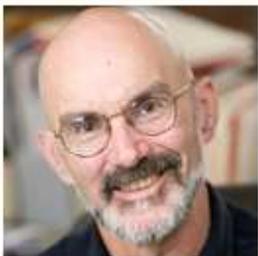


STEROIDS 1 Reference List and Supplemental Information

1:20: "Some of these explanations have been picked up and paraphrased, or otherwise plagiarized, from a researcher at Willamette University named Peter Harmer. Peter tends to be the most articulate courier of reason on this subject and his Australian-accented delivery helps shuttle the message with a bit more finesse."

W I L L A M E T T E  U N I V E R S I T Y



**Peter A. Harmer,
Ph.D., MPH, ATC,
FACSM**

Professor of Exercise
Science, Department
Chair

Human Anatomy,
Sports Medicine,
Epidemiology

Sparks Center 210
(503) 370-6470

Fax: (503) 370-6379

pharmer@willamette.edu

That paragraph contains no exaggeration; it actually undersells Peter. A brief (and totally inadequate) Harmer-history goes something like this:

Before he left Australia, he received his Diploma of Teaching from Riverna College of Advanced Education (1974). His next four degrees came from University of Oregon (B.S., M.S., M.Ed., and Ph.D.) and his MPH came from Oregon Health & Sciences University in 2006.

He's the Chief Medical Officer for the *U.S. Fencing Association*, was appointed to the medical commission of the *Federation Internationale d'Escrime*, and is a Senior Associate Research Scientist of the *Oregon Research Institute*.

He was a stud on a surf board, on a rugby field, and on a Ducati racetrack. He was a judo champion in Japan. He won a U.S. national championship in fencing (Vet 50). And in his free time, he writes grants and papers with his research partner (Fuzhong Li), covering everything from the use of steroids in sport to the risk of falls in the elderly.

The project he's working on currently (scheduled to end August 31, 2016) is a five-year \$3,150,000 grant from the *National Institute on Aging* to translate and implement an effective fall prevention program into a community-based practice (R01 AGO034956-01A1).

Before that, it was a four-year \$1,350,000 grant from the *National Center for Injury Prevention and Control* (R18 CE001723-01) which ended on August 31, 2012.

Before *that*, it was a four-year \$2,795,909 grant from *National Institutes of Health – Behavioral Medicine Interventions and Outcomes* to study the influence of Tai Chi on Parkinson's disease (R01 NS047130-01A2). That project ended on December 31, 2011 and the most recent paper he (along with his research colleagues) published off of it appeared in the *New England Journal of Medicine*.

Many of my arguments on the ethics of steroids in sport were inherited from publications, presentations, and conversations with Peter. Here's one of his recent papers on the subject: Harmer, P. (2010). Anabolic-androgenic steroid use among young male and female athletes: In the game to blame? *British Journal of Sports Medicine*, 44(1), 26-31.

3:05: *“We’ll begin with the most tedious and untenable argument; that there’s no reason to argue because there’s no evidence that steroids even work and thus and no reason to do them.... Traditionally, you either give healthy people useless doses – maybe 100 mg a week – or you work with a pathology like HIV-related wasting. And in that case, you get great results. Or the other option is you look at animal models. Or epidemiological surveillance, where the data come from observation rather than an intervention.”*

The “steroids don’t work” argument comes from articles like this:

0163-769X/88/0902-0181\$02.00/0
Endocrine Reviews
Copyright © 1988 by The Endocrine Society

Vol. 9, No. 2
Printed in U.S.A.

Androgen Abuse by Athletes

JEAN D. WILSON

Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas, Dallas, Texas 75235

Quotations from the article:

“This particular form of drug abuse stems from the convergence of several separate misconceptions. The first was the recognition that the administration of androgens to hypogonadal males causes an increase in nitrogen retention and an increase in muscle mass and lean body weight (5). It followed that the differences in muscle mass between men and women are largely due to differences in testosterone levels, and it was assumed that the administration of androgens in supraphysiological amounts to normal men would do even more than the normal amount.”

“In men with normal levels of plasma androgens the androgen receptor in most tissues appears either to be saturated or downregulated.... Thus, it has not been possible to separate the two types of actions at the pharmacological or physiological levels, and in normal men any anabolic actions obtained from exogenous androgens are inevitably limited in scope.”

“Ziegler assumed that androgens would enhance athletic performance, and he began to experiment in American weight lifters with the various agents that had been developed as candidates for pure anabolic steroids (8). He subsequently concluded that the effects of androgens are purely psychological (9, 10)... Because of secrecy surrounding the practice, a great deal of information about androgen abuse is based on hearsay. However, there can be no doubt that many athletes believed that the androgens do enhance strength, and, as a consequence, the “magic pills,” obtained through legal and illegal sources, began to be used widely, despite the lack of clear evidence that they do in fact improve athletic ability.”

“After more than 30 yr of use it is still not clear whether androgens do, in fact, enhance athletic performance.”

“The data in Table 2 have been separated into two groups: nine studies that failed to demonstrate an increase in muscle strength (13, 70-77) and seven studies in which androgen administration was reported to enhance muscular strength (78-84). Of the seven positive studies two (80, 83) are reported in such a way as to make uncertain the validity of the interpretation; e.g. differences in mean values are slight, and the ranges of variation are not provided so that it is not clear whether the differences are, in fact, significant.”

“In summary, neither enhancement of weight nor improvement in strength can be demonstrated consistently when androgens are administered double blind to athletes. Most reviewers in the field (1, 69, 85-87) have consequently concluded that a positive relationship between androgen use and athletic performance is unproven and that effects on weight and muscle mass are inconsistent.”

This same Jean Wilson went on to write a chapter in the book “Goodman & Gilman’s The Pharmacological Basis of Therapeutics” called “Androgens”. You can find it in the 9th edition (McGraw-Hill), which came out in 1996. In this chapter, he furthers his clinical hunches about the modest effects of testosterone (providing the men taking it have no gonadal obstacles to overcome).

Another article that suggests steroids might not work:

Elashoff, Jacknow, Shain, Braunstein. (1991). Effects of Anabolic-Androgenic Steroids on Muscular Strength. *Annals of Internal Medicine*, 115(5): 387-393.

“Anabolic steroids may slightly enhance muscle strength in previously trained athletes. No firm conclusion is possible concerning the efficacy of anabolic steroids in enhancing overall athletic performance.”

This isn’t where the “steroids don’t do anything” buck stops. The buck has yet to stop. It’s still moving. But it’s not worth further commentary.

