

Starting Strength

The Year in Strength Science 2011

by

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This will be the first in an annual series surveying the scientific literature on strength training. Hopefully, it won't also be the last, but that's really up to you. It's an experiment. I'm going to present a selection of papers published over the last year relating to That Thing We Do. While they'll all have *relevance*, they won't all have *quality*. The goal is to highlight some papers that may fruitfully change our practice, while exposing some papers which are just baloney, but which may be waved in your general direction as an excuse for doing something stupid.

We should all be prepared for an immersion into the ugly reality that is Rippetoe's Law: *95% of the shit that goes on everywhere is fucked up*. This unfortunate mandate applies doubly to biomedical research in general and strength research in particular. Anybody who has to wade through biomedical literature looking for valuable knowledge knows that it's comparable to looking for pearls in an outhouse bucket. I gloved up and did this thing, so you wouldn't have to. You're welcome.

To find candidate articles, I conducted searches in both PubMed and Google Scholar, using very general search terms that wouldn't surprise anybody: *squat, bench press, power clean, snatch exercise, anthropometry, weightlifting, injury, strength training, biomechanical analysis*, etc., in various combinations, constraining my searches to papers published in 2011. This, of course, got me the phone book – many hundreds of articles, most of which could be eliminated outright as irrelevant. For those that remained, PubMed identified still more, as related citations, which underwent a similar winnowing. At the end of this rather exhausting process I was left with about 250 articles, which I slowly whittled down to the 39 presented here. This selection was carried out by a completely implicit process: I kept the ones I thought would be worth discussing, and trashed the rest. This is monstrous presumption on my part, and I'm sure that my overall approach missed a Big Paper or six that should have been included but wasn't, or any number of papers that might have been more interesting to you than to me. But this is a maiden voyage, and with your input and help (not to mention a year of lead time, a luxury I didn't have for this first installment) we're sure to do better next time around.

Finally, I had to actually *read* the articles, and figure out what made them shine and/or suck. The results are presented here for your amusement and edification. Each article will be briefly summarized

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– in short, I will give you *my own* abstract of the paper, including the methods and results. Following that will be a brief discussion of the paper’s relative merits (or lack thereof). We’ll sum up with the practical relevance (if any) of the paper to people like us, who just want to get stronger in the safest, healthiest, most efficient way possible. Some of these papers I treat in depth, others more briefly. Some papers (“Also-Rans”) that were worthy of mention are also included for each category, with a one-line synopsis.

I organized the “winners” into the following categories:

- **Biomechanical Analysis (6 studies, 2 also-rans)**. No mystery here. These studies investigate the physics of exercise, analyzing power, work, force, torque, moment, and related variables, hopefully (but not always) with some practical end in mind.
- **Strength Training and Sports (2 studies, 2 also-rans)**. These studies investigate how (or whether) strength training can be applied to improve athletic performance.
- **Big Medicine (6 studies)**. Near and dear to [my heart](#). These are papers that look at the impact of strength training on health, disease, and aging.
- **Programming, adaptation and strength physiology (6 Studies, 4 Also-Rans)**. Yes, that’s a broad category, but once I started trying to divide it up into subordinate categories, they proliferated beyond my control, like bunnies.
- Nutrition, supplements, and drugs (**5 Studies, 1 Also-Ran**). What it says.
- **Training and coaching techniques and sports medicine (3 Studies, 1 Also-Ran)**. Helping people train. Helping hurt people get back to training.
- **Orts and leavings (1 Study)**. Leftover papers that I wanted to talk about but couldn’t pigeonhole elsewhere.

(Here’s a big surprise: there’s a lot of overlap between some of these categories, and in some cases my assignation to one category or another was *totally arbitrary*.)

In the **Summary Section**, I’ll get on my soapbox and editorialize a bit, because that’s just how I do.

Again, it’s an experiment. We have to decide as a community whether this kind of exercise is useful. If we do, it’s sure to undergo significant evolution by the time I do it again. I look forward to your feedback, and as we head into 2012 I invite you to send me citations that might be worth looking at for next year’s review. Let’s get to it.

BIOMECHANICAL ANALYSIS

1. [A biomechanical analysis of straight and hexagonal barbell deadlifts using submaximal loads.](#) Swinton PA, Steward A, Agouris I, et al. JSCR 2011 25(7)/2000–2009.

Abstract: The authors, from various institutions in the United Kingdom and New Zealand, note that hex bars (also known as trap bars, designed to encircle the body and thus to stand inside of when loaded) are theorized to reduce lumbar stress and joint moments, and speculate that deadlifts performed (with either bar type) at submaximal loads and high velocities will yield superior power generation. They recruited 19 male powerlifters near the end of a competitive training cycle. In the first session, the investigators determined their 1RMs with both types of bar. *No kinematic or kinetic analysis was performed during this first session.* The lifters came back a week later, were covered with markers, put on force plates, and asked to perform the exercise with both bars at various loads up to 80% of 1RM *as rapidly as possible.* The data from this session were used to determine peak joint moments, power output, and bar trajectories across different loads for both types of bar. Fairly straightforward data and statistical analyses were used. It's worth noting, however, that the authors specify that the starting point for each trial was defined as the point where the barbell was 2 mm above its initial resting position – in other words, after it had left the floor.

The authors found that lifters attained a greater 1RM with the hex bar. The differences were not impressive (265 +/- 41 kg vs. 245 +/- 39 kg), and the confidence intervals overlapped considerably. We could very well imagine that a larger and more diverse sample size might obliterate – or widen – this difference. In the submaximal weight/power tests, the hex bar resulted in lower peak moments at the lumbar spine, hip, and ankle – except at higher weights, where these differences disappeared – and an *increased* moment at the knee at all weights tested. The hex bar demonstrated a more vertical bar path at 80% 1RM, although its maximum horizontal displacement was actually greater than that of the straight bar. *All kinematic and kinetic outcomes apply only to the submaximal testing performed at peak power, not for the 1RM tests.* This is critical to keep in mind.

