Relations Between Performance Enhancement Drugs and Health-defining Parameters During the Competition Preparation Period of World-class Bodybuilders

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Research Article

Keywords: Fluctuations, performance enhancement drugs, health-defining parameters, competition preparation, world-class bodybuilders

Posted Date: August 6th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-779161/v1>

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Abstract

Fluctuations in biochemical parameters defining cardiovascular, liver and musculoskeletal health were analyzed to evaluate the health status of world class bodybuilders using performance enhancement drugs such as anabolic-androgenic steroids (AAS) [testosterone propionate, drostanolone propionate (Masteron), trenbolone acetate (Finajet), oxandrolone (Anavar), stanozolol (Winstrol) and boldenone undecylenate], human growth hormone (HGH) and “fat-burning drugs” [triiodothyronine (t3), clenbuterol, mesterolone (Proviron), tamoxifen citrate (Nolvadex) and 2,4-Dinitrophenol (2,4-DNP)], and a 6-month preparation period. Changes in serum levels of total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), aspartate aminotransferase (AST), alanine transaminase (ALT), and bilirubin, as well as body mass (BM) and total body fat percentage (BF%), were measured as a function of the competition preparation period. The study shows a continuous increase in BM, accompanied by a decrease in BF% to the physiologically optimal level. TC levels varied significantly within the *desired* concentration range ($TC < 5.18 \text{ mmol / L}$), and TG fluctuated between the ranges of concentrations *borderline high* ($1.69 \text{ mmol / L} \leq TG < 2.26 \text{ mmol / L}$) and *high* ($2.26 \text{ mmol / L} \leq TG < 5.65 \text{ mmol / L}$) concentration ranges. HDL-C levels remained within the *low* range ($TG < 1.03 \text{ mmol / L}$), whereas LDL-C fluctuated across all physiologically important ranges, that is, near-optimal / above *optimal* ($2.59 \text{ mmol / L} \leq LDL-C < 3.34 \text{ mmol / L}$), *borderline high* ($3.35 \text{ mmol / L} \leq LDL-C < 4.11 \text{ mmol / L}$), and *high* ($4.14 \text{ mmol / L} \leq LDL-C < 4.91 \text{ mmol / L}$). AST and ALT concentrations remained within the high-concentration region, i.e., $> 0.58 \text{ mkat / L}$ and $> 0.91 \text{ mkat / L}$. Bilirubin concentration fluctuated within the desirable limits ($3.42 \text{ mmol / L} \leq \text{Bilirubin} < 2.53 \text{ mmol / L}$), and urea concentration were between desirable ($3.2 \text{ mmol / L} \leq \text{Urea} < 7.9 \text{ mmol / L}$) and high values ($\text{Urea} \geq 7.9 \text{ mmol / L}$). Although most of the changes are within physiologically acceptable ranges, there are evident disturbances in LDL-C and Urea levels induced by performance-enhancing drugs. To our knowledge, this study is among the few, if not the only, that discloses the physiological changes associated with doping-assisted preparations for an international bodybuilding contest.

Introduction

A PubMed search for sports doping reports revealed ~ 3000 documents, nearly half of which focused on anabolic androgenic steroids (AAS). AAS are derivatives of the human hormone testosterone and are responsible for stimulating cellular protein biosynthesis¹. Their effectiveness depends on the number of metabolically available nutrients^{2,3} and genetic control of muscle growth⁴. Despite its illegality⁵ and possible health-related risks⁶, AAS helps individuals achieve increased lean body mass and strength⁷ for various recreational and professional sports. In particular, most competitors, besides Olympic and well-paid professionals, use AAS on a trial-and-error basis⁸.

To date, some scientific reports suggest that AAS are responsible for adverse health effects^{9–13}; however, the previous literature review did not confirm this¹⁴. Moreover, Hargens et al.¹⁵, in a study on the effects of AAS on strength-training athletes, pointed to the practical shortcomings of many study methods,

indicating that most focus on athletes using only one or two types of AAS. While such a method is justified from a clinical perspective, it has shortcomings from practical viewpoints and is irrelevant in professional sports practice.