The effectiveness of steroids has been demonstrated in animal models for years:

Exner, Staudte, Pette. (1973). Isometric training of rats--effects upon fast and slow muscle and modification by an anabolic hormone (nandrolone decanoate). II. Male rats. *Pflugers Arch.: European Journal of Physiology*, 345(1):15-22.

Lubek. (1984). Contractile responses of rat lateral gastrocnemius and soleus to dianabol (17 beta-hydroxy-17-methyl-1,4-androstadien-3-one) and exercise. *Steroids*, 44(6): 485-495.

Menschikowski, Jung, Junghans, Petzke, Albrecht. (1988). The influence of a steroid hormone and of physical exercise on protein metabolism in rats. *Experimental and Clinical Endocrinology*, 92(3): 341-348.

Joubert & Tobin. (1989). Satellite cell proliferation and increase in the number of myonuclei induced by testosterone in the levator ani muscle of the adult female rat. *Developmental Biology*, 131(2): 550-557.

And in humans, the research isn't particularly slim either, but here are a bunch of articles (at least to get you started) concerning the exact subject I mentioned in the Audible Chapter (muscle wasting in HIV patients):

Testosterone therapy in HIV wasting syndrome: systematic review and meta-analysis

Anthony Kong and Polly Edmonds

Lancet Infect Dis 2002; 2: 692-99

ORIGINAL CONTRIBUTION

Resistance Exercise and Supraphysiologic Androgen Therapy in Eugonadal Men With HIV-Related Weight Loss A Randomized Controlled Trial

Alison Strawford, PhD
Theresa Barbieri
Marta Van Loan, PhD
Elizabeth Parks, PhD
Don Catlin, MD
Norman Barton, MD, PhD
Richard Neese, PhD
Mark Christiansen, MD
Janet King, RD, PhD
Marc K. Hellerstein, MD, PhD

JAMA. 1999;281:1282-1290

See also pp 1275 and 1326.

1282 *JAMA*, April 14, 1999—Vol 281, No. 14

www.jama.com

Author Affiliations are listed at the end of this article.
Corresponding Author and Reprints: Marc K. Hellerstein, MD, PhD, Division of Endocrinology and

Metabolism, Department of Medicine, San Francisco General Hospital, University of California, San Francisco, CA 94110 (e-mail: march@nature.berkeley.edu).

Review

**Expert
Opinion**

informa
healthcare

Therapeutic effects of anabolic androgenic steroids on chronic diseases associated with muscle wasting

Jorn Woerdeman & Willem de Ronde[†]
VU University Medical Center, Department of Endocrinology, Amsterdam, The Netherlands

Expert Opin. Investig. Drugs (2011) 20(1)

Transcriptional Profiling of Testosterone-Regulated Genes in the Skeletal Muscle of Human Immunodeficiency Virus-Infected Men Experiencing Weight Loss

Monty Montano, John N. Flanagan, Lan Jiang, Paola Sebastiani, Matthew Rarick, Nathan K. LeBrasseur, Carl A. Morris, Ravi Jasuja, and Shalender Bhasin

Sections of Infectious Diseases (M.M., M.R.) and Endocrinology, Diabetes, and Nutrition (J.N.F., L.J., N.K.L., C.A.M., R.J., S.B.), Center for HIV-1/AIDS Care and Research (M.M., M.R.), School of Medicine, and Department of Biostatistics (P.S.), School of Public Health, Boston University, Boston, Massachusetts 02118

International Journal of Impotence Research (2003) 15, Suppl 4, S21-S25
© 2003 Nature Publishing Group All rights reserved 0955-9930/03 \$25.00
www.nature.com/ijir



Role of testosterone in maintaining lean body mass and bone density in HIV-infected patients

A Dobs^{1*}

¹Division of Endocrinology & Metabolism, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA
International Journal of Impotence Research (2003) 15, Suppl 4, S21-S25. doi:10.1038/sj.ijir.3901032

Anabolic steroids for the treatment of weight loss in HIV-infected individuals (Review)

Johns KKJ, Beddall MJ, Corrin RC



This is a reprint of a Cochrane review, prepared and maintained by The Cochrane Collaboration and published in *The Cochrane Library* 2009, Issue 1

<http://www.thecochranelibrary.com>



4:03: *“This is where people figure out the ways in which steroids alter one’s chemistry. And that alteration of chemistry is how the steroids actually work. This is what allows us to explain things like that double peak on a bodybuilder’s biceps. There’s no way the pennation angle of the fibers would change to that magnitude without drugs.”*



Pictured to the left is Ronnie Coleman (who I reference at a later point in the Audible Chapter). Ronnie Coleman (born May 13, 1964) won eight Mr. Olympia titles. His last first place finish was in 2005, when he was 41.

The photo says a lot more than my paragraph did. Looking at his physique, the chemistry at play is clearly enhanced by needles. One needs no drug test; “eyeballing it” is sufficient.

Image source:

<http://forums.musculardevelopment.com/showthread.php/6745-Best-Double-Bicep/page5>

4:47: *“When you inject a dose of testosterone into one of your glute-cheeks, it’ll eventually find its way to the target cells, the target muscle cells. It’ll pass the membranes of those cells, and bind to androgen receptors. Unless you’re old and don’t have a very vigorous lifting history. Then you probably don’t have very many receptors. So your body will just have to figure out ways to dispose of all of that excess testosterone. Those ways include converting it into stuff like dihydrotestosterone, which messes up your prostate.”*

This is actually a complicated relationship. It has a lot to do with hormonal ratios, but A) DHT is one of the principle hormones in this ratio, and B) giant doses of steroids don’t *only* affect DHT. Here are a few articles that discuss the hormonal relationships affecting the prostate:

DIHYDROTESTOSTERONE AND THE PROSTATE: THE SCIENTIFIC RATIONALE FOR 5 α -REDUCTASE INHIBITORS IN THE TREATMENT OF BENIGN PROSTATIC HYPERPLASIA

GERALD ANDRIOLE,^{*,†} NICHOLAS BRUCHOVSKY, LELAND W. K. CHUNG,
ALVIN M. MATSUMOTO, ROGER RITTMASER,[‡] CLAUD ROEHRBORN, DAVID RUSSELL
AND DONALD TINDALL[‡]

From the Washington University in St. Louis (GA), St. Louis, Missouri, British Columbia Cancer Agency (NB), Vancouver, British Columbia, Canada, Winship Cancer Institute, Emory University (LWKC), Atlanta, Georgia, University of Washington School of Medicine and Geriatric Research, Education and Clinical Center, Veterans Affairs Puget Sound Health Care System (AMM), Seattle, Washington, GlaxoSmithKline, (RR), Research Triangle Park, North Carolina, University of Texas Southwestern Medical Center at Dallas (CR, DR), Dallas, Texas, and Mayo Clinic College of Medicine (DT), Rochester, Minnesota

EFFICACY AND SAFETY OF A DUAL INHIBITOR OF 5-ALPHA-REDUCTASE TYPES 1 AND 2 (DUTASTERIDE) IN MEN WITH BENIGN PROSTATIC HYPERPLASIA

CLAUS G. ROEHRBORN, PETER BOYLE, J. CURTIS NICKEL, KLAUS HOFNER, AND GERALD ANDRIOLE, ON BEHALF OF THE ARIA3001, ARIA3002, AND ARIA3003 STUDY INVESTIGATORS

UROLOGY **60**: 434–441, 2002.