Discussion: This paper purports to do more than just compare hex bars to straight bars. It attempts to show that deadlifts can be performed as a type of power ($F \cdot d/t$) training at submaximal weights and high velocities. This sounds reasonable, and it's probably right, but it's important to point out that the study does not demonstrate that deadlifts modified in this manner are superior to Olympic lifts for *training* power production. Both hex and straight bar deadlifts generated peak powers in the neighborhood of 4500 W in this study, comparable to power outputs reported for the clean. *But these subjects did not perform cleans or snatches,* so we don't know what kind of power they could have produced with those exercises.

The overall design of the study seems curious. Why were the kinematic/kinetic studies performed for the low-weight/high velocity deadlifts, but not the standard “slow” deadlifts – the most commonly performed variety? I've tried to wrap my head around this, but it just looks like a huge lapse to me.

Practical Implications: There are no surprises here, just confirmation of what most of us would have guessed. The hex bar changes the biomechanics of the deadlift, and doing deadlifts faster generates more power. Okay, but...so what? The data here argues strongly that the hex bar decreases the load on the spinal erectors. That's great, unless *I want to train my spinal erectors,* in which case I want to increase that load. The authors, to their credit, say as much in the discussion.

So, if I want a knee-dominant exercise that keeps my back more vertical and minimizes my lumbar moment, this data shows that I can certainly buy a hex bar and do that. Or...I *could* just use the bar I have, and do sumo deadlifts. Or front squats.

And if I want to train power in a range similar to that attained by doing the power clean, I could buy a hex bar and do high-velocity deadlifts at 30% of 1RM. Or...I *could* just use the bar I have, and *do power cleans*.

2. [Effects of fatigue on bilateral ground reaction force asymmetries during the squat exercise](#). Hodges SJ, Patrick RJ, Reiser RF. JSCR (2011) 25(11):3107-11.

Abstract: The authors, from the Colorado State University Departments of Health and Exercise Science and Biomedical Engineering, note that functional asymmetries in lower extremity strength *might* predispose to injury. (This, of course, implies they *might not*.) They investigated the effect of fatigue on such asymmetries during the barbell squat. Seventeen young men and women performed squats on force plates, and force asymmetries were assayed. Most subjects placed more load on their non-dominant foot (the left, in most cases). Subjects then performed 5 sets of 8 reps at 90% 8RM. Asymmetries did not increase with fatigue, and even showed a trend toward decrease.

Discussion: The study is reasonably well-conducted, though small.

Practical Application: In case you worried about this...don't.

3. [Effect of loading on peak power of the bar, body and system during power cleans, squats, and jump squats](#). McBride JM, Haines TL, Kirby TJ. J Sports Sci (2011) 29:11: 1215-21.

Abstract: This study comes from the Neuromuscular Laboratory in the Department of Exercise Science at Appalachian State University in North Carolina. It's a very small study, in which 9 young males with respectable PC 1RMs (213 lbs, average) performed PCs, squats or jump squats at various loads up to 90% 1RM. They looked at body power, bar power and system power. I won't go into the details of the methodology and findings; they're not problematic and you can pull the paper if you're interested. In brief: Peak bar power was attained at 90% 1RM for the squat and 80% 1RM for the PC. Body peak power was at 10% 1RM for the squat and 90% 1RM for the PC. System peak power maxed out at 50% 1RM for the squat and 80% 1RM for the clean. The authors draw the (hardly contentious) conclusion that bar, body and system power output vary according to exercise and loading, and that their observations *may* (read: or *may not*) have implications for training.

Discussion: Wading through the literature, I've seen quite a bit of this stuff, suggesting that athletes can generate more power at some mid-range percentage of their 1RM. We saw some of this in the deadlift hex-bar study, and the whole area of investigation seems to be quite in vogue. One particularly glaring deficiency in all of the literature that I've seen on this topic is the failure to document consistent form throughout the loading. It's trivial to hypothesize that form is more likely to degrade as weight approaches 1RM, and that degradations in form are likely to involve "power leaks" – bent elbows on the first pull, failure to maintain spinal extension, etc. If this were the case, the proper approach might be to correct the form, not the weight.

As noted by the authors of this paper, the exact location of the Sweet Spot (x% 1RM) varies from study to study, but implicit in all of this work is that maybe this x% 1RM loading is where we should train to maximize power. But I've seen no actual proof of that – in other words, no study that I'm aware of demonstrates that guys who train at x% of their 1RM with explosive lifts can generate

more useful power *in the field* and thereby actually *perform* better at MMA, football, golf, chess or whatever, compared to guys who just train those lifts up to their 1RM, and beyond.

And so, if I may, here's a question: If you were to focus your training at, say, 60% of your 1RM, because it ostensibly "maxed your power," *what would happen to that 1RM?* I don't think that 1RM would go up much if you didn't train it. In fact, it might go down. And then you'd be training at 70% of your 1RM, and you'd have to drop the weight to get back to the Sweet Spot. And soon you'd have to adjust down again. And again. This way lies madness.

And so, here's another question: Assuming that training at 60% of your 1RM really was better for training explosiveness, *what would happen if you actually increased your 1RM?* Wouldn't that, you know, make your 60% 1RM *even bigger?* I'm just counting on my fingers and toes here, but yes, I think it would.

And so, here's still another question: *wouldn't an athlete training at 60% of a 1RM that was greater than his previous 1RM be generating more power than he was before?* Why, I do believe that might be the case.

