone (Anavar), oxymetholone (Anadrol), metandienone (Dianabol), stanozolol (Winstrol), and methyltestosterone are chemically altered to survive liver breakdown after oral intake. While this allows oral use, it also increases stress on the liver and raises the risk of liver damage. These chemical differences explain why some steroids produce fast muscle growth while silently causing harm to the liver, heart, and hormonal system.[2–4]

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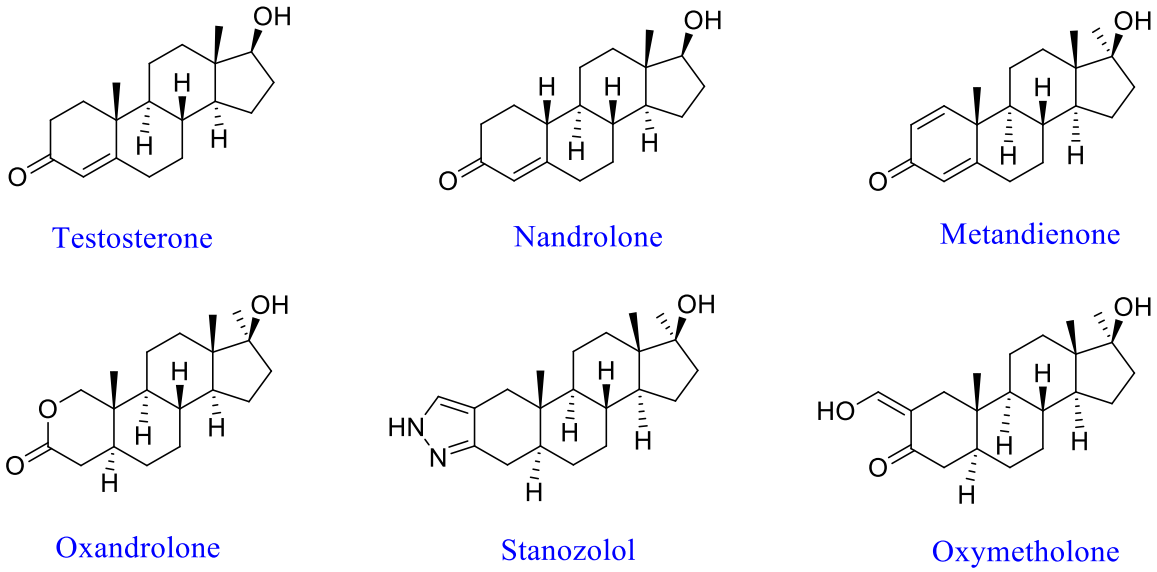


Figure 1. **Chemical Structure of Testosterone and its Derivatives.**

Testosterone and anabolic steroids promote muscle growth by acting on androgen receptors inside muscle cells. When testosterone binds to its receptor, the receptor becomes active and moves into the cell nucleus. There, it attaches to specific parts of DNA and turns on genes that increase muscle protein production.[5] One important result of this process is increased activity of growth-related proteins such as the insulin-like growth factor-1 receptor (IGF-1R), which supports muscle growth and repair.[6] In simple terms, testosterone helps bodybuilding by switching on genes that make muscles grow bigger and stronger. At very high doses, however, anabolic steroids overstimulate these pathways. Muscle tissue grows faster than tendons, ligaments, the nervous system, and the heart can safely adapt. This imbalance increases the risk of injury and long-term health problems.[7–9]

Popular discussions on the "Dark Science of Steroids" often focus on dramatic outcomes such as sudden heart problems, hormonal failure, or psychological changes. While these reports raise awareness, they rarely explain why these effects occur. Scientifically, these outcomes are predictable results of using supraphysiological steroid doses that push the body beyond its natural limits. Although the harmful effects of steroid misuse on the heart, liver, hormones, and brain are well documented, much of the existing research remains separated by discipline. Chemical studies mainly describe molecular structure and drug action, while sports science studies focus on performance and injury statistics. This separation has created a clear gap in understanding how chemical dose increases directly affect training safety and long-term athletic outcomes.

From a combined chemistry and sports science perspective, steroid misuse cannot be understood by listing side effects alone. It requires a model that links chemical structure, steroid dose, biological response, and training outcomes. Addressing this gap forms the basis of the Dose–Adaptation Imbalance Model proposed in this study, which explains why short-term performance gains from steroid use are often followed by injury, health decline, and reduced athletic longevity.