© 2002, Elsevier Science Inc.

Differentiation; research in biological diversity

Author Manuscript

NIH Public Access

Androgens and estrogens in benign prostatic hyperplasia: past, present and future

Tristan M. Nicholson and William A. Ricke

Androgens and estrogens in benign prostatic hyperplasia: past, present and future

Differentiation. 2011 Nov-Dec; 82(4-5): 184–189.

5:47: *“With a cycle of steroids, you see increases in the number of myonuclei, and through that, the possibility of protein accretion through a larger domain. In rats, this elevated number appears to go away after a while. It might only last a couple weeks, depending on a lot... among us upright folk, inclined to play (or otherwise appreciate) football, the myonucleic perks appear to last many more moons. And this is one explanation of where “muscle memory” comes from. And it makes the thirty-day penalty seem as arbitrary as every other steroid law, rule, or policy.”*

The narrative is told with the summation of available literature. Studies to be summed span the next three pages:

Testosterone-induced increase in muscle size in healthy young men is associated with muscle fiber hypertrophy

INDRANI SINHA-HIKIM,¹ JORGE ARTAZA,¹ LINDA WOODHOUSE,¹
NESTOR GONZALEZ-CADAVID,¹ ATAM B. SINGH,¹ MARTIN I. LEE,¹ THOMAS W. STORER,¹
RICHARD CASABURI,² RUOQUING SHEN,¹ AND SHALENDER BHASIN¹
¹*Division of Endocrinology, Metabolism, and Molecular Medicine, Charles R. Drew
University of Medicine and Science, Los Angeles, 90059; and* ²*Division of Respiratory
and Critical Care Physiology and Medicine, Harbor-University of California
at Los Angeles Medical Center, Torrance, California 90509*

Received 7 November 2001; accepted in final form 10 March 2002

Am J Physiol Endocrinol Metab 283: E154–E164, 2002.
First published March 12, 2002; 10.1152/ajpendo.00502.2001.

Address for reprint requests and other correspondence: S. Bhasin, UCLA School of Medicine, Div. of Endocrinology, Metabolism, and Molecular Medicine, Charles R. Drew Univ. of Medicine and Science, 1731 E. 120th St., Los Angeles, CA 90059 (E-mail: sbhasin@ucla.edu).

0193-1849/02 \$5.00 Copyright © 2002 the American Physiological Society

<http://www.ajpendo.org>

Journal of Public Health and Biological Sciences
ISSN 2305-8668

Vol. 1, No. 4 Oct–Dec 2012, p.134-142
URL: <http://www.jphbs.com>

Review Article

ADDITION OF NEW MYONUCLEI IS A PRE-REQUISITE FOR SKELETAL MUSCLE GROWTH

Muhammad Mustafa Qamar^{*1}, Ayesha Basharat²

¹*Department of Exercise Physiology and Sports Medicine, Orebro University, Sweden;* ²*Department of
Physiotherapy, University of Sargodha, Pakistan*

*Corresponding author: Muhammad Mustafa Qamar (dearqamar@hotmail.com), Department of Exercise Physiology and Sports Medicine, Orebro University, Sweden.



Myonuclei acquired by overload exercise precede hypertrophy and are not lost on detraining

J. C. Bruusgaard, I. B. Johansen, I. M. Egner, Z. A. Rana, and K. Gundersen¹

Department of Molecular Biosciences, University of Oslo, 0371 Oslo, Norway

Edited by Gerald D. Fischbach, The Simons Foundation, New York, NY, and approved July 16, 2010 (received for review December 4, 2009)

Author contributions: J.C.B. and K.G. designed research; J.C.B., I.B.J., I.M.E., Z.A.R., and K.G. performed research; J.C.B., I.B.J., I.M.E., and K.G. analyzed data; and J.C.B., I.B.J., I.M.E., and K.G. wrote the paper.

www.pnas.org/cgi/doi/10.1073/pnas.0913935107

PNAS Early Edition

Age-related differences in apoptosis with disuse atrophy in soleus muscle

Christiaan Leeuwenburgh,¹ Cathy M. Gurley,² Beau A. Strotman,² and Esther E. Dupont-Versteegden^{2,3}

¹Biochemistry of Aging Laboratory and Department of Aging and Geriatric Research, University of Florida, Gainesville, Florida; and Departments of ²Geriatrics and ³Physiology and Biophysics, University of Arkansas for Medical Sciences, Little Rock, Arkansas

Submitted 23 August 2004; accepted in final form 10 January 2005

Address for reprint requests and other correspondence: E. E. Dupont-Versteegden, Dept. of Geriatrics, Univ. of Arkansas for Medical Sciences, 4301 West Markham #807, Little Rock, AR 72205 (E-mail: dupontesthere@uams.edu).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

R1288

0363-6119/05 \$8.00 Copyright © 2005 the American Physiological Society

<http://www.ajpregu.org>

Myonuclear number and myosin heavy chain expression in rat soleus single muscle fibers after spaceflight

D. L. ALLEN, W. YASUI, T. TANAKA, Y. OHIRA, S. NAGAOKA,
C. SEKIGUCHI, W. E. HINDS, R. R. ROY, AND V. R. EDGERTON

Department of Physiological Science, University of California, Los Angeles 90095-1527; Brain Research Institute, University of California, Los Angeles 90095-1726; Space Life Science Payload Office, National Aeronautics and Space Administration Ames Research Center, Moffett Field, California 94035-1000; Department of Physiology and Biomechanics, National Institute of Fitness and Sports, Kanoya City, Kagoshima 891-23; and Space Experiment Group, National Space Development Agency of Japan, Tsukuba City 305, Japan

J Appl Physiol. (1996). 81: 145-151.