We previously showed ¹⁴ that competitive bodybuilders experiment with various AAS, which, in their judgment, allow them to obtain better musculature and vascularization and concomitantly decrease their level of adipose tissue to a physiological minimum. Thus, a simplistic approach does not provide an accurate picture of the influence of AAS on human physiology in contemporary sports, especially in sports such as bodybuilding, weightlifting, and wrestling.

This study is an explicit analysis of the health-defining parameters of world-class bodybuilders as a function of self-administration doping, including AAS [testosterone propionate, drostanolone propionate (Masteron), trenbolone acetate, oxandrolone (Anavar), stanozolol (Winstrol), and boldenone undecylenate], human growth hormone (HGH) and 'fat burning drugs' [triiodothyronine (t3), clenbuterol, mesterolone (Proviron), tamoxifen citrate (Nolvadex), and 2,4-Dinitrophenol (2,4-DNP)], performed on a group of eight top amateur/professional bodybuilders during the competition preparation period. It is also an attempt to establish the extent to which AAS are used in competitive sports. Because of the lack of readily available scientific reports on the topic, most use YouTube and other unsaturated Internet sources to learn about the topic. Furthermore, some scientific reports also provide misleading information on the physiological implications of AAS without specific evidence. Some also duplicate specific health-related comments without proper substantiation of the given statements. Our study stresses that the data presented in this report should not be associated with any particular bodybuilding organization or sports event. To our knowledge, this study is the first to analyze the competitive use of sports doping and its relationship to basic physiological parameters that define the health of competitors in a real-life scenario.

Methods

The study was carried out by gaining access to competitor notes and medical examination results encompassing the time, amount, and brand of self-administered specific drugs used during 6 months of preparation for international competition.

Study Subjects: All experiments and methods were performed in accordance with the relevant guidelines and regulations. All experimental protocols were approved by the Medical Chamber Licensing Committee, Gdansk, Poland: KB-20/14. Informed consent was obtained from all subjects. The study was conducted with the funds of the participants and the authors. The study was carried out in a group (N = 8) of top European male bodybuilders from different countries and nationalities in Europe who used sports doping drugs during the preparation period for the contest. Due to the extreme difficulty in collecting the data, the presented data was collected with the underlying objective of studying a group of at least five competitors for a comparable competition preparation period. The ages of the study subjects ranged from 30 to 35 years ($M = 32.49$, $SD = 1.47$). Competitors' BM was between 97.2 and 110.4 ($M = 104.4$, SD

= 4.28). Doping information is presented as ranges (minimum-maximum) of specific drug amounts and is shown in Table 1.

An analysis of the competitors' notes exposed a specific diet for the competition preparation period consisting of specific protein-fat-carbohydrate ratios. Thus, until the fifth month of preparation, the dietary regime consisted of six meals between 7:00 and 23:00 containing ~ 40% protein, ~ 20% fat, and ~ 40% carbohydrates. Competitors consumed 3.8–4.2 g of protein/kg of body mass/day, 1–1.2 g of fat/kg of body mass/day, and 3–4 g of carbs/day/kg of body mass. This ratio provided ~ 200% Cal of the calculated basal metabolic rate (BMR) for each competitor. During the final 5 and 6 months of the preparation period, carbohydrate and fat levels gradually decreased to zero, decreasing total caloric consumption to ~ 150% and ~ 100% of BMR, respectively.

Study protocol: In this study, the following biochemical parameters were analyzed: 1) cardiovascular health (serum cholesterol levels [total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL-C) and low-density lipoprotein (LDL-C)]); 2) liver health (aspartate aminotransferase (AST), alanine transaminase (ALT), and bilirubin levels; 3) bone mineral density (BMD); and 4) total body fat (tissue) (BF%).

To avoid direct contact between study competitors and the research team, all subjects were asked to perform a blood biochemical analysis in an accredited laboratory. All laboratory measurements were made in the morning hours after a fast of 12 hours (no food or drink, except water). The analytical procedure provided in the following is derived by the analysis of laboratory-accredited protocols.

