



A Structured Review and Critical Analysis of RCTs on Nandrolone Decanoate's Cardiac Effects in Young Exercising Man

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Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

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ABSTRACT

Aims: The cardiovascular risks associated with anabolic-androgenic steroid (AAS) use, particularly nandrolone decanoate (ND), remain a topic of debate due to conflicting evidence from different types of studies. While observational research frequently reports adverse cardiac effects, findings from randomized controlled trials (RCTs) suggest otherwise. This review aims to critically assess the available RCTs evaluating the impact of ND on cardiac morphology and function, with a focus on their methodological quality and clinical relevance.

Study Design: Structured review of randomized controlled trials.

Methods: A systematic literature search was conducted to identify RCTs that examined the effects of ND on cardiac function. The analysis included study design, sample size, inclusion/exclusion

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criteria, cardiac evaluation methods, and reported outcomes. The methodological quality of each study was assessed using the Jadad scale to ensure a rigorous evaluation of the evidence.

Results: The two reviewed RCTs, which were of moderate to high methodological quality, did not report clinically significant cardiac dysfunction in healthy individuals after short-term exposure to ND. However, these findings contrast with several observational studies, where users—often exposed to uncontrolled conditions and higher doses—frequently present with structural and functional cardiac changes. A key limitation of the RCTs was their small sample sizes, short follow-up durations, and reliance on conventional imaging techniques, which may not be sensitive enough to detect subtle cardiac alterations.

Conclusion: While current RCTs do not provide strong evidence of cardiovascular harm from ND, their methodological limitations underscore the need for further research. Future trials with larger sample sizes, higher statistical power, and more advanced cardiac imaging techniques will be crucial in clarifying the true impact of ND on the heart. Until more definitive data are available, routine cardiac monitoring should be considered for individuals using ND, particularly those on prolonged or high-dose regimens.

Keywords: *Nandrolone decanoate; anabolic steroids; cardiac function; ventricular hypertrophy; cardiovascular risk.*

1. INTRODUCTION

The use of anabolic-androgenic steroids (AAS) has become a widespread global phenomenon, raising significant concerns regarding their potential adverse effects (Sagoe et al., 2014). Among these compounds, nandrolone decanoate (ND) is one of the most used, and frequently linked to cardiovascular risks (Abdullah et al., 2024). Observational studies suggest possible cardiac alterations, such as ventricular hypertrophy and diastolic dysfunction, reinforcing concerns that its use may compromise long-term cardiovascular health (Patanè et al., 2020). However, much of this evidence comes from methodologically weak studies, often lacking proper control of essential variables, making it difficult to draw definitive conclusions (Goldman and Basaria, 2018).

Randomized controlled trials (RCTs), recognized as the gold standard for establishing causality, could provide more reliable insights into the true cardiovascular effects of ND (Goldman and Basaria, 2018). However, the limited RCTs available suffer from small sample sizes, short follow-up periods, and reliance on conventional cardiac assessment methods, which may not be sensitive enough to detect early or subclinical changes (Hartgens et al., 2003; Chung et al., 2007). This knowledge gap fuels ongoing uncertainty about ND's safety, particularly among users exposed to prolonged or high-dose regimens.

Given this scenario, it is imperative to critically evaluate the existing evidence and identify the

limitations of studies conducted thus far. This structured review aims to analyze published RCTs on the cardiovascular effects of ND, assessing their methodological quality and the reliability of their findings. A clearer understanding of ND's actual risks is essential to guide future research, improve clinical safety, and provide more definitive answers regarding its impact on human cardiac morphology and function.

2. METHODS

A structured scientific search (Higgins et al., 2021) was conducted in the PubMed database (www.pubmed.gov) using a combination of MeSH terms and synonymous terms, along with Boolean operators (OR, AND, NOT) to identify specific studies addressing the use of ND and mapping its cardiovascular effects in humans, excluding studies conducted on animal models. Both authors independently screened all studies and reached full agreement on the inclusion of the specific studies identified. The complete search strategy and the results obtained are presented in the Table 1.