And so here's yet another question: *if you train to actually increase your weight on explosive lifts, won't you also be training your 60% lifts long the way, as you warm up to your new 1RM?* Hmm. I don't see why not.

And so, finally, here's my last question.

Practical Application: What are we supposed to actually *do* with these results?

Okay, I'll try to be constructive. If anything, these findings would seem to have implications for programming. One could hypothesize based on results like these that most of the volume in a clean workout should be at Sweet-Spot weight (provided we can agree on what that weight is), while still shooting for increases in 1RM at lower volume. But until somebody studies that hypothesis and shows that such programming improves actual performance, I'm not sure how this information should change our practice.

4. [Kinetic comparisons during variations of the power clean](#). Comfort P, Allen M, Graham-Smith P. JSCR (2011) 25(12):3269-73.

Abstract: There's already some data in the literature on rate of force development (RFD), peak ground reaction force (Fz) and peak power with different loads and variations of the clean – and it's all over the place. This study, from the University of Salford in Manchester, England, put 16 rugby players, with some experience in performing the clean, on force plates and measured these variables at 60% 1RM for the power clean (PC), hang power clean (HPC), mid-thigh power clean (TPC) and mid-thigh clean pull (CPull). They report minimal differences between the PC and the HPC, but both the TPC and the CPull demonstrated higher peak power output, Fz and RFD than the PC and HPC. The authors conclude that the HPC and TPC are preferable to PCs when training to maximize these power parameters.

Discussion: This paper was discussed on the SS forum earlier this year. I've now given it a second look, and I haven't changed my mind: there are so many questions about this small study that I don't think it proves anything. We have only the most vague documentation of how these movements were actually performed. (To be fair, this shortcoming appears to be endemic in the literature.) We don't know *exactly* what differentiated a TPC from a HPC, or whether these variants were done from a static position or with a stretch reflex, or whether these findings varied with load – because the authors don't tell us. As Rippetoe has previously pointed out, having the subjects perform HPCs and TPCs

with 60% of the 1RM full movement subjects the study to a possible bias toward higher RFD for these movements compared to the PC – “nobody except an inexperienced lifter does as much from the hang as they do from the floor.”

The presentation of the data lacks clarity – it’s not clear if the authors are reporting means +/- 95% confidence intervals or some other measure of error. They *are* reporting a range of values around the mean, however it is that they derived that range, and it looks to me like the ranges they report are huge. Most critically, for me, is that it’s not entirely clear how RFD was calculated. The sum total of methods provided in this regard is: “*Instantaneous RFD was determined by dividing the difference in consecutive vertical force reading by the time interval between the readings.*” That doesn’t exactly clear things up. *Which readings?* It would have been trivial, and quite elegant, to include representative force-time curves for each group as figures and *show* us the power signatures for each movement. But they didn’t.

Practical Application: What are we to do with this data, especially since the details of its collection and presentation are unclear? Even if we were able to peer through the murk here and actually figure out what it is that the investigators thought they demonstrated, the end points of the study would still be poor surrogate markers for actual athletic performance. It very well may be that variants of the power clean performed at different loads will optimize training. But this paper doesn’t prove it. There’s nothing here to change our practice.

5. [Weightlifting performance is related to kinematic and kinetic patterns of the hip and knee joints.](#) Kipp K, Redden J, Sabick M, Harris C. JSCR (2011) epub ahead of print.

Abstract: This is a collaboration between authors from across the US. It is a strictly descriptive study, in which ten lifters were fitted with joint markers and performed cleans at 85% of 1RM on force plates while being photographed by 6 video cameras. Kinematic and kinetic data were subject to several very technical transformations to “normalize” the data and generate a covariant matrix. The purpose was to extract biomechanical variables that correlated with weightlifting performance (which for these authors simply boiled down to greater lift mass). This was accomplished by performing a “functional principle component analysis” on the data, a technique that is frankly quite beyond the skill set of Your Humble Narrator. Once principle component functions were extracted from the covariant matrix produced by this procedure, they were subjected to simple linear regression analyses to determine whether and to what degree they correlated with relative lift mass. The authors report greater lift mass was associated with less knee extension in the first pull and a peak in knee extension torque during the second pull. Greater lift mass was also associated with a stable trunk position and hip position during the first pull, with minimal hip extension until the second pull.

Discussion: The methods section of this paper is an interesting but challenging read; I went through it twice before I began to get a clear picture of what was going on. This would have been a more palatable process if the paper were well-written, which it isn’t. The conclusions, on the other hand, are easy to grok: Guys who lift more weight save their peak knee extension moment for the second pull, and start the second pull with a more horizontal back – the *trebuchet* effect. This does not seem particularly contentious, and comports well with previous literature, not to mention the Starting Strength model (see *SS:BBT*, 3d Ed, pgs 202-210). That said, we have to take a little care here. The methods section of this paper will be somewhat opaque even to many other exercise scientists, so it’s difficult to know whether the data analysis methods were robust. Let those of us who don’t spend much time with functional principle component analysis of covariant matrices just assume these guys knew

what they were doing. The authors basically derived biomechanical patterns from 10 *experienced* lifters at 85% of 1RM and correlated them with performance – that is, these movement patterns were taken to be “representative” of biomechanics at 1RM. The authors felt this was justified because “technical aspects of competitive weightlifting performance stabilize at loads above 80% of 1RM.” This seems to me to be an extraordinary claim, and the authors support it with but a single citation, a paper that appeared in *Теория И Практика Физической Культуры* in 1979. Yeah, I would certainly look that up, except all my Russian exercise science journals got thrown out with my vintage comic books and back issues of *Hustler* when my Mom cleaned out the attic in ‘86. As it stands, we don’t know how things would have been different for *these* athletes at their 1RM *because the investigators didn’t look*. The authors themselves concede that there are problems with their use of relative joint angles in kinematic analysis, and call for expanded studies including more in-depth modeling and myographic analysis.