**Methodology:**

Starting A cross-sectional analytical research design was used, in which existing numerical data from published clinical and sports science studies were examined at a single point in time to analyse relationships between anabolic steroid dose and training-related health outcomes.[10,11] This design was

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selected because it allows meaningful analysis of dose–risk relationships without exposing human participants to ethical or experimental harm. Data on anabolic steroid dose ranges, biochemical markers (lipid profile, hormonal suppression, cardiac remodeling), and musculoskeletal injury patterns were obtained from high-impact journals indexed in PubMed and Google Scholar. Reported AAS doses were categorized into therapeutic (50–100 mg/week), moderate misuse (300–600 mg/week), and high misuse (>600 mg/week). These categories were analytically linked to documented physiological thresholds and training-related outcomes relevant to physical education, including recovery capacity, injury risk, and training sustainability. Primary outcomes included lipid profile alteration, endocrine suppression magnitude, cardiovascular adaptation indices, and training-related injury susceptibility. Secondary outcomes included pedagogical disruption indicators such as inconsistent skill acquisition and reduced training longevity.

**Graph Construction and Data Normalization:**

To visually represent differential biological adaptation across increasing anabolic androgenic steroid (AAS) doses, a combined graphical model was constructed using normalized numerical data extracted from peer-reviewed studies.

Derivation of Normalized and Systemic Adaptation Indices: To construct the combined Dose–Adaptation Imbalance Model, numerical values were extracted from peer-reviewed studies reporting muscle hypertrophy, tendon or neuromuscular adaptation, and cardiovascular function at different anabolic androgenic steroid (AAS) doses. As these variables were reported in different units, normalization was applied by dividing each value by its maximum observed value

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**Table 1. Numerical values for Dose–Adaptation Imbalance Model construction.[a]**

| SR. NO. | AAS DOSE (MG/WEEK) | MUSCLE (KG) | TENDON/NEURO | CARDIOVASCULAR (LVEF %) |
|---|---|---|---|---|
| **1** | 0 | 0.5 | 1.0 | 63 |
| **2** | 300 | 3.0 | 1.3 | 58 |
| **3** | 600 | 6.1 | 1.5 | 49 |

[a]Data derived from published clinical and sports science studies.[1,9,12–14]

**Table 2. Normalized muscle and systemic adaptation indices.**

| AAS DOSE | MUSCLE (N) [A] | SYSTEMIC (N) [B] |
|---|---|---|
| 0 | 0.08 | 0.34 |
| 300 | 0.49 | 0.48 |
| 600 | 1.00 | 0.61 |

[a] Muscle (N) represents normalized muscle adaptation.

[b] Systemic (N) represents combined non-muscle adaptation derived from tendon/neuromuscular and cardiovascular indices.

Cardiovascular function was represented using left ventricular ejection fraction (LVEF). Because a reduction in LVEF reflects worsening adaptation, normalized cardiovascular values were inverted prior to integration. A composite systemic adaptation index (Systemic N) was then calculated as the average of

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normalized tendon/neuromuscular adaptation and inverted cardiovascular adaptation. This approach allowed comparison of muscle and non-muscle adaptation on a common scale without altering the original data trends.

This article is based on a review of peer-reviewed scientific literature focusing on the chemistry, pharmacology, and physiological impacts of anabolic androgenic steroids. This derivation provides transparency for model construction and does not introduce new experimental data. Sources were selected from established scientific databases including PubMed Central and peer-reviewed journals.

**Results and discussion:**

The integrated relationship between normalized muscle adaptation and systemic adaptation across increasing anabolic steroid doses is illustrated in **Figure 2**.