3. RESULTS

As seen at the end of the previous section (Table 1), only two randomized studies were identified in the search, Hartgens F, et al. (2003) (Hartgens et al., 2003) and Chung T, et al. (2007), and they will be described in detail.

Table 1. Database, Search Strategy and Results

Database	PubMed
Search Strategy	("Nandrolone"[Mesh] OR (19-Nortestosterone) OR (17beta-Hydroxy-19-Nor-4-Androsten-3-One) OR (17beta Hydroxy 19 Nor 4 Androsten 3 One) OR Estrenolone OR Nortestosterone OR (17-Hydroxy-Estr-4-Ene-3-One) OR Norandrostenolone OR (17 beta Hydroxyestr 4 en 3 one decanoate) OR (19 Nortestosterone Decanoate) OR (19 nor 4 Androstene 17 beta ol 3 one 17 decanoate) OR Retabolil OR Retaboly OR Decadurabolin OR Decadurobolin)) AND ((randomized controlled trial[pt] OR controlled clinical trial[pt] OR randomized[tiab] OR placebo[tiab] OR drug therapy[sh] OR randomly[tiab] OR trial[tiab] OR groups[tiab] NOT (animals [mh] NOT humans [mh])))
Results:	Total: 913 studies
ND + Randomized Controlled Trials + Humans	Selected / included: 2 studies

In the first study, Hartgens F, et al. (2003) published the results of two studies in a single article (Hartgens et al., 2003). The first was an uncontrolled study that evaluated 32 bodybuilders who self-administered AAS, with varying cycles, including differences in doses, combinations, and durations of use. The second study, which will be used for the purposes of this article, was a randomized, placebo-controlled trial that assessed the cardiovascular effects (morphological and functional measures) through two-dimensional Doppler echocardiography in 16 healthy men, before and after the administration of 200 mg/week of ND for 8 weeks (Hartgens et al., 2003).

According to the authors, the measurements performed and detailed in the methodology were: "aortic diameter (AD), left atrium diameter (LA), left ventricular end diastolic diameter (LVEDD), interventricular septum thickness (IVS), posterior wall end diastolic wall thickness (PWEDWT), left ventricular mass (LVM), left ventricular mass index (LVMI), ejection fraction (EF) and right ventricular diameter (RVD). Evaluation of diastolic function was performed by measuring E and A peak velocities and calculation of E/A ratio. In addition, acceleration and deceleration times of the E-top(ATM and DT, respectively) were measured. For evaluation of factors associated with stroke volume the aorta peak flow (AV) and left ventricular ejection times (LVET) were determined".

To minimize potential confounders, all subjects underwent a comprehensive medical examination to assess their health status and to exclude any relevant medical conditions, such as hypertension, diabetes mellitus, liver disease or

abnormal liver enzyme levels, hereditary hypercholesterolemia, elevated serum total cholesterol (>6.5 mmol/L), infertility, and smoking. Additionally, diet, training, and anti-doping controls (to detect the presence of other substances potentially used inadvertently by participants) were rigorously maintained, with no differences observed between the placebo and ND groups throughout the study, as measured at baseline, week four, and week eight. As results, the researchers did not observe any clinically significant morphological or functional changes in any of the echocardiographic parameters assessed (Hartgens et al., 2003).

In a second study, Chung T, et al. (2007) evaluated the comparative effects of testosterone (n = 10) and ND (n = 10) on cardiac function in 30 healthy young men (placebo group: n = 10) in a randomized, double-blind, placebo-controlled clinical trial. Participants received 200 mg/week of testosterone, ND, or placebo for 4 weeks, with detailed assessments and hemodynamic monitoring performed through transthoracic Doppler echocardiography.

As stated by the authors, the measurements conducted and described in the methodology included: "Left ventricular (LV) function (LV ejection fraction, LV modified TEI index), right ventricular (RV) function (ejection area, tricuspid annular systolic planar motion, RV modified TEI index) as well as cardiac afterload (mean arterial pressure, systemic vascular resistance) and overall cardiac contractility (stroke volume, cardiac output)".