Practical Application: This is an imperfect and incomplete study, but on balance it is another data point in support of a model of Olympic lifting in which a more horizontal back and more acute knee at the start of the second pull optimizes performance.

6. [Using 3-dimensional kinematics to identify feedback for the snatch: a case study.](#) Ho KWL, Williams MD, Wilson CJ, Meehan DL. JSCR (2011) 25(10):2773-80.

Abstract: In this Australian study, investigators used an elaborate setup involving six high speed cameras, digital analysis software and joint markers to study the snatch in a single athlete, comparing kinematic characteristics of successful vs. failed attempts. Contrary to much coaching wisdom, they found no evidence that bar velocity impacted the success of a lift at heavy loads. Rather, their data indicates that the critical determinants of success in the snatch have to do with the starting position, particularly hip and pelvic angles.

Discussion: The paper is a technical *tour de force*, with some very specialized data gathering and statistical manipulations. In particular, the partition analysis used by the authors is, like covariant matrix analysis, a bit beyond my skill set, and I had to take their findings in this regard at face value.

Practical Application: The method used here is beyond the means of individual athletes, and probably impractical for even most if not all national weightlifting teams. The big take-home point from this biomechanical analysis is the finding that – for at least one lifter in the world – the *principle determinants of a successful snatch are the diagnostic angles in the starting position*. Imagine that.

Also Ran:

[Heart rate variability analysis using a ballistocardiogram during Valsalva manoeuvre and post exercise.](#) Shin JH, Hwang SH, Chang MH, Park KS. *Physiol Meas* (2011) 32:1239-64. -- Of interest primarily for its methodology and description of ballistocardiography. No immediate practical implications for trainees.

[Hip muscle activation and knee frontal plane motion during weight bearing therapeutic exercises.](#) Lubahn AJ, Kernozek TW, Tyson TL. *Int J Sports Phys Ther* (2011) 6(2):92-103. -- Another silly EMG study, but at least the investigators documented how the exercises were performed. Sadly, that makes this study stand out.

STRENGTH TRAINING AND SPORTS

1. [A Novel Technique: Strength Training for American Football Tackling](#). Tvrdy D. JSCR 2011 (33) 1:76-80.

Abstract: This isn't a research study, it's a "Guest Column" in the Journal of Strength Conditioning and Research. The author states that "*one of the biggest drawbacks to the gym setting is that it is often too static to have enough carry over to a dynamic environment such as the playing field.*" He provides absolutely no supporting evidence for this claim, and goes on to assert that "*the more an individual task can be recreated in a gym setting the more likely it is to carry over to performance in the field.*" This would be important if true, but again the author provides absolutely no supporting empirical evidence, either in the form of original research or peer-reviewed references, for such a claim. He's a physical therapist, and we'll simply have to take his word for it, just as we'll have to trust that the goofy exercises he presents in a series of photographs will make football players better at tackling. These exercises all involve a pulley machine, some dumbbells, a step-up box, and...a towel. That's it. That's all there is here: a guy who made up some exercises and claims they will help training for football. As far as I can tell, *nobody actually involved with the sport was involved in the development or evaluation of these exercises.* The improvement (or decline) in tackling performance produced by these strange towel-and-dumbbell movements is unknown...*because the author didn't test them.* Really. No footballs were harmed in the production of this article.

Discussion: I include this *item* not because it is informative or even interesting, but because it is so beautifully representative of the commonly held belief that strength training for any sport should emulate the movements of that sport. So martial artists should punch with kettlebells, football players should tackle dumbbells *with a towel*, and somebody, I suppose, should invent a 50-lb iron for golfers. Herein lies the stubbornly persistent and very fundamental confusion about the distinction between *training* and *practice* – a confusion that is given standing in the literature by articles like this one. The *JSCR* is (right or wrong) one of the most prestigious journals in strength science, and indeed the astute reader will have noticed its overrepresentation in the articles presented herein (not by choice or design, just how it worked out). But when you see crap like this in a peer-reviewed academic journal, it really gives you pause. Calling it a guest editorial doesn't excuse it. If *JAMA* included a "guest editorial" from a physician's assistant that basically boiled down to "here are some pictures of this technique I made up, which I haven't actually bothered to evaluate in any meaningful way, but which I suggest you all start using on patients," I'd like to think that *Heads Would Roll*. Apparently we're dealing with a different yardstick here. Unbelievable.

Practical Application: *Towels are really useful*, as anybody who's ever read the *Hitchhiker's Guide* already knows.

2. [Changes in strength over a two year period in professional rugby union players](#). JSCR (2011) epub ahead of print.

Abstract: It's a shame that this one appears in *JSCR*, because that means it's not available without NSCA membership, and this paper is a must-read. This observational study comes from the School of Exercise in the College of Biomedical and Health Sciences at Edwin Cowan University in Perth, Australia. The authors note the preponderance of short-term studies in novice trainees, and instead turn their attention to experienced athletes who are already strong. They observed the strength,

body composition, and training patterns of twenty professional union rugby players over two years. These athletes engaged in periodized training regimens variously emphasizing strength, hypertrophy, power, maintenance, recovery, and sport-specific skills. Conditioning training was emphasized during the pre-season. Strength training frequency varied with training period and ranged from 4-12 sessions per week. Multi-joint exercises were the order of the day: squats, deadlifts, bench presses, chin-ups, bent rows, shoulder presses, and cleans. Information on diet was not reported. The authors report practically significant increases in strength, lean mass, and total mass over two years. The magnitude of improvement was not associated with the age of the athletes (mean age at the end of the study was 26). The magnitude of strength improvement was negatively correlated with initial strength level, meaning that *the guys who were the strongest at the beginning of the study period gained the least strength*.