Activated satellite cells fail to restore myonuclear number in spinal cord transected and exercised rats

ESTHER E. DUPONT-VERSTEEGDEN,¹ RENÉ J. L. MURPHY,² JOHN D. HOULÉ,²
CATHY M. GURLEY,¹ AND CHARLOTTE A. PETERSON¹

Departments of ¹Geriatrics and ²Anatomy, University of Arkansas for Medical Sciences, and Central Arkansas Veterans Health Care System, Little Rock, Arkansas 72205

Am J Physiol Cell Physiol. (1999). 277:C589-C597.

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

0363-6143/99 \$5.00 Copyright © 1999 the American Physiological Society

Maintenance of myonuclear domain size in rat soleus after overload and growth hormone/IGF-I treatment

G. E. McCALL,¹ D. L. ALLEN,¹ J. K. LINDERMAN,³ R. E. GRINDELAND,³

R. R. ROY,² V. R. MUKKU,⁴ AND V. R. EDGERTON^{1,2}

¹Department of Physiological Science and ²Brain Research Institute, University of California, Los Angeles 90095; ³National Aeronautics and Space Administration Ames Research Center, Moffett Field 94035; and ⁴Genentech, South San Francisco, California 94080

J Appl Physiol. (1998). 84:1407-1412

8750-7587/98 \$5.00 Copyright © 1998 the American Physiological Society

<http://www.jap.org>

And lastly, the drug testing policies/penalties for the major American sports are described here (<http://www.csnbayarea.com/blog/biz-ball/sports-drug-testing-policies-nfl-nba-nhl-olympics>):



CSNBAYAREA.COM

Sports drug testing policies: NFL, NBA, NHL, Olympics

January 9, 2013, 1:15 pm

PENALTIES -- NFL

First: A four game suspension without pay.

Second incident: Eight game suspension, no salary.

Third: One year suspension, no pay.



7:15: *"Bulgaria's reputation for mothering hulking, veiny weightlifters is partly a vestige of the 1950's when steroids were still legal in the Olympic Games. They weren't banned until the sixties. And technically the first Olympic athlete to test positive for a banned substance was not a Bulgarian weightlifter, but a Swedish pentathlete who was stripped of his Bronze due to... alcohol. But before the International Olympic Committee announced their ban on performance enhancing drugs, like steroids and alcohol, researchers did a fair amount of testing. They wanted to know what kinds of effects these drugs had on the body."*

"A Brief History of Anti-Doping" by the World Anti-Doping Agency:

<http://www.wada-ama.org/en/about-wada/history/>

In Mexico City, 1968, Hans-Gunnar Liljenwall drank two beers before the shooting event:

<http://www.sok.se/inenglish/mexicocity1968.4.18ea16851076df63622800011056.html>

8:28: *“The East Germans are another great example. In 1972, the women won five total medals; no golds, definitely no records. They weren’t good. Four years of drugs later, those women won eighteen medals, eleven of them gold, and set eight world records. There were only twelve individual events that year. They won gold in ten of them. And at the end of those races, you’d see the wet champ hoist one of her arms in the air, clenching a fist into the international gesture of victory. And beneath that fist was a giant man arm. Then the owner of that man arm would climb out of the pool and start celebrating with a booming man voice. When those voices were called into question, one of the East German coaches dismissed that question with “we came here to swim, not sing.”*

The “We came here to swim, not sing” comment was discussed in an article in *The New York Times* on December 3rd, 1991 titled “OLYMPICS; Coaches Concede That Steroids Fueled East Germany’s Success in Swimming.” And here is a slightly more recent article that discusses the episode in more detail:

Clinical Chemistry 43:7
1262–1279 (1997)

Doping in Sports
Symposium

Hormonal doping and androgenization of athletes: a secret program of the German Democratic Republic government

WERNER W. FRANKE^{1*} and BRIGITTE BERENDONK²

12:51: *“The rational question is: what are the risks associated with appropriate doses of steroids? A handful of Peeps. The first risks we can eliminate are gender changes and the necessitation of catheters. That won’t happen if you aren’t being ridiculous with your dose. You might see some side effects like pimples and mood changes. But who cares? Let’s be honest: if you had a multi-million dollar contract on the line, would a couple temporary back zits really weigh into your decision? The question itself is sufficient as an answer.”*

There’s plenty of bad information on this subject. Here are a couple mediocre places to start:

Review

Cell
PRESS

The *Sturm und Drang* of anabolic steroid use: angst, anxiety, and aggression

Joseph G. Oberlander and Leslie P. Henderson

Department of Physiology and Neurobiology, Dartmouth Medical School, Hanover, NH 03755, USA

Review

Trends in Neurosciences June 2012, Vol. 35, No. 6

Effects of Androgenic-Anabolic Steroids in Athletes

Fred Hartgens¹ and Harm Kuipers²

- 1 Department of Surgery, Outpatient Clinic Sports Medicine, University Hospital Maastricht, and Sports Medicine Center Maastricht, Maastricht, The Netherlands
- 2 Department of Movement Sciences, Maastricht University, Maastricht, The Netherlands

13:54: *“Because your liver is what controls all of your lipid levels (HDLs, LDLs, triglycerides, that stuff), the first pass portal metabolism of oral anabolic steroids will affect your profile. You see the same effect when women take estrogen orally. But those women can bypass these changes if they take their estrogen by other means; injections or patches or vaginal suppository. Likewise, you’ll experience the lipid-compromising (mostly HDL-lowering) effects if you eat stanozolol (the Ben Johnson drug), but if you shut your mouth and go for the needle, you can bypass a lot of that.”*

Cholesterol levels are still altered by steroids, but the severity is bypassed when that first pass hepatic metabolism is bypassed. Harm Kuipers (a Dutch physiologist with an M.D. and a Ph.D.) discusses this (and other liver issues) in his chapter “Anabolic Steroids: Side Effects”, which appears in the online resource *Encyclopedia of Sports Medicine and Science* (1998). The “gist” is captured by this quotation:

“Anabolic steroids may exert a profound adverse effect on the liver. This is particularly true for orally administered anabolic steroids. The parenterally administered anabolic steroids seem to have less serious effects on the liver. Testosterone cypionate, testosterone enanthate and other injectable anabolic steroids seem to have little adverse effects on the liver.”

The entire encyclopedia is available online: <http://www.sportsci.org/encyc/index.html>

To be more specific, what I compared in my Audible Science Chapter was the oral drug stanozolol to injectable steroids. The study that made this exact comparison was done by Paul Thompson.