Moreover, the sample was carefully selected to avoid potential confounding factors related to

testosterone or ND effects in cardiac function. Thus, the exclusion criteria adopted by the researchers were: (i) contraindications to testosterone administration (breast or prostate cancer); (ii) previous or current use of androgens; (iii) use of disallowed drugs (including illicitly obtained substances) that may interfere with androgen absorption, distribution, metabolism, excretion, or action (e.g., androgens, anti-androgens, finasteride); (iv) any chronic medical condition requiring regular medication or likely to interfere with safe participation; and (v) significant cardiac disease or abnormal baseline echocardiogram (Chung et al., 2007).

The results demonstrated that neither of the two androgens (Testosterone and ND) caused clinically significant changes in cardiac function. The testosterone group showed alterations that, although statistically significant, were minor, affecting diastolic function and the ECG. However, all these changes remained within normal range limits and were not considered clinically relevant by the authors. In contrast, ND did not produce any significant effects on the evaluated cardiac function or ECG (Chung et al., 2007).

The studies cited and their respective results are summarized in Table 2.

The assessment of the methodological quality, by Jadad Scale (Jadad et al., 1996), of the two randomized controlled trials identified through the structured search with a systematic approach in PubMed indicated that they exhibited moderate to high quality (3/5 and 5/5 points). The details are presented in Table 3.

4. DISCUSSION

The main finding observed in this review was that no clinically significant deleterious changes in cardiac morphology or function were found in healthy young individuals in randomized controlled trials assessed with moderate to high methodological quality, following the short-term administration of ND (Hartgens et al., 2003; Chung et al., 2007).

This finding differs from what is generally postulated in the scientific literature (Abdullah et al., 2024), and these discrepancies, between findings from observational studies and randomized controlled trials (RCTs) can, at least to some extent, be attributed to fundamental

methodological differences (Hartgens et al., 2003; Chung et al., 2007; Higgins et al., 2021; Fanaroff et al., 2020). RCTs are conducted in a highly controlled environment, where participants are carefully selected, undergo baseline assessments to exclude preexisting conditions, and have their individual, familial, and lifestyle-related risk factors balanced across groups (Higgins et al., 2021; Fanaroff et al., 2020). Furthermore, in these studies, the substances under investigation are standardized, sourced from the pharmaceutical industry, administered in moderately supraphysiological doses, and used for a relatively short duration (Hartgens et al., 2003; Chung et al., 2007).

In contrast, observational studies often reflect a far more complex and uncontrolled reality. These studies lack randomization, participants may have diverse health backgrounds without a structured baseline assessment, and risk factors are unevenly distributed between groups (Fanaroff et al., 2020; Câmara, 2024). Additionally, these studies frequently involve individuals who use multiple substances simultaneously (abuse of licit and illicit drugs), including drugs obtained from the underground market (adulteration, contamination), often in extremely high doses—sometimes up to 30 times the therapeutic levels—and for prolonged, uninterrupted periods (Câmara, 2024; Câmara, 2024). Such methodological discrepancies must be considered when interpreting findings, as direct extrapolations between these different types of evidence can lead to misinterpretations (Câmara, 2024; Câmara, 2024).

Still within this context of methodological quality and its differences, Goldman A. and Basaria S. (2018) (Goldman and Basaria, 2018) emphasize that the most severe adverse effects, particularly cardiovascular ones, are often reported in studies with methodological limitations that lack the rigor necessary to establish clear causal relationships. Case reports, case series, retrospective case-control studies, cross-sectional analyses, and uncontrolled cohort studies frequently present significant constraints, making their conclusions less reliable (Goldman and Basaria, 2018; Fanaroff et al., 2020). Furthermore, as highlighted by Fanaroff AC et al. (2020), when comparing observational data with randomized clinical trials, the absence of proper randomization can introduce considerable bias, making it difficult to accurately assess the risks and benefits of an intervention.