4.5 ml of blood was collected in Mint Green Top (Lithium Heparin Gel) and centrifuged within 2 hours from sample collection for 10 minutes at 2800 rpm. Plasma TC level was measured enzymatically^{29,30} in a series of conjugated reactions that hydrolyze cholesterol esters and oxidize the 3-OH group in cholesterol. The product of these reactions, 4-(p-benzoquinone-monoimino)-phenazone, is quantified by measuring the absorbance at $\lambda = 500$ nm. HDL-C cholesterol levels were analyzed using four reaction procedures, resulting in quinone-imine dye, whose concentration is directly proportional to HDL-C levels and measured at $\lambda = 600$ nm. Serum TG levels are measured enzymatically via a series of coupled reactions in which TGs are hydrolyzed to produce glycerol. Glycerol is oxidized to 4-(p-benzoquinone-monoimino)-phenazone, whose absorbance is measured at 500 nm. LDL-C levels were calculated using the following formula: $LDL-C = TC - HDL-C - (TG/5)$. AST and ALT activities were assessed by means of the kinetics of a set of enzymatic reactions in which the final step (i.e., oxidation of NADH to NAD⁺), directly proportional to the activity of ALT or AST, can be measured colorimetrically at $\lambda = 340$ nm.

Standing height was measured using a stadiometer with a fixed vertical backboard and an adjustable headpiece with an accuracy of 0.1 cm. Body mass was determined using a digital weight with an accuracy of 0.1 kg.

For the analysis of bone mineral density (BMD) and body fat percentage (BF%), dual-energy X-ray absorptiometry (DXA), a standard for clinical diagnosis, was used³¹. All BMD and BF% measurements

were performed using the Lunar Prodigy Primo GE Healthcare.

Statistical Analysis: The normality of the distribution of the samples was verified using the Shapiro-Wilk test ³². The equality variances for the response variables as a function of sampling were analyzed using Level's test ³³. In case the Level's test showed variance heterogeneity across all measured parameters, Welch's procedure (Welch ANOVA) was used for a time-dependent analysis^{34,35}. Post hoc analysis was performed using the Games-Howell test for multiple pairwise comparisons with unequal variances ³⁶. The null hypothesis for the statistical tests was verified at $P < 0.05$.

Results

Descriptive statistics including mean, standard deviation (SD), median, interquartile range (IQR), minimum, and maximum values of study parameters are compiled in Table 2.

Table 2

Changes in the health-defining parameters of world-class bodybuilders as a function of the preparation period for competition. Triglyceride levels – TG (mmol / L); Total Cholesterol – TC (mmol / L); High-density Lipoprotein levels – HDL-C (mmol / L); Low-density Lipoprotein levels – LDL-C (mmol / L); Aspartate Aminotransferase – AST (mkat / L); Alanine Transaminase – ALT (mkat / L); Bilirubin (mmol / L); Urea (mmol / L); Total Body Fat Percentage – BF%; Bone Mineral Density – BMD (g / cm³).