As I’m writing this, Paul is the current Director of Cardiology at Hartford Hospital in Connecticut. In 1972, he qualified for the Olympic Marathon Trials in Eugene, Oregon. Four years later, he finished 16th in the Boston Marathon. In 1988, Paul was the Sports Medicine Analyst for the Olympic Games in Seoul (i.e., the Ben Johnson games). He’s been a guest on *Good Morning America* nine times, and in 2012, in the most trivial event of his career, he let me analyze one of his databases (STOMP: NIH R01HL081893-01A2) to write an abstract for ACSM (by extension of this permission, he was one of my coauthors).¹

¹ It was a ridiculously boring abstract. I presented it in Providence (slide show) on November 9th, 2012, and in Indianapolis (poster) on May 31st, 2013. It appears in *MSSE*, issue 45(5), page S394. It’s available online here: <http://acsmannualmeeting.org/wp-content/uploads/2013/04/Friday-Abstracts.pdf>

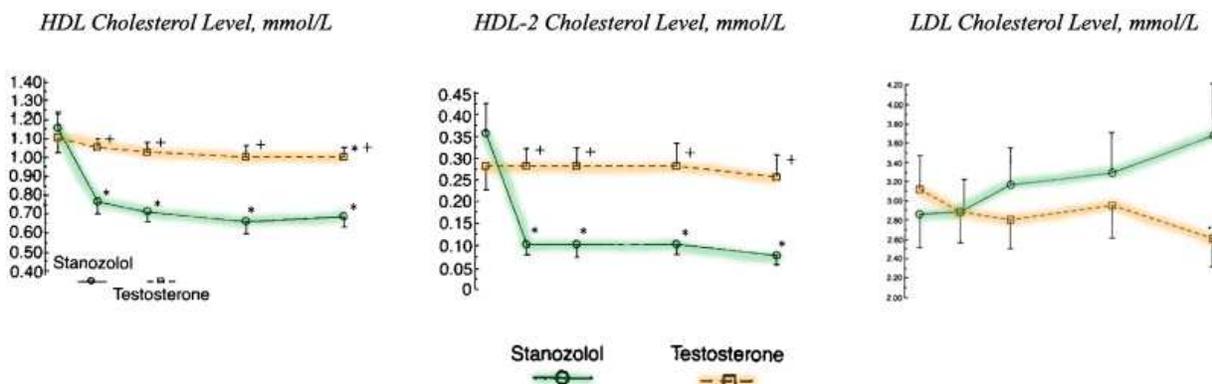
The paper in which Paul compared the serum effects of testosterone to stanozolol is this:

Contrasting Effects of Testosterone and Stanozolol on Serum Lipoprotein Levels

Paul D. Thompson, MD; Eileen M. Cullinane; Stanley P. Sady, PhD; Claire Chenevert; Ann L. Saritelli; Mina A. Sady; Peter N. Herbert, MD

Oral anabolic steroids produce striking reductions in serum concentrations of high-density lipoprotein (HDL) cholesterol. We hypothesized that this effect related to their route of administration and was unrelated to their androgenic potency. We administered oral stanozolol (6 mg/d) or supraphysiological doses of intramuscular testosterone enanthate (200 mg/wk) to 11 male weight lifters for six weeks in a crossover design. Stanozolol reduced HDL-cholesterol and the HDL₂ subfraction by 33% and 71%, respectively. In contrast, testosterone decreased HDL-cholesterol concentration by only 9% and the decrease was in the HDL₃ subfraction. Apolipoprotein A-I level decreased 40% during stanozolol but only 8% during testosterone treatment. The low-density lipoprotein cholesterol concentration increased 29% with stanozolol and decreased 16% with testosterone treatment. Stanozolol, moreover, increased postheparin hepatic triglyceride lipase activity by 123%, whereas the maximum change during testosterone therapy (+25%) was not significant. Weight gain was similar with both drugs, but testosterone was more effective in suppressing gonadotropic hormones. We conclude that the undesirable lipoprotein effects of 17- α -alkylated steroids given orally are different from those of parenteral testosterone and that the latter may be preferable in many clinical situations.

(*JAMA*. 1989;261:1165-1168)



In the “Comment” section of the paper, Paul and his colleagues write: “Oral administration of stanozolol reduced HDL-cholesterol concentrations 33% after only one week of treatment. Testosterone, in contrast, induced comparably little change.”

As I mentioned in the Audible Science Chapter, you see the same issues with estrogen (although the lipoprotein changes occur in the opposite direction; estrogen causes HDL to go up, not down). Like steroids, if you bypass the first pass portal circulation, you reduce the magnitude of these changes.

The article to the right is an editorial that discusses this issue (and other related estrogen-HDL topics).

Although the title is quite bad², the paper illustrates what needs illustrating: oral estrogen affects the HDL profile in ways that other modes of administration do not.

Thompson’s JAMA paper also discusses estrogen:

“Estrogens may also modulate plasma lipoprotein levels but the effects depend on the route of administration. Transdermal estrogens may not change levels of plasma lipids and other plasma proteins whereas oral estrogens increase concentrations of HDL cholesterol, renin substrate, and several other hepatic proteins.”

14:26: *“Another common concern is the remodeling of the heart one sees with prolonged steroid exposure. More specifically, left ventricular hypertrophy... With steroid use, you do see a slight thickening of the wall. This results in the dimensions of the pumping chamber becoming a bit narrower... And while this could be problematic, you’d have to do an awful lot of drugs to get a meaningful effect. Plus, if you adjust for whole body muscle mass, the heart isn’t that much bigger. What this means is that, if all you did was wake up in the morning with another 15lb of lean body mass, having no idea where it came from, but knowing it didn’t come from drugs, your heart would have to remodel itself in order to compensate for that. And once it did, it wouldn’t look that much different from the steroid heart. Steroid hearts are thicker, yes. But so is your entire skeletal muscle system. That creates a need for additional circulation. Even without the steroids, your heart will adapt to accommodate. So lift weights without steroids (or grow for any other reason, like puberty) and you’re going to see a similar effect on the heart. You can exacerbate this with steroids, but the effect is just not that enormous. Especially when it gets compared to actual hypertrophic cardiomyopathy, which is a genetic condition that really thickens the heart.”*



Arteriosclerosis, Thrombosis, and Vascular Biology

JOURNAL OF THE AMERICAN HEART ASSOCIATION

Estrogen and HDL : All that Glitters Is not Gold
David M. Herrington and John S. Parks

Arterioscler Thromb Vasc Biol. 2004;24:1741-1742
doi: 10.1161/01.ATV.0000142357.90351.92

Copyright © 2004 American Heart Association, Inc. All rights reserved.
Print ISSN: 1079-5642. Online ISSN: 1524-4636

² “All that Glitters Is not Gold” is not just a painful cliché, but an abused derivation of Shakespeare’s line “All that glitters is not gold”. The original “glitters” line appears in *The Merchant of Venice*, Act II, scene VII.