Table 2. Descriptive characteristics of the studies by Hartgens F, et al. (2007), and Chung T, et al. (2007)

Author Year	Study Type	ND group	ND dose	Duration	Cardiovascular Outcome
Hargens F, et al. 2003	RCT	16 healthy young man	200 mg / week	8 weeks	No clinically significant adverse effects in morphology and function
Chung T, et al. 2007	RCT	10 healthy young man	200 mg / week	4 weeks	No clinically significant adverse effects in morphology and function

Table 3. Jadad Scale Evaluation of the Studies

Criterion	Hartgens F, et al. (2003)	Chung T, et al. (2007)
Study described as randomized	1	1
Randomization method described and appropriate	0	1
Study described as double-blind	1	1
Blinding method described and appropriate	0	1
Description of withdrawals and dropouts	1	1
Total Score	3	5

Specifically, regarding ND, in a comprehensive review conducted by Patanè FG et al. (2020), studies were presented investigating potential mechanisms through which ND abuse could induce cardiovascular damage, ranging from alterations in the cardiac protein expression of myosin isoforms, enzymatic and autonomic changes, to indirect effects such as increased hematocrit, blood pressure, and dyslipidemia. However, the cited references by the authors in the cardiovascular adverse effects section are mostly derived from other review studies, and pathophysiological mechanisms research conducted in animal models, and based on the latter, making direct extrapolation to humans inappropriate, despite being explanatory (Patanè et al., 2020).

In this regard, an important point for reflection is that animal models, frequently employed in the investigation of adverse effects of AAS in various aspects, including, therefore, ND (Ferreira et al., 2025; Shirpoor et al., 2019), play a crucial role in biomedical research, allowing for the exploration of physiological and pathological mechanisms due to their cellular and molecular similarities with humans (Carvalho and Guimarães, 2010; Feijó et al., 2009). However, the direct extrapolation of these findings to human clinical practice must be approached with caution, as

interspecies differences can significantly impact the observed effects (Ritskes-Hoitinga and Pound, 2022; Hartung, 2024). As highlighted in a publication in *Arquivos Brasileiros de Cardiologia*, by Carvalho VO. & Guimarães GV (2010) the physiology of the cardiovascular system in small rodents presents substantial differences compared to humans, which may compromise the applicability of experimental results to clinical practice (Carvalho and Guimarães, 2010).

Having established these essential considerations regarding the discussion of different scenarios—controlled and uncontrolled, human and animal models—and their potential limitations in inferring direct and specific causality of ND in humans, it is also necessary to address some key aspects of the RCTs presented.

In this regard, Kindermann W. and Urhausen A. (2004) highlighted that the study by Hartgens F. et al. (2003) did not assess diastolic function, a crucial parameter for the early detection of pathological myocardial hypertrophy. More recent studies, although observational, suggest that detrimental alterations in both systolic and diastolic function can be found in AAS users and that these changes may become permanent once established, persisting even after

discontinuation of use (Abdullah et al., 2024; Câmara and Viana, 2025).

Furthermore, the randomized clinical trials analyzed relied on conventional echocardiography for cardiac function assessment (Hartgens et al., 2003; Chung et al., 2007)]. However, with advancements in imaging technology, more sensitive and refined methods—such as cardiac magnetic resonance imaging and speckle-tracking analysis—have become available and are increasingly employed in recent observational studies. These techniques provide a more detailed and comprehensive evaluation of cardiac morphological and functional changes, allowing for the early detection of potential adverse effects (Abdullah et al., 2024; Câmara and Viana, 2025).