	Descriptive statistics	Month: 1 (N = 8)	Month: 2 (N = 8)	Month: 3 (N = 8)	Month: 4 (N = 8)	Month: 5 (N = 8)	Month: 6 (N = 8)
TC	min	3.83	4.37	3.68	3.45	4.16	4.51
	max	4.53	5.03	4.15	3.77	4.5	5.54
	Mean (SD)	4.15 (0.22)	4.64 (0.26)	3.87 (0.17)	3.56 (0.11)	4.34 (0.14)	5.03 (0.36)
	Median (IQR)	4.13 (4.01, 4.29)	4.60 (4.42, 4.84)	3.80 (3.77, 3.99)	3.53 (3.47, 3.60)	4.34 (4.23, 4.46)	5.00 (4.83, 5.26)
TG	min	2.11	2.07	2.08	1.9	2.02	2.06
	max	2.42	2.44	2.28	2.16	2.27	2.38
	Mean (SD)	2.22 (0.11)	2.28 (0.16)	2.20 (0.06)	2.02 (0.08)	2.13 (0.08)	2.21 (0.11)
	Median (IQR)	2.18 (2.13, 2.26)	2.33 (2.12, 2.42)	2.19 (2.18, 2.24)	2.00 (1.99, 2.04)	2.12 (2.10, 2.17)	2.21 (2.13, 2.30)
HDL	min	0.49	0.55	0.58	0.53	0.59	0.56
	max	0.56	0.65	0.7	0.62	0.7	0.64
	Mean (SD)	0.52 (0.02)	0.60 (0.03)	0.63 (0.04)	0.58 (0.03)	0.64 (0.03)	0.60 (0.02)
	Median (IQR)	0.52 (0.51, 0.53)	0.60 (0.60, 0.62)	0.62 (0.61, 0.65)	0.58 (0.57, 0.61)	0.64 (0.61, 0.66)	0.60 (0.59, 0.61)
LDL	min	3.39	3.62	2.95	2.77	3.47	4.08
	max	3.74	4.02	3.7	3.46	3.85	4.95
	Mean (SD)	3.59 (0.12)	3.88 (0.15)	3.37 (0.22)	3.12 (0.23)	3.72 (0.14)	4.47 (0.29)
	Median (IQR)	3.64 (3.52, 3.66)	3.93 (3.77, 4.00)	3.41 (3.31, 3.47)	3.18 (3.00, 3.24)	3.75 (3.63, 3.84)	4.46 (4.34, 4.59)
AST	min	1.38	0.97	1.44	1.7	0.8	1.15
	max	1.73	1.09	1.6	1.9	0.91	1.39

	Descriptive statistics	Month: 1 (N = 8)	Month: 2 (N = 8)	Month: 3 (N = 8)	Month: 4 (N = 8)	Month: 5 (N = 8)	Month: 6 (N = 8)
	Mean (SD)	1.60 (0.12)	1.04 (0.04)	1.50 (0.06)	1.84 (0.07)	0.88 (0.04)	1.27 (0.08)
	Median (IQR)	1.59 (1.55, 1.71)	1.05 (1.03, 1.05)	1.50 (1.46, 1.53)	1.85 (1.83, 1.89)	0.88 (0.86, 0.90)	1.27 (1.21, 1.32)
ALT	min	1.82	1.19	2.04	2.09	1.12	1.44
	max	2.1	1.39	2.35	2.41	1.3	1.68
	Mean (SD)	1.99 (0.09)	1.28 (0.07)	2.18 (0.10)	2.26 (0.11)	1.21 (0.06)	1.58 (0.09)
	Median (IQR)	2.00 (1.94, 2.04)	1.27 (1.23, 1.31)	2.15 (2.12, 2.25)	2.27 (2.18, 2.33)	1.21 (1.18, 1.24)	1.60 (1.51, 1.66)
Bilirubin	min	6.27	6.2	6.83	5.56	7.52	7.07
	max	7.49	7.33	7.75	6.63	9.24	8.26
	Mean (SD)	6.83 (0.44)	6.92 (0.37)	7.29 (0.39)	6.14 (0.40)	8.29 (0.61)	7.84 (0.45)
	Median (IQR)	6.75 (6.55, 7.18)	6.96 (6.81, 7.16)	7.30 (6.90, 7.66)	6.20 (5.95, 6.40)	8.11 (7.94, 8.70)	8.07 (7.48, 8.13)
Urea	min	7.18	6.47	9	9.6	4.85	4.43
	max	8.94	7.46	10.29	10.66	5.82	4.85
	Mean (SD)	8.29 (0.57)	6.99 (0.36)	9.52 (0.40)	10.02 (0.35)	5.40 (0.31)	4.61 (0.14)
	Median (IQR)	8.40 (8.11, 8.59)	7.08 (6.75, 7.22)	9.39 (9.30, 9.68)	10.07 (9.75, 10.16)	5.42 (5.21, 5.63)	4.62 (4.49, 4.67)
BMD	min	1.14	1.28	1.28	1.32	1.38	1.35
	max	1.46	1.51	1.57	1.52	1.5	1.5
	Mean (SD)	1.34 (0.11)	1.40 (0.07)	1.44 (0.09)	1.44 (0.07)	1.43 (0.05)	1.44 (0.05)
	Median (IQR)	1.38 (1.31, 1.41)	1.40 (1.37, 1.44)	1.44 (1.39, 1.49)	1.44 (1.41, 1.49)	1.42 (1.39, 1.45)	1.44 (1.42, 1.48)
BF%	min	6.81	6.10	5.72	4.81	4.07	4.31
	max	8.39	7.35	6.82	6.28	5.63	5.79