Discussion: Although this is a strictly observational study, not an experiment, it is one of the better papers published in the strength literature this year, an excellent profile of training for advanced athletes, much closer to their genetic potential than the average Joe, observed over a meaningful interval. Their sport requires high levels of agility, power, skill, endurance, and performance in all energy systems (*Broad Domains*, anyone?). These individuals managed to improve performance, strength and body composition over a two-year period despite already being on the flat part of the adaptation curve, not to mention the rigors of their sport. The observational and statistical methods are appropriate, and the authors do not draw conclusions their data cannot support. This paper underscores the importance of combating the law of diminishing returns by engaging in carefully programmed periodized training with multi-joint exercises and carefully timed skill and conditioning drills.

Practical Application: The most immediate practical relevance of this paper will be found, of course, by those engaged in professional sports like rugby, soccer and football. Most of us don't train at this advanced level. But many of us are intermediate trainees engaged in other physical disciplines or sports, and we are confronted with challenges that are similar in kind if not in degree. This paper is an illustration of how balanced, carefully periodized training can help us move the ball forward. If these guys can get stronger, anybody can.

Also Ran:

[The strength and conditioning practices of strongman competitors.](#) Winwood PW, Keogh JWL, Harris NK. JSCR (2011)25(11)3118-3128. A survey-based study that may be of practical interest to some readers.

[Using squat testing to predict training loads for lower-body exercises in elite karate athletes.](#) Wong DP, Tan ECH, Chaouachi A, et al. JSCR (2011) 24:3075-80. Supports the use of a 6RM squat to predict loading for lower-body training, even in athletes that engage in unilateral-movement activities (lunging, asymmetric sports like karate or fencing). An interesting read.

BIG MEDICINE

1. [Role of a 12-week resistance training in preserving the heart against ischemia-reperfusion-induced injury.](#) Soufi FG, Saber MM, Ghiassie R, Alipour M. *Cardiol J* (2011) 18(2): 140-5.

Abstract: This paper comes to us from the Islamic Republic of Iran, where rat life is cheap. The authors divided twenty male rats into trained and sedentary groups. The sedentary rats were left in their cages, where they presumably spent their time eating *halal* rat chow *ad libitum* and watching *Days of Our Lives* dubbed in Farsi. Trained rats underwent a 12-week squat program.

That's right: Rat squats. The authors used an apparatus that was first described in the literature by Tamaki *et al* in 1992. (A similar setup was used in [this paper](#) by do Cormo *et al*, which is also worth looking at.) These rats were highly motivated, as the experimental apparatus incorporates a shock mechanism that compels the animals to perform the required exercise. (This of course immediately suggests a useful modification of training platform design for high school and college athletes, but that was not the focus of this paper.) After twelve weeks, the animals from both groups were subjected to reversible coronary ischemia for 35 minutes – in other words, they were given a heart attack. After the ischemic interval the coronary artery was opened (like a patient going to the cath lab) and the animals were reperfused. Hemodynamic parameters were measured during reflow. The rats were killed at 80 minutes of reperfusion and the size of the infarct (the volume of dead heart tissue) was measured. Trained animals had higher post-ischemic coronary flow and lower diastolic pressures. These are surrogate markers for myocardial oxygen delivery and myocardial oxygen demand, respectively, and both were better in animals that squatted. Infarct volume, measured (correctly) as a percentage of the zone at risk, was significantly lower in trained animals (39% vs. 29%).

Discussion: Speaking as one who does ischemia-reperfusion research (brain, not heart), I have to say *most* of the misgivings I have about the methods in this paper are mere quibbles. Overall, the study is adequately done, with three important caveats:

- Sedentary rats were not subject to shocks, and so this variable was not controlled.
- Eighty minutes hardly represents a long-term outcome.
- Infarct size is a disease-oriented measure, not a patient-oriented outcome. What would have been far more interesting is to have observed any differences between the groups in mortality, morbidity or progression to heart failure in the long term.

Practical Application: The study is one more very flawed data point supporting what we already know: *Do your squats; they're good for you. Even if you're an Iranian rat.* Because, clearly, strong rats are harder to break – although this study doesn't tell us whether they're more useful.

Of course, the real reason I just *had* to include the article is this:

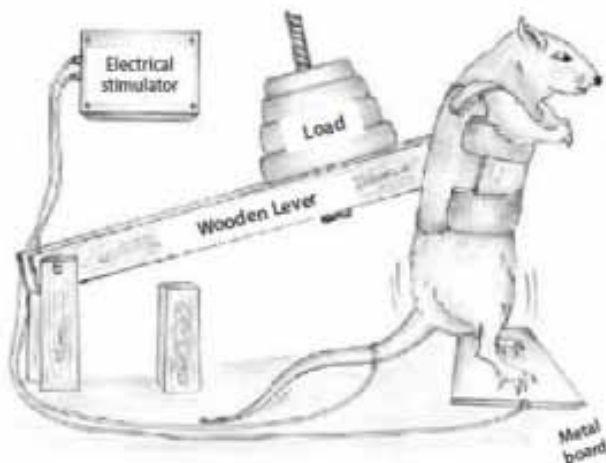


Figure 1. Strength training apparatus adapted from Tamaki *et al*, 1992. Reproduced from [do Carmo *et al*](#) under the provisions of a Creative Commons Attribution License.

2. [Exercise training for adults with chronic kidney disease](#). The Cochrane Library 2011 (10).