Speckle tracking and strain imaging, for instance, allow for precise quantification of myocardial deformation in multiple directions, enabling the early detection of subclinical dysfunctions, often before any measurable decline in ejection fraction occurs (Sitia et al., 2010; Brandt et al., 2024). Additionally, cardiac MRI stands out for its superior tissue characterization capabilities, aiding in the differentiation of pathologies and the identification of myocardial fibrosis (Magalhães et al., 2024). These innovations not only deepen our understanding of cardiovascular pathophysiology but also facilitate earlier diagnoses and more informed therapeutic decisions, ultimately improving patient outcomes (Sitia et al., 2010; Brandt et al., 2024; Magalhães et al., 2024).

Another relevant methodological and statistical consideration is the small sample size of both RCTs, which collectively assessed only 26 individuals (Hartgens et al., 2003; Chung et al., 2007). Additionally, only the study by Chung T. et al. (2007) included a sample size calculation and statistical power analysis, with an 80% power to detect an absolute increase or decrease of 6.5%. Therefore, given these potential methodological and statistical limitations, the validity of the findings may be impacted, particularly in detecting early clinical small relevant effects.

Although the study by Hartgens et al. (2003) was conducted as a randomized, double-blind, placebo-controlled trial, certain methodological shortcomings limit its overall robustness. The lack of a detailed description of the randomization process makes it difficult to assess whether an appropriate method was used, leaving room for potential selection bias.

Similarly, while the study was classified as double-blind, it does not specify how blinding was maintained for both participants and investigators, raising concerns about potential performance and detection bias. These methodological gaps resulted in a two-point deduction on the Jadad scale, placing the study within a moderate quality range (Hartgens et al., 2003).

Moreover, other potentially important factors related to cardiovascular outcomes, such as alterations in lipid profile (particularly increased LDL and reduced HDL), hematological changes (elevated hematocrit), autonomic dysfunction, and dysregulation of the renin-angiotensin-aldosterone system (RAAS), were not assessed in these studies (Wenbo and Yan, 2023; Fadah et al., 2023).

Considering all these aspects and controversies, it appears that a knowledge gap persists regarding the specific cardiovascular effects of ND. On the one hand, we have RCTs with methodological limitations—particularly concerning assessment accuracy—that fail to demonstrate clinically significant adverse effects (Hartgens et al., 2003; Chung et al., 2007). On the other hand, we have uncontrolled observational studies conducted in a complex real-world setting, which employed more advanced analytical techniques and identified significant deleterious alterations (Abdullah et al., 2024; Rasmussen et al., 2018; Baggish et al., 2017).

From a therapeutic standpoint, in diseased individuals, multiple RCTs conducted in humans—providing potentially more extrapolable findings than those derived from animal models—have already investigated the use of ND at comparable doses (at least 200 mg/week) for periods between 12 weeks and 12 months (Cattran et al., 1977; Johansen et al., 2006; Gascón et al., 1999; Williams et al., 1974; Navarro et al., 2002). As reported by the authors, although the assessment of cardiac morphology and function was not the specific objective, these studies did not observe increased cardiovascular adverse events (Table 4), even among individuals with higher baseline risk (such as chronic kidney disease patients) (Cattran et al., 1977; Johansen et al., 2006; Gascón et al., 1999; Williams et al., 1974; Navarro et al., 2002), reinforcing and, over a longer duration of use, the non-clinically significant findings of the studies by Hartgens F et al. (2003) and Chung T et al. (2007).

Table 4. Summary of RCT Studies on therapeutic use of ND

Author / Year	Disease	ND Dose	Study duration	Adverse cardiovascular events
Catran et al. (1977)	Uremic anemia	200 mg/week	12 months	No serious events reported
Johansen et al. (2006)	Hemodialysis patients	100 mg (women) / 200 mg (men) per week	12 weeks	No serious events reported
Gascón et al. (1999)	Anemia in elderly hemodialysis patients	200 mg/week	6 months	No serious events reported
Williams et al. (1973)	Anemia in chronic renal failure	200 mg/week	13 weeks	No serious events reported
Navarro et al. (2002)	Patients on peritoneal dialysis	200 mg/week	6 months	No serious events reported

Thus, in our view, well-designed RCTs with appropriate sample size calculations, sufficient statistical power, and the incorporation of more advanced echocardiographic and imaging techniques would be highly valuable and strongly recommended to either corroborate or refute the findings of the two studies reviewed here, regarding the use of ND in healthy young man.