Descriptive statistics	Month: 1 (N = 8)	Month: 2 (N = 8)	Month: 3 (N = 8)	Month: 4 (N = 8)	Month: 5 (N = 8)	Month: 6 (N = 8)
Mean (SD)	7.62 (0.48)	6.71 (0.44)	6.50 (0.37)	5.83 (0.44)	4.60 (0.46)	4.77 (0.45)
Median (IQR)	7.49 (7.45, 7.94)	6.56 (6.48, 7.02)	6.59 (6.41, 6.77)	5.94 (5.85, 5.99)	4.48 (4.37, 4.68)	4.70 (4.52, 4.82)

Changes in body mass (BM) and percentage of total body fat (BF%): BM continuously increases during the training period from a mean value of 105 kg to 109 kg (Fig. 1A). Simultaneously, BF% decreases from the percentage that defines athletes ($5 \leq \text{BF\%} < 11$) to the optimal level of body fat ($\text{BF\%} < 5$)³⁷ (Fig. 1B). Since discussing monthly changes in bone mineral density (BMD) is meaningless, we scrutinize only the mean BMD values for the first and final months of the competition preparation period (Table 2). Thus, in the first month, the BMD is equal to $1.34 \pm 0.11 \text{ g / cm}^2$, whereas in the last it equals $1.44 \pm 0.5 \text{ g / cm}^2$.

Changes in Serum Total Cholesterol: Serum TC levels vary significantly within the desirable range (Fig. 2A, Table 2), reaching a minimum in the fourth month and a maximum in the sixth month. In the sixth month of the preparation period, only three of the eight competitors were found to have borderline high levels of TC ($5.18 \text{ mmol / L} < \text{TC} < 6.19 \text{ mmol / L}$)³⁸. In contrast, the values for five competitors fell within the desirable range ($\text{TC} < 5.18 \text{ mmol / L}$)³⁸.

Changes in serum triglycerides: Serum TG concentration fluctuates on the border of borderline high ($1.69 \text{ mmol / L} \leq \text{TG} < 2.26 \text{ mmol / L}$) and high values ($2.26 \leq \text{TG} < 5.65$)³⁸ (Fig. 2B, Table 2).

Changes in Serum High-density lipoprotein cholesterol; HDL-C levels vary significantly during the preparation period (Fig. 2C, Table 2), with three distinct motifs, that is, an increase during months one to two and four to five and a decrease between months five and six. Although HDL-C levels fluctuate substantially, their values remain within the low range concentrations ($\text{TG} < 1.03 \text{ mmol / L}$)³⁸.

Changes in serum low-density lipoprotein cholesterol: LDL-C levels fluctuate within the near-optimal/above optimal ($2.59 \text{ mmol / L} \leq \text{LDL-C} < 3.34 \text{ mmol / L}$), borderline high ($3.35 \text{ mmol / L} \leq \text{LDL-C} < 4.11 \text{ mmol / L}$), and high ($4.14 \text{ mmol / L} \leq \text{LDL-C} < 4.91 \text{ mmol / L}$)³⁸ levels (Fig. 2D, Table 2). It attains a minimum in the fourth month and a maximum in the sixth month of the competition preparation period.