The only article one really needs to be consulted came out in 1992 by... Paul Thompson (back in his Brown University days). This:

JACC Vol. 19, No. 2
February 1992:278-82

Left Ventricular Function Is Not Impaired in Weight-Lifters Who Use Anabolic Steroids

PAUL D. THOMPSON, MD, FACC, ARA SADANIANTZ, MD, FACC,
EILEEN M. CULLINANE, MS, KURT S. BODZIONY, MS,* DON H. CATLIN, MD,†
GEORGE TOREK-BOTH, PhD,† PAMELA S. DOUGLAS, MD, FACC‡

Providence and Kingston, Rhode Island; Los Angeles, California; Boston, Massachusetts

Recent reports suggest that anabolic steroid use might deleteriously affect left ventricular function. To examine this possibility, the present study measured left ventricular size and function with use of Doppler echocardiographic techniques in 23 weight lifters: 12 who were currently using anabolic steroids and 11 who reported that they had never used these drugs. Drug users had administered anabolic steroids to themselves for at least three cycles over the past year. All studies were interpreted by blind review and group assignment was confirmed by urine testing. Average age, years of exercise training and body weight, as well as heart rate and blood pressure at rest were similar in both groups.

Cardiac dimensions (mean \pm SD) including left ventricular diastolic cavity diameter (57 ± 3 vs. 56 ± 5 mm), septal thickness (10 ± 2 vs. 9 ± 1 mm), posterior wall thickness (8 ± 1 vs. 8 ± 1 mm) and myocardial mass (149 ± 27 vs. 135 ± 21 g) did not

differ between the anabolic steroid users and nonusers, respectively. Left ventricular systolic and diastolic function at rest were also similar in the users and nonusers: left ventricular fractional shortening ($38 \pm 5\%$ vs. $41 \pm 5\%$); peak rate of wall thickening (4.3 ± 1.1 vs. 4 ± 1.1 s^{-1}) and thinning (-5.9 ± 1.6 vs. -5.6 ± 2.1 s^{-1}); left ventricular filling rate (3.1 ± 0.6 vs. 3.3 ± 0.5 s^{-1}), as well as early (81 ± 12 vs. 83 ± 12 cm/s) and atrial maximal inflow velocities (41 ± 6 vs. 41 ± 9 cm/s) and their ratio (2.01 ± 0.49 vs. 2.16 ± 0.67) were not different between groups.

These results suggest that anabolic steroid use was not associated with left ventricular hypertrophy or clinically detectable systolic and diastolic dysfunction in a small sample of weight lifters who were using these drugs.

(J Am Coll Cardiol 1992;19:278-82)

In case you think Paul is biased (he's not, but if you think he is), here are some articles by other people that cover the same points:

0195-9131/85/1705-0701\$2.00/0
MEDICINE AND SCIENCE IN SPORTS AND EXERCISE
Copyright © 1985 by the American College of Sports Medicine

Vol. 17, No. 6
Printed in U.S.A.

Left ventricular size and function in body builders using anabolic steroids

RICHARD C. SALKE, THOMAS W. ROWLAND, and
EDMUND J. BURKE

*Department of Movement Science, Springfield College, and the
Department of Pediatrics, Baystate Medical Center,
Springfield, MA 01199*

In this study, Salke and colleagues took fifteen steroid-using bodybuilders, fifteen steroid *non-using* bodybuilders, and fifteen non-steroid-using, non-exercising non-bodybuilders.

I quote from the Methods:

“The total amount of anabolic steroids reportedly used by the athletes was very large, amounting to 10 to 20 times that which would be normally recommended by a pharmaceutical manufacturer.”

I quote from the Results:

“All subjects demonstrated a normal cardiac examination... There were no significant electrocardiographic differences in mean resting pulse, intervals, axes, or precordial voltages among the three groups. Left ventricular hypertrophy as defined as an R-wave greater than 20 mm in lead V₆ was identified in one steroid user, one weight training non-steroid user, and two controls. No ST-T wave changes were observed. No significant echocardiographic difference in either absolute or size-relative left ventricular dimensions and functions were observed between the steroid and non-steroid weight training subjects. Dissimilarities between these two groups and control individuals were discovered in absolute ventricular septal thickness and left ventricular wall size, but when these measurements were related to body surface area, only septal width remained significantly greater in both groups of weight lifters.”

I quote from the Discussion:

“The finding that standardization of left ventricular wall thickness to body size showed no difference from inactive controls supports similar results reported by Longhurst et al. (8). In that study augmented left ventricular mass in competitive weight lifters was not increased in relation to lean body mass. These findings thus support the concept that increased myocardial mass observed with weight training parallels gains in skeletal muscle bulk.” And in a concluding paragraph of the conclusion: *“This study revealed no echocardiographic evidence that anabolic steroids potentiate the myocardial responses to weight training. Similar absolute or size-relative left ventricular chamber dimensions, septal or free wall thickness, septal:free wall ratio, contractility, and mass/volume relationships were observed in body builders taking high-dose steroids compared to those training without these drugs. Likewise, no differences in electrocardiogram findings were observed between the two groups. By inference, corollaries may be drawn that 1) anabolic steroids do not augment protein synthesis in the cardiac muscle of healthy individuals, and 2) acute systemic blood pressure elevations with static exercise are not affected by the drugs.”*



Echocardiographic Findings in Power Athletes Abusing Anabolic Androgenic Steroids

Behzad Hajimoradi, MD and Hashem Kazerani, MD

Asian J Sports Med. 2013 March; 4(1): 10-14.

This next study (seen to the left) was effectively the same experiment as Salke et al.'s, but done eighteen years later. These researchers took fifteen bodybuilders who had a couple-year history of steroid use, fifteen bodybuilders not on steroids, and fifteen controls.

And to these forty-five bodybuilders and non-bodybuilders, they did a bunch of cardiovascular tests. A quotation from the results:

“There was no significant difference in left ventricular size or function either systolic or diastolic in comparison to cases and control groups. The only difference was in diastolic size of septum and free wall but observed differences were only significant (P = 0.05) between first (athletic with AAS abuser) and third group (non athletic and nonuser). The difference between the above-mentioned indexes were not significant between two groups of athletes.”