Finally, until more robust, detailed and adequate quality evidence is available regarding the true influence, safety and efficacy, of high doses of ND, it would be prudent to ensure that every user or patient undergoes comprehensive cardiac function and morphology assessment, facilitating the early detection of cardiovascular any damage.

5. CONCLUSION

The available RCTs, conducted with moderate to high methodological quality, did not demonstrate clinically significant adverse effects on cardiac morphology or function in young, healthy individuals following short-term administration of ND (Hartgens et al., 2003; Chung et al., 2007). However, these findings contrast with those from observational studies, which frequently report deleterious cardiovascular changes in chronic AAS users (Abdullah et al., 2024; Rasmussen et al., 2018; Baggish et al., 2017). This discrepancy likely stems from fundamental methodological differences, such as the absence of randomization, heterogeneous participant profiles, and the use of substantially higher doses in uncontrolled settings (Fanaroff et al., 2020; Câmara, 2024).

Despite these differences, it is important to recognize the limitations of the analyzed RCTs, particularly their small sample sizes and reliance on conventional echocardiographic methods,

which may not be sufficiently sensitive to detect early or subtle cardiac changes (Hartgens et al., 2003; Chung et al., 2007). The integration of more advanced imaging techniques, such as cardiac magnetic resonance imaging and speckle-tracking analysis, could enhance the precision of future assessments (Sitia et al., 2010; Brandt et al., 2024; Magalhães et al., 2024). Moreover, while therapeutic clinical trials have suggested the safety of ND at similar doses (200 mg/week) over longer periods (Catran et al., 1977; Johansen et al., 2006; Gascón et al., 1999; Williams et al., 1974; Navarro et al., 2002), extrapolating these findings to recreational users requires caution due to factors such as polypharmacy and individual susceptibilities.

Given these considerations, there is a pressing need for well-designed RCTs with larger sample sizes, appropriate statistical power calculations, and advanced cardiac imaging methodologies to further clarify the cardiovascular effects of ND. Until stronger and more definitive evidence becomes available, it would be prudent to ensure that any individuals using high doses of ND undergo comprehensive cardiac evaluations, facilitating the early detection of potential risks and optimizing long-term cardiovascular monitoring.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

The authors declare that generative AI was used solely during the final stage of manuscript preparation (post-writing) and exclusively for linguistic refinement in the English language (Name: ChatGPT; Version: GPT-4; Model: OpenAI's Large Language Model; Source: OpenAI - <https://openai.com>). No original

text was generated or substantively edited by the AI.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that they have no known competing financial interests OR non-financial interests OR personal relationships that could have appeared to influence the work reported in this paper.