Changes in Aspartate Aminotransferase: AST levels fluctuate during the preparation period and are in the high concentration region ($\text{AST} > 0.58 \text{ mkat / L}$)³⁹, reaching a minimum in the second and fifth months of the preparation period (Fig. 3A, Table 2). AST activity decreases significantly during the first to second and fourth to fifth months and increases during the second to fourth and fifth to sixth months.

Changes in Alanine Aminotransferase: The pattern of changes in ALT is analogous, although more pronounced, to that observed for AST. ALT levels fluctuate during the competition preparation period, adopting values in the high concentration region ($\text{AST} > 0.91 \text{ mkat / L}$)³⁹, reaching a minimum in the second and sixth months (Fig. 3B, Table 2). There is a substantial decrease in ALT activity during the 1st to 2nd and 4th to 5th months interspaced with an increase during the 2nd to 4th and 5th to 6th months of the contest preparation period.

Changes in Bilirubin: Bilirubin concentration fluctuates significantly during the entire preparation period, adopting values within the desirable range ($3.42 \text{ mmol / L} \leq \text{Bilirubin} < 2.53 \text{ mmol / L}$)⁴⁰ (Fig. 3C, Table 2). Bilirubin concentration reaches a minimum in the fourth month and a maximum throughout the fifth and sixth months of the preparation period. A minuscule increase in bilirubin concentration spanning months one to three is followed by a substantial decrease between the third and fourth months and a considerable increase between the fourth and fifth months.

Changes in Urea Concentration: The concentration of urea changes significantly during all competition preparation months from the desirable ($3.2 \text{ mmol / L} \leq \text{Urea} < 7.9 \text{ mmol / L}$) to high values ($\text{Urea} \geq 7.9 \text{ mmol / L}$)⁴⁰, reaching a maximum in the fourth month and a minimum during the fifth and sixth months (Fig. 3D, Table 2).

Discussion

To our knowledge, this is the only report that discloses the extent of doping used in preparation for a bodybuilding competition. Although a general overview of the results presented does not reveal obvious changes that lead to health-related problems, they indicate possible homeostatic disturbances.

The study shows that self-administration of AAS increases serum testosterone levels to concentrations greater than 52.05 nm / L , that is, double that of the analogous 'normal' male age group (20.5 nm/L)^{41–44}.

Administration of AAS administration leads to an increase in BMD and confirms previous studies that revealed similar findings, ie, an increase in BMD in response to testosterone administration^{45,46}. Furthermore, the reported values are significantly higher than those observed for similarly aged European men⁴⁷. BF% decreases in response to drug administration.

An analysis of the literature on testosterone propionate intake reveals that it may cause mild myocyte hypertrophy of the heart muscle¹⁶. In addition, it may be accompanied by myocardial dysfunction and accelerated coronary atherosclerosis¹⁷. This study method cannot confirm or disprove these observations. However, the cross-correlation of our observations with the correlations between coronary heart disease and serum lipid levels⁴⁸ indicates that the development of such a medical condition is probable. We cannot confirm the previous observations that indicate Winstrol²¹, Anavar and other AAS^{49,50} as potent hepatotoxic agents²¹. Although the observed changes in AST, ALT, and bilirubin levels may

indicate a hepatotoxic response, the observed elevation of AST and ALT may also be the product of micromuscular injuries^{51,52} occurring during heavy-load training. Since, at the time of the survey, all competitors had been doping for more than 5 years, the expected changes in AST, ALT, and bilirubin levels should be greater in magnitude and predictably fall into the adversity brackets defined by the 'Common Toxicity Criteria for Adverse Events'⁵³, ie between 1×ULN and 1.5×ULN to 3×ULN to 8×ULN, for AST and ALT, respectively.

We cannot confirm or disprove health-related adversity^{54,55} of boldenone undecylenate, a veterinary steroid known for its propensity to increase body mass and appetite⁵⁶ as well as reproductive function⁵⁴. Furthermore, this study method does not confirm the adverse correlations between T3 and TC, HDL-C, and LDL-C concentrations^{57–59}. Although indirectly, through a decrease in body fat tissue, we confirmed the previous study indicating an increase in lipid metabolism²² attributed to T3 intake. The study also confirms previously reported alterations in AST and ALT levels⁶⁰ as a function of T3 administration.