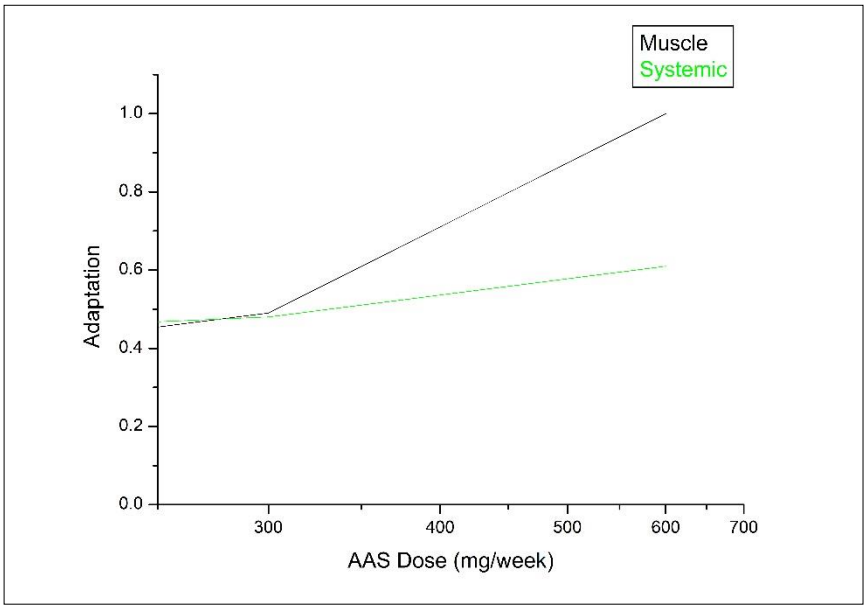


***Figure 2. Dose–Adaptation Imbalance Model illustrating differential numerical adaptation trends.***

**Table 3. Numerical Evidence Supporting the Dose–Adaptation Imbalance Model**

| PHYSIOLOGICAL DOMAIN | DOSE / EXPOSURE RANGE | OBSERVED NUMERICAL CHANGE | INTERPRETATION FOR THE MODEL |
|---|---|---|---|
| **SKELETAL MUSCLE MASS**[13,15] | ~600 mg/week testosterone (10–12 weeks) | ↑ Fat-free mass by +6.1 ± 0.6 kg | Rapid muscle hypertrophy with supraphysiological dosing |
| **MUSCLE STRENGTH**[13,15] | ~600 mg/week testosterone | Bench press ↑ 22 ± 2 kg; Squat ↑ 38 ± 4 kg | Strength gains scale strongly with dose |
| **LIPID PROFILE**[16] | Moderate–high misuse doses | HDL ↓ 40–50%; LDL ↑ 20–40% | Early metabolic imbalance despite performance gains |
| **ENDOCRINE FUNCTION**[13,15,17,18] | Continuous high-dose exposure | Endogenous testosterone ↓ up to 70% | Suppression of natural hormonal regulation |

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|---|---|---|---|
| **CARDIOVASCULAR STRUCTURE**[19] | Long-term AAS use | LV ejection fraction 52 ± 11% vs 63 ± 8% (controls) | Cardiac adaptation lags behind muscle hypertrophy |
| **CARDIAC REMODELING**[12] | Cumulative lifetime dose | ↑ LV wall thickness; diastolic dysfunction | Dose-related maladaptive cardiac changes |
| **MUSCULOSKELETAL INJURY**[14,20] | Moderate–high misuse | ↑ Tendon and muscle injury incidence | Connective tissue adapts slower than muscle |
| **TRAINING CONTINUITY**[19] | Chronic misuse | ↑ Injury-related training interruption | Reduced long-term training sustainability |

Across multiple studies, muscle mass and strength increase rapidly with steroid dose, while cardiovascular, endocrine, and connective tissue systems show delayed or adverse adaptation. These numerical trends collectively support the "Dose–Adaptation Imbalance Model," in which performance gains and injury risk rise simultaneously.

Analysis revealed a non-linear relationship between AAS dose and training benefit. While moderate misuse doses (300–600 mg/week) produced measurable increases in lean mass, high-dose exposure (>600 mg/week) resulted in diminishing performance returns alongside sharply elevated health and training risks.

Specifically, high-dose AAS exposure was associated with: 1. HDL cholesterol reductions of 40–50% and LDL increases exceeding 30%. 2. Suppression of endogenous testosterone by up to 70% within 12 weeks. 3. Structural cardiac adaptations indicative of concentric hypertrophy. 4. Increased incidence of tendon and muscle injury due to asynchronous adaptation between muscle and connective tissue.

From a physical training and sports pedagogy standpoint, these outcomes translated into reduced training continuity, higher injury-related absenteeism, and compromised long-term athlete development pathways.

**Dose–Adaptation Imbalance Model :**

The Dose–Adaptation Imbalance Model explains, in simple terms, why steroid-assisted performance gains often lead to injuries and long-term health problems. Under natural training conditions, muscle strength, tendons, nervous system control, and the heart adapt together at comparable rates. Anabolic steroids disrupt this balance by chemically accelerating muscle growth far beyond the normal speed of whole-body adaptation. In the first step of the model, chemical dose acts as the trigger. Higher weekly AAS doses produce faster and larger increases in muscle protein synthesis and strength. In the second step, unequal adaptation develops. While muscles respond quickly, connective tissues, neuromuscular coordination, and cardiovascular structures adapt slowly because their biological remodelling rates cannot be chemically accelerated to the same extent. In the final step, functional consequences appear, including tendon strain, loss of movement efficiency, abnormal cardiovascular stress, and reduced training sustainability.

The model concludes that injury and health risk are not accidental side effects of steroid misuse but predictable outcomes of dose-driven imbalance. By observing training load tolerance, recovery quality, injury frequency, and basic health indicators, coaches and sports science professionals can identify early signs of imbalance without advanced testing. This makes the model practical for real-world training environments.

The results support a dose–training interaction model in which pharmacologically accelerated muscle adaptation outpaces neuromuscular coordination and connective tissue remodelling, creating a hidden injury risk environment within training programs.