REFERENCES

- Sagoe D, Molde H, Andreassen CS, Torsheim T, Pallesen S. The global epidemiology of anabolic-androgenic steroid use: a meta-analysis and meta-regression analysis. *Ann Epidemiol.* 2014 May;24(5):383-98. doi: 10.1016/j.annepidem.2014.01.009.
- Abdullah R, Bjørnebekk A, Hauger LE, Hullstein IR, Edvardsen T, Haugaa KH, et al. Severe biventricular cardiomyopathy in both current and former long-term users of anabolic-androgenic steroids. *Eur J Prev Cardiol.* 2024 Mar 27;31(5):599-608. doi: 10.1093/eurjpc/zwad362.
- Patanè FG, Liberto A, Maria Maglito AN, Malandrino P, Esposito M, Amico F, et al. Nandrolone decanoate: Use, abuse, and side effects. *Medicina (Kaunas).* 2020 Nov 11;56(11):606. doi: 10.3390/medicina56110606.
- Goldman A, Basaria S. Adverse health effects of androgen use. *Mol Cell Endocrinol.* 2018 Mar 15;464:46-55. doi: 10.1016/j.mce.2017.06.009.
- Hartgens F, Cheriex EC, Kuipers H. Prospective echocardiographic assessment of androgenic-anabolic steroids effects on cardiac structure and function in strength athletes. *Int J Sports Med.* 2003 Jul;24(5):344-51. doi: 10.1055/s-2003-40705.
- Chung T, Kelleher S, Liu PY, Conway AJ, Kritharides L, Handelsman DJ. Effects of testosterone and nandrolone on cardiac function: A randomized, placebo-controlled study. *Clin Endocrinol (Oxf).* 2007 Feb;66(2):235-45. doi: 10.1111/j.1365-2265.2006.02715.x.
- Higgins JP, Thomas J, Chandler J, editors. *Cochrane Handbook for Systematic Reviews of Interventions.* Version 6.2. Cochrane; 2021. <https://training.cochrane.org/handbook>
- Jadad AR, Moore RA, Carroll D, Jenkinson C, Reynolds DJ, Gavaghan DJ, et al. Assessing the quality of reports of randomized clinical trials: Is blinding necessary? *Control Clin Trials.* 1996 Feb;17(1):1-12. doi: 10.1016/0197-2456(95)00134-4.
- Fanaroff AC, Califf RM, Harrington RA, et al. Randomized trials versus common sense and clinical observation: JACC review topic of the week. *J Am Coll Cardiol.* 2020 Aug 4;76(5):580-589. doi: 10.1016/j.jacc.2020.05.069.
- Câmara LC. Possible scenarios of testosterone and anabolic-androgenic steroids use in and outside medicine. *J. Adv. Med. Med. Res.* 2024, 36, 346-352. Doi: 10.9734/jammr/2024/v36i115646.
- Câmara LC. Complexities in assessing health risks of anabolic steroid abuse. *J. Adv. Med. Pharm. Sci.* 2024, 26, 151-153. doi: 10.9734/jamps/2024/v26i12740.
- Ferreira LD, Aguilar BA, Bernal JVM, Melo KY, Gerolim ME, Paixão TEV, et al. Chronic treatment with nandrolone decanoate reduces left ventricular contractile response even when combined with strength training. *Steroids.* 2025 Feb;214:109556. doi: 10.1016/j.steroids.2024.109556.
- Shirpoor A, Heshmatian B, Tofighi A, Eliasabad SN, Kheradmand F, Zerehpooosh M. Nandrolone administration with or without strenuous exercise increases cardiac fatal genes overexpression, calcium/calmodulin-dependent protein kinase II δ , and monoamine oxidase activities, and enhances blood pressure in adult Wistar rats. *Gene.* 2019 May 20;697:131-137. doi: 10.1016/j.gene.2019.02.053.
- Carvalho VO, Guimarães GV. Desafios da ciência translacional. *Arq Bras Cardiol.* 2010;94(1):4-5.
- Feijó A, Cordova FM, Ledur PF, Denardin CC, Dall'Igna DM. Modelos animais na pesquisa biomédica. *Scientia Medica.* 2009;19(1):3-10.
- Ritskes-Hoitinga M, Pound P. The role of systematic reviews in identifying the limitations of preclinical animal research, 2000-2022: Part 2. *J R Soc Med.* 2022 Jun;115(6):231-235. doi: 10.1177/01410768221100970.
- Hartung T. The (misleading) role of animal models in drug development. *Front. Drug Discov.* (4): 2024. doi: 10.3389/fddsv.2024.1355044.