A quotation from the discussion:

“In the current study there was not any statistically significant difference in LV systolic and diastolic dimensions between cases and control groups. Systolic and diastolic function in all groups was relatively similar and it is suggestive of no effect, or minimal effect of chronic anabolic steroid abuse on size, function and stiffness of the heart.”

Entire conclusion:

“AAS abuse has no effect on systolic or diastolic function and LV size of the heart, and just may lead to accentuation of physiologic hypertrophic response to weight training sports.”

These aren't the only articles that support my point. I just chose them so I could show that the information is neither new nor outdated. It should be said that there are some articles that state the opposite (stating that there *are* structural changes to the heart that result from steroid use). However, after going through the hide-and-seek effort of finding them, it's rarely worth the effort; the results aren't all that interesting or meaningful.³ In contrast, here are a couple more studies that failed to find a significant difference between bodybuilders taking drugs and bodybuilders just being bodybuilders:

Urhausen A, Holpes R, Kindermann W. (1989). One- and two-dimensional echocardiography in bodybuilders using anabolic steroids. *European Journal of Applied Physiology and Occupational Physiology*, 58(6): 633-640.

Palatini P, Giada F, Garavelli G, Sinisi F, Mario L, Michieletto M, Baldo-Enze G. (1996). Cardiovascular effects of anabolic steroids in weight-trained subjects. *Journal of Clinical Pharmacology*, 36(12): 1132-1140.

Now if you're a bodybuilder with about nine years of monstrous doses, you might be more vulnerable to this side effect. Aaron Baggish, a physician at Mass. General, looked at a group of weight lifters who met that description (nine monstrous years) and found that their hearts didn't squeeze as well. They had compromised left ventricular function. Baggish published his findings in *Circulation* in 2010.

³ See Di Bello et al.'s article "Effects of anabolic-androgenic steroids on weight-lifters' myocardium: an ultrasonic videodensitometric study" which appeared in *MSSE* in 1999 or Papamitsou et al.'s article "Testosterone-induced hypertrophy, fibrosis and apoptosis of cardiac cells—an ultrastructural immunohistochemical study" from *Medical Science Monitor* in 2011, where they pumped rats full of testosterone or saline and then removed and inspected their hearts)

In that same issue, Paul Thompson published an editorial, providing commentary on those findings. In that commentary: "The AAS users were remarkable for both their steroid dose and duration of use."

Both articles (Baggish's and Thompson's) appear below:

Original Articles

Long-Term Anabolic-Androgenic Steroid Use Is Associated With Left Ventricular Dysfunction

Aaron L. Baggish, MD; Rory B. Weiner, MD; Gen Kanayama, MD, PhD; James I. Hudson, MD, ScD; Michael H. Picard, MD; Adolph M. Hutter, Jr, MD; Harrison G. Pope, Jr, MD

Background—Although illicit anabolic-androgenic steroid (AAS) use is widespread, the cardiac effects of long-term AAS use remain inadequately characterized. We compared cardiac parameters in weightlifters reporting long-term AAS use to those in otherwise similar weightlifters without prior AAS exposure.

Methods and Results—We performed 2D tissue-Doppler and speckle-tracking echocardiography to assess left ventricular (LV) ejection fraction, LV systolic strain, and conventional indices of diastolic function in long-term AAS users ($n=12$) and otherwise similar AAS nonusers ($n=7$). AAS users (median [quartile 1, quartile 3] cumulative lifetime AAS exposure, 468 [169, 520] weeks) closely resembled nonusers in age, prior duration of weightlifting, and current intensity of weight training. LV structural parameters were similar between the two groups; however, AAS users had significantly lower LV ejection fraction (50.6% [48.4, 53.6] versus 59.1% [58.0%, 61.7%]; $P=0.003$ by two-tailed Wilcoxon rank sum test), longitudinal strain (16.9% [14.0%, 19.0%] versus 21.0% [20.2%, 22.9%]; $P=0.004$), and radial strain (38.3% [28.5%, 43.7%] versus 50.1% [44.3%, 61.8%]; $P=0.02$). Ten of the 12 AAS users showed LV ejection fractions below the accepted limit of normal ($\geq 55\%$). AAS users also demonstrated decreased diastolic function compared to nonusers as evidenced by a markedly lower early peak tissue velocity (7.4 [6.8, 7.9] cm/s versus 9.9 [8.3, 10.5] cm/s; $P=0.005$) and early-to-late diastolic filling ratio (0.93 [0.88, 1.39] versus 1.80 [1.48, 2.00]; $P=0.003$).

Conclusions—Cardiac dysfunction in long-term AAS users appears to be more severe than previously reported and may be sufficient to increase the risk of heart failure. (*Circ Heart Fail.* 2010;3:472-476.)

Key Words: myocardial contraction ■ mechanics ■ systole ■ diastole ■ anabolic agent

Circulation

Heart Failure

JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association 
Learn and Live

Anabolic-Androgenic Steroids : Worse for the Heart Than We Knew?

Matthew W. Parker and Paul D. Thompson

Circ Heart Fail 2010;3:470-471;

DOI: 10.1161/CIRCHEARTFAILURE.110.957720

Circulation: Heart Failure is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75214

Copyright © 2010 American Heart Association. All rights reserved. Print ISSN: 1941-3289. Online ISSN: 1941-3297

16:08: *“If you’re taking oral steroids, and the first metabolic checkpoint is thus your liver, on that liver, blood-filled cysts can form. If these cysts rupture, that’s a problem. You bleed into your gut. If you bleed into your muscle, the muscle will compress the bleed and ultimately stop it. You don’t have that safety net in your gut. So this is bad.”*

This is called peliosis hepatis (hepatis; not hepatitis). The article shown below is a good place to start. It’s a case study about a bodybuilder who had taken both testosterone and stanazolol... And trenbolone acetate, methandienone, nandrolone, boldenone undecylenate, oxymetholone, and insulin.

Surgical Practice



doi:10.1111/j.1744-1633.2010.00501.x

Case report

Massive hepatic haemorrhage caused by anabolic steroid-induced peliosis hepatis: Successful treatment by radiofrequency ablation

Fabio Garofalo,¹ Riccardo E. Vandoni,^{4*} Adriano Guerra,¹ Mario Alerci,² Stefano Crippa³ and Philippe Gertsch¹

Departments of ¹Surgery and ²Radiology, Ospedale San Giovanni, Bellinzona, ³Istituto Cantonale di Patologia, Locarno and ⁴Department of Surgery, Hôpital du Jura Bernois, Moutier, Switzerland

*Author to whom all correspondence should be addressed.
Email: riccardo.vandoni@bluewin.ch
Received 3 September 2009; accepted 15 January 2010.