Abstract: The Cochrane Collaboration is a multinational organization of volunteers who conduct *systematic reviews* of the biomedical literature. This paper is an example. A systematic review begins with a question, in this case whether evidence in the biomedical literature supports the use of exercise training for adults with chronic kidney disease. The authors then pull and review all the relevant literature and pool the data, using an explicit methodology that excludes poorly-conducted or irrelevant studies and gives primacy to placebo-controlled, randomized clinical trials with patient-oriented outcomes. This sort of *meta-analysis* is sensitive to the GIGO (garbage-in-garbage-out) dynamic, but it is nevertheless a useful if imperfect method of making a very large and powerful study out of smaller studies. The authors of this systematic review extracted 45 randomized controlled studies, of which 32 could be pooled. The resultant meta-analysis demonstrated multiple benefits of exercise (of all types) for patients with CKD. The authors note that strength training interventions, while helpful, are underrepresented in the literature, and call for increased emphasis on study of resistance training in future controlled trials.

Discussion: Patients with CKD are *sick*, especially those who have progressed to dialysis-dependent renal failure. These patients almost always have significant comorbidities and are at constant risk of sepsis, lethal levels of serum potassium, arrhythmia, nervous system dysfunction, pericardial tamponade, pleural effusion, and sudden cardiac death. If there were ever a population in which you would think the rewards of exercise weren't worth the risk, this would be it (along with congestive heart failure...but see below). And yet, here is another data point affirming that even these very ill patients – even dialysis-dependent and post-transplant patients – can benefit from exercise.

Practical Application: There are problems here, not least the under-representation of resistance training in the data, and all the caveats inherent to meta-analyses. But the take-home point is salient: exercise seems to help *everybody*. Big Medicine.

3. [Intermittent vs. continuous exercise training in chronic heart failure: A meta-analysis](#). Smart NA, Dieberg G, Giallauria F. Int J Cardiol (2011) epub ahead of print.

Abstract: Another meta-analysis, this one from an Italian-Australian collaboration, and focused on heart failure. The question here is whether intermittent exercise (high-intensity with rest intervals) is superior to continuous exercise (uninterrupted, lower-intensity work) for patients with congestive heart failure (CHF). The authors didn't have much to work with: the extracted studies incorporated only 446 patients. The meta-analysis revealed that intermittent exercise elicited larger increases in peak VO₂ than continuous exercise, and that the best improvements in studied parameters were found when strength training and intermittent exercise were combined.

Discussion: We need to be careful not to take too much from this study. This meta-analysis was smaller than some controlled trials, used a disease-oriented rather than a patient-oriented outcome, and the average length of the included studies was 12 weeks, allowing for no evaluation of long-term outcome. Which is too bad, because this is an area of tremendous importance. The burden of congestive heart failure is huge in developed countries, and it's getting worse. This disease has the appearance of being indolent, but it's actually deadly. The five-year survival for CHF just absolutely sucks. You'd be better off with a generic cancer. The end point for most CHF patients is a malignant arrhythmia and sudden cardiac death. Again, these are patients you might think deserve a pass from heavy exercise. But there's a lot of data to suggest otherwise, and this paper also indicates a benefit, although it's too

limited to derive conclusions about morbidity and mortality, which are the real end-points of interest. Finally, I'd like to quote a statement the authors make in the introduction: "*The underlying theory is that higher intensity, intermittent stress is more likely to promote peripheral adaptations and produce concurrent improvement in functional capacity.*" Does that ring any bells?

Practical Application: Patients with congestive heart failure can tolerate, and probably benefit from, high intensity intermittent exercise. The implications vis-à-vis barbell training are clear. This is an area that is screaming for fruitful research. Somebody needs to do a long-term study of CHF patients under the bar.

4. [Effects of resistance training on body composition, muscle strength and quality of life in postmenopausal life.](#) Bonganha V, Modeneze DM, Madruga VA, Vilarta R. Arch Gerontol Geriatr (2011) epub ahead of print.

Abstract: Here is a counterpoint to the Big Medicine hypothesis. The authors, working at the University of Campinas in San Paulo Brazil, recruited 85 postmenopausal women into the study. Of these, all but 32 were excluded or left the study for "personal reasons." Demographics and other information on the original cohort of 85 are not provided; only the final study group is described. Exclusion criteria included obesity, heart disease, diabetes and "severe musculoskeletal problem," which is not defined. It is not clear if women who quit the study left before or after randomization to treatment group (TG) or control group (CG). Women in the TG undertook a 16-week resistance training (RT) program consisting of leg presses, bench presses and various machine exercises (lat pulldowns, hamstring curls and the like), three times a week. None of the women were on hormone replacement therapy (HRT) and all subjects were *advised not to change their eating habits*. Quality of Life was assayed before and after the study by a standardized WHO questionnaire. The authors report increases in the TG group for strength in bench press, leg press and curls, but no change in body composition or QoL.

Discussion: The findings of this study are at odds with many others, and so in that limited sense the paper is an important one to consider. But it suffers from serious limitations. How, *exactly*, did an original set of 85 get culled to 32, and when and how, *exactly*, did randomization occur? The authors tell us that 43 of the original 85 started the study but only 32 completed it, and *these 32 were randomized to 16 TG and 16 CG*. That's *really* fishy. The exclusion criteria almost guarantee that subjects who were most likely to derive a benefit from therapy were not studied. The exercise regimen did not incorporate full-body exercises likely to elicit salutary systemic responses. The study was too short to identify long-term benefits. One might argue the lack of any change in body composition in the TG group reflects a poorly-designed regimen. And attempting to drive strength and body composition improvements in these patients with RT while advising patients to keep their diets exactly the same is just goofy.

Practical Application: This study is perhaps most useful for illustrating how *not* to program strength training for postmenopausal women.