- Kindermann W, Urhausen A. Left ventricular dimensions and function in strength athletes. Re: Hartgens F, Cheriex EC, Kuipers H. Prospective echocardiographic assessment of androgenic-anabolic steroids effects on cardiac structure and function in strength athletes. *Int J Sports Med.* 2004 Apr;25(3):241-2; author reply 243-4. doi: 10.1055/s-2004-817850.
- Câmara LC, Viana DP. Cardiac dysfunction and recovery after anabolic-androgenic steroid abuse: Is reversibility possible? *Cardiol. Angiol. Int. J.* 2025, 14, 13-15. Doi: 10.9734/ca/2025/v14i1466.
- Sitia S, Tomasoni L, Turiel M. Speckle tracking echocardiography: A new approach to myocardial function. *World J Cardiol.* 2010 Jan 26;2(1):1-5. doi: 10.4330/wjc.v2.i1.1.
- Brandt Y, Lubrecht JM, Adriaans BP, Aben JP, Gerretsen SC, Ghossein-Doha C, et al. Quantification of left ventricular myocardial strain: Comparison between MRI tagging, MRI feature tracking, and ultrasound speckle tracking. *NMR Biomed.* 2024 Sep;37(9):e5164. doi: 10.1002/nbm.5164.
- Magalhães TA, Carneiro ACC, Moreira VM, Trad H, Lopes MMU, Cerci RJ, et al. Diretriz de tomografia computadorizada e ressonância magnética cardiovascular da Sociedade Brasileira de Cardiologia e do Colégio Brasileiro de Radiologia – 2024. *Arq Bras Cardiol.* 2024;121(9):e20240608. Doi: 10.5935/abc.2014S006.
- Wenbo Z, Yan Z. The uses of anabolic-androgenic steroids among athletes: Its positive and negative aspects – A literature review. *J Multidiscip Healthc.* 2023 Dec 29;16:4293-4305. doi: 10.2147/JMDH.S439384.
- Fadah K, Gopi G, Lingireddy A, Blumer V, Dewald T, Mentz RJ. Anabolic-androgenic steroids and cardiomyopathy: An update. *Front Cardiovasc Med.* 2023 Jul 26;10:1214374. doi: 10.3389/fcvm.2023.1214374.
- Rasmussen JJ, Schou M, Madsen PL, Selmer C, Johansen ML, Ulriksen PS, et al. Cardiac systolic dysfunction in past illicit users of anabolic-androgenic steroids. *Am Heart J.* 2018 Sep;203:49-56. doi: 10.1016/j.ahj.2018.06.010.
- Baggish AL, Weiner RB, Kanayama G, Hudson JI, Lu MT, Hoffmann U, Pope HG Jr. Cardiovascular toxicity of illicit anabolic-androgenic steroid use. *Circulation.* 2017 May 23;135(21):1991-2002. doi: 10.1161/CIRCULATIONAHA.116.026945.
- Cattran DC, Fenton SS, Wilson DR, Oreopoulos D, Shimizu A, Richardson RM. A controlled trial of nandrolone decanoate in the treatment of uremic anemia. *Kidney Int.* 1977 Dec;12(6):430-7. doi: 10.1038/ki.1977.134.
- Johansen KL, Painter PL, Sakkas GK, Gordon P, Doyle J, Shubert T. Effects of resistance exercise training and nandrolone decanoate on body composition and muscle function among patients who receive hemodialysis: A randomized, controlled trial. *J Am Soc Nephrol.* 2006 Aug;17(8):2307-14. doi: 10.1681/ASN.2006010034.
- Gascón A, Belvis JJ, Berisa F, Iglesias E, Estopiñán V, Teruel JL. Nandrolone decanoate is a good alternative for the treatment of anemia in elderly male patients on hemodialysis. *Geriatr Nephrol Urol.* 1999;9(2):67-72. doi: 10.1023/a:1008306301255.
- Williams JS, Stein JH, Ferris TF. Nandrolone decanoate therapy for patients receiving hemodialysis: A controlled study. *Arch Intern Med.* 1974 Aug;134(2):289-92. doi:10.1001/archinte.1974.00320200099013.
- Navarro JF, Mora C, Macía M, García J. Randomized prospective comparison between erythropoietin and androgens in CAPD patients. *Kidney Int.* 2002 Apr;61(4):1537-44. doi: 10.1046/j.1523-1755.2002.00271.x.

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