An animal model study shows that clenbuterol decreases TC levels and leads to fluctuations in TG levels⁶¹. When administered orally, clenbuterol can also affect kidney⁶² and liver²³ functions. Although our study does not confirm these observations, there are noticeable fluctuations in serum lipid levels, indicating impairment of lipid metabolism. Furthermore, we were unable to confirm the reported correlations between AST, ALT, and bilirubin levels in response to oral administration of clenbuterol^{23,24}. However, this report confirms a moderate increase in AST and ALT levels in response to AAS intake, described by previously by Abdulredha²³. In particular, the magnitude of the increase in AST and ALT levels may be masked by the physiological properties of clenbuterol, decreasing AST, ALT and bilirubin levels²⁴.

This study indirectly confirmed reports indicating Proviron as a prominent body mass increasing agent^{25,63}.

Although, Nolvadex, a hepatotoxic⁶⁴ selective estrogen receptor modulator⁶⁵, is a potent drug against gynecomastia²⁶, this study did not confirm Nolvadex-induced hyperlipidemia⁶⁶ and hepatotoxicity⁶⁷. Most likely, it is due to the action of clenbuterol, resulting in decreased levels of hepatotoxicity markers.

However, this report confirms previous findings that HGH increases lean muscle mass and decreases body fat tissue by approximately 2 kg and 0.9 kg²⁷, respectively, in a few weeks. Despite this fact, we could not confirm reports indicating that HGH use/abuse might cause health problems^{68–70}.

A previous study reported that extreme abuse of 2,4-DNP by bodybuilders for fat burning purposes resulted in death when taken in excessive amounts, that is, 3–46 mg of 2,4-DNP per kg of body weight per day^{71 72}. This report shows that competitive bodybuilders consume, on average, between 0.9 mg and 1.9 mg of 2,4-DNP per day/kg of body mass, which amounts to ~ 40% of the lowest lethal dose⁷³.

Conclusions

Some reports indicated that AAS and other sports-doping drugs, such as HGH, clenbuterol, tamoxifen-citrate and 2,4-DNP, are serious health risk factors. However, contrary to this study, they did not reflect real-life scenarios, ie, the administration of sports doping drugs during the preparation of competition. The results presented in this report neither unambiguously confirm nor disprove the observation indicating the detrimental influence of performance enhancement on human health. Although there are evident statistically significant changes in LDL-C and Urea levels induced by performance-enhancing drugs, this study showed that most changes are within physiologically acceptable ranges. However, to uncover the relationships between competitive sport, AAS use and health, more studies reporting 'field-related' data are required.

Declarations

Authors' Contributions

Significant manuscript writers: IZZ and MW. Concept and design: IZZ, MW, BT, MG, LM, RT. Data Analysis and Interpretation: IZZ and MW. Statistical expertise: IZZ. All authors read and approved the final manuscript.

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Table

Due to technical limitations, table 1 docx is only available as a download in the Supplemental Files section.

Figures

Fig 1 A-B

Figure 1

Changes in body mass (BM) and total body fat percentage (BF%), resulting from the administration of sport-doping drugs as a function of a competition preparation period.

Fig 2

Figure 2

Changes in serum levels of A) Total Cholesterol (TC), B) Triglyceride (TG), C) High-density Lipo-protein Cholesterol (HDL-C), and D) Low-density Lipoprotein Cholesterol (LDL-C), rendered by administration of

sport-doping drugs as a function of a competition preparation period. * $p < 0.5$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$.

Fig 3

Figure 3

Changes in serum levels of A) Aspartate Aminotransferase (AST), B) Ala-nine Aminotransferase (ALT), C) Bilirubin, and D) Urea, rendered by administration of sport-doping drugs as a function of the competition preparation period. * $p < 0.5$, ** $p < 0.01$, *** $p < 0.001$, **** $p < 0.0001$.

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