5. [A prospective study of muscular strength and all-cause mortality in men with hypertension.](#) Artero EG, Duck-chul L, Ruiz Jr, et al. JACC (2011) 57(18):1831-7.

Abstract: This is a long-term (23-year) observational study of hypertensive men whose muscular strength was assayed by 1RM for leg and bench press. The authors found that hypertensive men had

lower age-adjusted death rates if they were stronger. They conclude that strength seems to protect men from all-cause mortality.

Discussion: I could quibble about the finer points of this paper, but it's not necessary. Here's the hard truth: *Any conclusion that strength was protective for this population cannot be supported by this data.* Sure, I *want* to believe strength is protective. In fact, I *do* believe it, based on other limited data, my clinical and personal experience, and physiologic reasoning. But this paper doesn't prove causation, only correlation...the same strong correlation between strength and mortality that pops up every time somebody looks. But you can't really call it science or claim a causal effect if you don't do an experiment, and *this isn't an experiment.* It's just an observation – the same kind of observation that has led scientists down the garden path on, say, dietary fat and heart disease. Observations are compelling, and they have value, but we have to remember that studies like this can only generate hypotheses, not prove them.

Practical Application: None. But it's an encouraging read.

6. [Muscle mass gain after resistance training is inversely correlated with trunk adiposity gain in postmenopausal women.](#) Orsatti FL, Nahas EAP, Orsatti CL et al. JSCR (2011) epub ahead of print.

Abstract: This paper also comes from Brazil, which is apparently a hotbed of strength training research. The authors recruited a pool of 83 relatively healthy, postmenopausal women. Of these, twenty-two actually completed a 9-month machine-based RT program composed of the Usual Suspects: pec-deck, curls, knee extension, lat pulldowns, ham curls, and of course the ubiquitous leg press. Trainees were carefully instructed to breathe in on the eccentric phase and out on the concentric phase, “to avoid apnea.” Truncal adiposity (TA, basically belly fat), strength, and levels of IGF-1 (a trophic factor) and interleukin-6 (IL6, a recognized marker of inflammation, sarcopenia, and poor health in elderly populations) were studied, along with various other surrogate markers. The investigators report that women who gained more belly fat gained less muscle mass and had higher levels of IL6, which is bad. Those who gained less belly fat put on more muscle and had higher levels of IGF-1, which is good. The authors conclude that their results suggest that increased belly fat during RT increases IL6.

Discussion: The title says it all: this paper found *correlation*, not *causation*. The correlations between IGF-1 and muscle hypertrophy and between IL6 and sarcopenia are well-described, and this paper does little besides describe them again. True, their data suggest that belly fat increases IL6, but do not *demonstrate* it. The results also suggest that IGF-1 and IL6 may be biomarkers to guide training in an elderly population, but I would suggest that hypertrophy, strength, and function would be better markers. Finally, the authors report that, out of the 22 women who did RT, all got stronger, and those changes in strength were similar. But only eleven gained muscle mass, and those gains were tiny. The study is to be lauded for its relatively long interval, but the lack of a control group and the failure to document dietary patterns in the subjects make this a weak observational study at best.

Practical Application: On my reading, the most important practical finding of the paper has nothing to do with the statistical correlations the authors report. Rather, the most important observation is buried in the methods section. The sample pool consisted of women who were being followed at the hospital's Outpatient Menopause Unit. Eighty-three sedentary but otherwise healthy women were selected to participate, *and all but 39 refused, most stating they were too busy.* Of the 39 who started the study, *17 more dropped out.* These “findings” do not appear to be unusual, and to my mind they represent a huge, untapped area of study, as in other areas of medicine: the issue of *treatment*

compliance. For those of us interested in bringing Barbell Medicine to the people who need it, this is perhaps the greatest barrier to success, and it's an issue that is screaming for more research.

PROGRAMMING, ADAPTATION AND STRENGTH PHYSIOLOGY

1. [Muscle damage and muscle remodeling: no pain, no gain?](#) Flann KL, LaStayo PC, McClain DA et al. *J Exp Biol* (2011) 214:674-9.

Abstract: The study was conducted by investigators from the Department of Biological Sciences at the University of Arizona and the Departments of Physical Therapy; Exercise and Sports Science; and Orthopedic Surgery at the University of Utah. The intent was to determine whether muscle damage and soreness are necessary for adaptive muscle remodeling and strength acquisition. The investigators specifically note that strength acquisition without muscle damage – and attendant inflammation – would be particularly beneficial in designing programs for elderly, sarcopenic, and chronically ill populations. Fourteen *young, healthy university students who had not trained in the previous year* were recruited, and randomized (through a “stochastic” procedure that is not described) into “pretrained” (PT) and “naïve” (NA) groups. Both groups underwent training on an Eccentron ergometer, a recumbent bike-like gizmo in which the subject resists a force pedal with an eccentric contraction of the quads and hip extensors. The device is able to program and record energy expenditure, which allowed the investigators to deliver exactly the same amount of work volume to all participants over an 11-week study period. However, this total work volume included a 4-week “ramp up” in resistance for the PT group from 75 kJ to 250 kJ, while the NA group jumped in cold at 250 kJ in week 4, and had to make up for lost time. Muscle strength was assayed as maximal voluntary *isometric* contraction of the knee extensors. Surrogate markers for “muscle damage” were creatine kinase (CK) and muscle soreness, measured by spectrophotometry and a visual analog scale, respectively. Quadriceps volume was measured by MRI. Muscle biopsy subjected to PCR analysis was used to assay IGF-1Ea levels as a marker of trophic response to training. The NA group reported significantly greater levels of muscle soreness than the PT group during week 4 only. The PT group's CK levels did not rise above baseline, while the NA group had markedly higher levels for the first four weeks of their training (weeks 4-8), after which their levels were not significantly different from the PT group's. Increases in strength, muscle volume and elaboration of mRNA for IGF-1Ea were similar between the groups. The authors conclude that muscle remodeling, hypertrophy and strength increases can occur in the absence of “any discernible” damage to the muscle.