Surgical Practice (2010) **14**, 111–115 © 2010 The Authors
Journal compilation © 2010 College of Surgeons of Hong Kong

17:08: *“But having said that, Tylenol can do the exact same amount of harm. Among high school athletes, ibuprofen frequently does that harm. It just harms different body systems.”*

While I could cite scientific literature, sometimes it says more about the scale of a problem when that problem has its very own *Wikipedia* page. So I direct you to the page dedicated to acetaminophen (i.e., Tylenol) toxicity. This: https://en.wikipedia.org/wiki/Acetaminophen_toxicity

First paragraph:

“Paracetamol toxicity is caused by excessive use or overdose of the analgesic drug paracetamol (called acetaminophen in North America). Mainly causing liver injury, paracetamol toxicity is one of the most common causes of poisoning worldwide. In the United States and the United Kingdom it is the most common cause of acute liver failure.[1][2]”

19:55: *“It seems obvious that it’s the sport that’s killing the athletes; not the drugs. The incidence of steroid-related harm is like a thousandth the rate of sport-related harm. And nobody’s arguing that, because the sport is a thousand times riskier than steroids, it should be 1000 times more illegal. We blame steroids when what we should be blaming is the sport.”*

Many of the themes and examples used throughout this section are derived from previous arguments made by Peter Harmer (see page one). The sentences that open and close this paragraph come straight from Peter. I can’t remember his *exact* phrasing (the last time we had this discussion was ten years ago), but they’re definitely near plagiarisms, if not more than that. My point: credit here goes to Peter. And credit also goes to Peter for the line (or something like it): “The sport only cares if the ball goes in the hoop” (22:36).

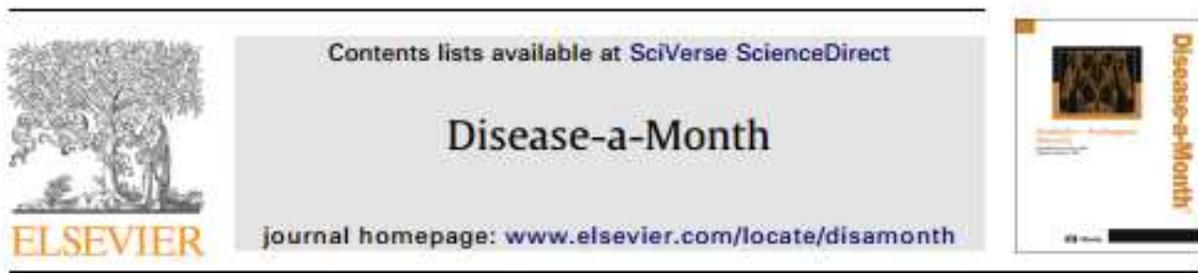
24:24: *“Next argument: steroids strip the soul from the sport. They contravene the very spirit of the sports we’ve come to cherish.”*

Peter Harmer has addressed this theme (“steroids contravene the spirit of sport”) both well and at length. At 28:35, I say “regulatory rules are changing all the time in every sport. That doesn’t interact with any souls? The entire sport is completely changed. A shot clock is added, the javelin is differently constructed, pads are incorporated into football uniforms. Every sport is in perpetual change to cater to a public in demand of newer and greater entertainments.” The examples I give here (pads, javelin, and shot clock) are probably examples Peter has previously given (circa ten years ago). I have no idea, but to be safe (and probably accurate), I yield full credit of these examples (and the larger point) to Peter.

25:28: *“The ancient Greeks were swallowing a bunch of plant seeds and potions – essentially anything they thought would give them the slightest advantage – so that Zeus might be proud. Ground up tigers, whatever. Eat it all. They were even drugging the horses in the chariot races... In the 1860’s, swimmers in Amsterdam were taking speedballs, which were mixtures of heroin and cocaine. In 1904, Thomas Hicks collapsed, quite famously, across the finish line of his gold medal marathon, hopped up on a combination of brandy and strychnine. It wasn’t until 1935 that testosterone was first synthesized and the 1950’s when anabolic steroids were becoming available.”*

There are a lot of sources that speak to this subject. Here’s one:

Disease-a-Month 59 (2013) 226–248



Anabolic—Androgenic Steroids

Donald G. Barceloux, MD, Robert B. Palmer, PhD

27:06: *“And you have to admit that the Olympics would be way more entertaining if world records were shattered every single time you watched it. No Bulgarian has peed out of a urethra since puberty, but they’re snatching three thousand pounds. That would be entertaining.”*

Lorne Michaels knows this.

On October 8, 1988, *Saturday Night Live* aired the first episode of their fourteenth season. During the *Weekend Update*, Dennis Miller announces:

“In response to what its sponsors claim is an idea whose time has come, the first All-Drug Olympics opened today in Bogota, Columbia. Athletes are allowed to take any substance whatsoever before, after, and even during the competition. So far, 115 world records have been shattered! We go now to correspondent Kevin Nealon, live in Bogota for the Weightlifting Finals. Kevin?”

We’re then taken to a skit in which Kevin Nealon is providing commentary for a clean and jerk attempt by Soviet weightlifter Sergei Akmudov (Phil Hartman).

Available online: <http://www.nbc.com/saturday-night-live/video/update-all-drug-olympics/n9691/>

36:22: *“What steroids do, or at least do best, is enhance recuperative power. They don’t create muscle; they allow you to train harder. So the people using steroids are probably training harder than you are. And the steroids are just helping them to do that.”*

There’s no shortage of articles on this subject. Here’s one (and you can go from there):

Genetics & Molecular Biology 87	
<h2>Effect of Nandrolone Decanoate on Skeletal Muscle Repair</h2>	
Authors	R. F. Piovesan ¹ , K. P. S. Fernandes ¹ , A. N. Alves ¹ , V. P. Teixeira ¹ , J. A. Silva Junior ¹ , M. D. Martins ² , S. K. Bussadori ¹ , R. Albertini ¹ , R. A. Mesquita-Ferrari ¹
Affiliations	¹ Mestrado em Ciências da Reabilitação, Universidade Nove de Julho, São Paulo, Brazil ² Patologia Bucal Universidade Federal do Rio Grande do Sul, Porto Alegre, Brazil

Bibliography

DOI <http://dx.doi.org/10.1055/s-0032-1311652>

Published online:
July 12, 2012

Int J Sports Med 2013; 34:
87–92 © Georg Thieme

Verlag KG Stuttgart · New York
ISSN 0172-4622

Piovesan RF et al. Nandrolone decanoate and myogenic factors... Int J Sports Med 2013; 34: 87–92