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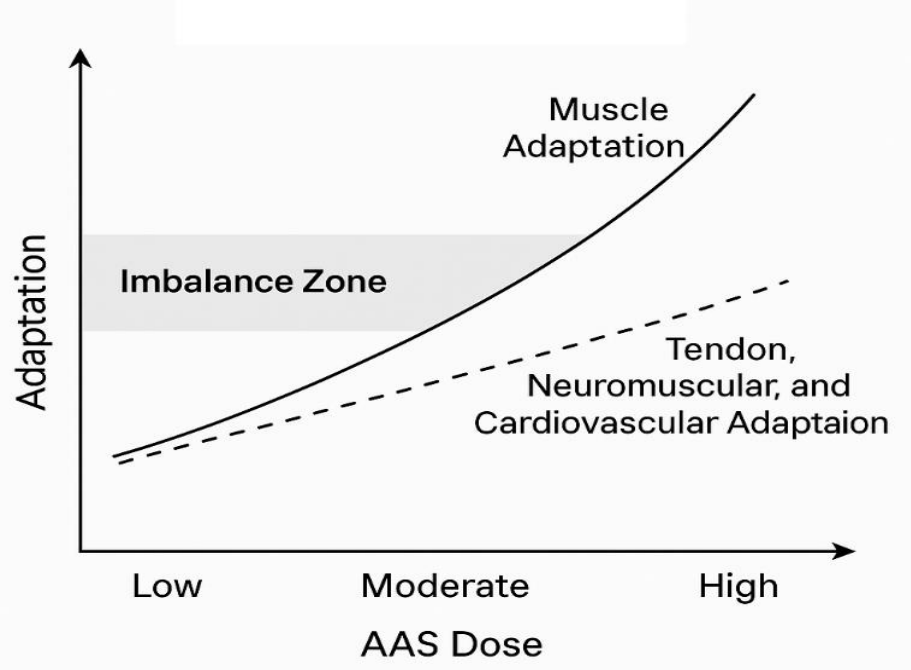


*Figure 3.* ***Dose–Adaptation Imbalance Model***

The figure depicts two adaptation curves plotted against increasing anabolic steroid dose and training exposure. The muscle adaptation curve rises steeply, reflecting documented increases in fat-free mass (+6 kg) and strength (+20–40 kg) at supra physiological doses (~600 mg/week). In contrast, the systemic adaptation curve (representing cardiovascular, endocrine, and connective tissue systems) increases gradually or declines, as evidenced by reduced HDL cholesterol (−40–50%), suppressed endogenous testosterone (−70%), and decreased left ventricular ejection fraction (~52% vs 63% in non-users). The widening gap between these curves represents the imbalance zone, where training loads exceed biological safety limits, resulting in elevated injury risk and long-term health consequences. The figure demonstrates that steroid-related harm is dose-dependent and predictable, rather than incidental. The separation between the muscle and systemic adaptation curves visually defines the imbalance zone, which widens at higher doses and represents increasing divergence between performance gains and biological safety.

Although AAS can significantly improve muscle mass and physical performance, these benefits come at the cost of systemic physiological damage. Cardiovascular complications are among the most dangerous outcomes, often developing silently and manifesting as sudden cardiac events. Hormonal suppression frequently persists even after cessation, indicating long-term endocrine damage.

From a sports education perspective, the normalization of steroid use in gym culture creates misinformation regarding safety. Many users underestimate the chemical potency of these substances and the cumulative effects of prolonged exposure.

**Conclusions:**

Our This study demonstrates that anabolic steroid misuse produces a measurable imbalance between rapid muscle growth and the slower adaptation of connective tissue, neuromuscular control, and cardiovascular function. Through the Dose–Adaptation Imbalance Model, the findings clarify why increases in strength and muscle size at higher steroid doses are frequently followed by injury, impaired recovery, and

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adverse health outcomes. Importantly, the model allows dose-related risk to be understood using existing clinical and sports science data, without the need for unethical experimental exposure.

From an applied perspective, the results indicate that steroid-related complications are dose-dependent and predictable. As chemical dose increases, muscle adaptation outpaces systemic adaptation, creating a high-risk training environment marked by recurrent injuries, reduced training continuity, and long-term physiological strain. Recognizing this imbalance can help sports science and training professionals identify early warning signs, such as delayed recovery, unexplained performance decline, and repeated musculoskeletal injuries.

Overall, the model provides a clear and easy way to understand how chemical dose escalation affects training safety. It can be used by sports science professionals to promote dose awareness, injury prevention, and safer training decisions. The model may also support future research aimed at developing educational and preventive strategies for athletes, bodybuilders, and coaches, with the goal of improving long-term performance sustainability and health.

**Declarations:**

This study is an analytical research paper using secondary quantitative data derived from previously published peer-reviewed studies. No human or animal participants were directly involved, and therefore ethical committee approval was not required.

**Competing interests:**

The authors declare no competing interests.

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