Discussion: Where to begin? On the face of it, this study would seem to present a challenge to the idea that strength adaptations are triggered by disruptions in muscular homeostasis and structural microtrauma. The experiments *as designed* were well-executed, although the failure of the authors to describe the “stochastic process” used to randomize subjects into groups raises a little red flag. The study certainly indicates that trainees can “ramp up” to higher work volumes with a minimum of soreness. I'm going to go out on a limb and suggest that most of us aren't surprised at that. Even so, I would recommend that anybody interested in the biology of strength training get the paper and read it for themselves: it is well-written, represents a not-half-bad stab at the question, and will probably be considered an important paper, right or wrong.

But there are tremendous limitations here. The study is a *beautiful illustration of the novice effect*. These were untrained individuals, and all *achieved similar strength levels despite significant differences in*

programming. The course of training was brief – less than three months – and the increases in strength and muscle volume were quite modest. We don't have any idea what kind of soreness and muscle damage, if any, would have been necessary to sustain linear progress for these trainees, assuming that progress would continue with the cute little bike thingy they were using (it wouldn't). It's important to note that while the NA group had more soreness and CK release, this was true only at the beginning, when they were dumped into the study without the ramp-up of the PT group, and these differences disappeared by the end of the training period. The trainees, while novices, were young and healthy – *not the frail, elderly, sick, sarcopenic population of apparent interest to the investigators.* It's entirely possible that such a special population would have responded quite differently to either training regimen. And while the investigators talk about the importance of minimizing “inflammation” in these special populations, no direct assay of inflammation was performed in this study. The use of an isometric assay of strength after an isokinetic training regimen is curious. Finally – and this is critical – the investigators *did not actually assay muscle damage.* They measured *surrogate markers* of muscle damage – soreness and CK release, both of which are subject to adaptation. Muscle biopsies were performed, but they were used only for PCR. No histological evaluation of muscle tissue was undertaken.

Practical Application: This paper falls far short of demonstrating that strength adaptation occurs in the absence of inflammation or microtrauma to muscle tissue. It does have limited value, though, for those of us interested in training special populations. It reminds us to allow these individuals adequate time to adapt. Debilitating soreness in *any* trainee indicates unproductive overload and is at cross-purposes to rational strength programming and training safety, not to mention business success. But...you already knew that, right?

2. [Associations of exercise-induced hormone profiles and gains in strength and hypertrophy in a large cohort after weight training.](#) West DWD, Phillips SM. Eur J Appl Physiol (2011) epub ahead of print.

Abstract: A prevalent view in strength science is that resistance training results in the systemic elaboration of trophic factors such as testosterone, growth hormone (GH), and insulin-like growth factor (IGF-1), which in turn mediate gains in muscle hypertrophy and strength. This view is challenged by West and his colleagues at McMaster University in Canada. In this paper, which appears to be an attempt to mine data obtained in the course of work first published in 2007, they assayed serum levels of these hormones in 56 young men at the midpoint of a 12 week training program, and derived correlations between these levels and gains in LBM and strength. They report no correlations between exercise-induced elevations in GH, IGF-1 or testosterone and gains in lean mass or strength. Increases in muscle fiber cross-sectional area were weakly associated with GH and cortisol; LBM correlated with cortisol alone.

Discussion: West's research is an important challenge to one of the prevalent models of the biology of strength adaptation, and I look forward to seeing more of his work. This paper, however, is not particularly convincing, as it has several critical limitations:

1. This paper appears to be a post-hoc analysis of material collected for an earlier study, which was an evaluation of the effect of fat-free milk after resistance training on lean body mass accretion. Taking data from a study designed to investigate hypothesis A and re-analyzing that data to investigate hypothesis B is not the most robust approach.
2. The investigators evaluated only serum hormone concentrations, and only for particular isoforms of the hormones in question. GH, for example, has multiple splice variants, and recent literature suggests that not all isoforms of IGF-1 promote muscle hypertrophy to

the same degree. The samples they evaluated had been collected for the 2007 study and maintained at -800C during the interval.

3. Serum hormone concentrations aren't even close to being the whole story. The investigators did not evaluate hormone binding or downstream signaling responses, nor did they look at tissue levels of the hormones in question. This latter point is particularly critical, as there is some evidence in the literature for autocrine (same-cell) and paracrine (neighboring cells) trophic stimulation of muscle hypertrophy.
4. The investigators evaluated only very short-term hormone responses (up to 120 min after exercise), and during a single day at the midpoint of a 12-week RT program.
5. The subjects were evaluated during the early novice period, when strength acquisition might be partially due to factors other than hypertrophy (such as neuromuscular adaptation)

Items 2-5, taken together, suggest a very narrow, snapshot assessment of hormonal response to exercise.

Practical Application: There's nothing here to change our training practice, but as I have noted, this paper, flawed as it is, presents a challenge to the way many of us think about hormonal mediation of hypertrophy – and *it could be right*. This question deserves a dispositive study with a well-controlled, in-depth investigation designed specifically to answer it, not a *post-hoc* analysis of data obtained for other purposes.

3. [Strength and neuromuscular adaptation following one, four, and eight sets of high intensity resistance exercise in trained males.](#) Marshall PWM, McEwen M, Robbins DW. Eur J Appl Physiol (2011) 111:3007-16.

Abstract: One of the better papers of the lot comes to us from Down Under, specifically the Universities of Western Sydney and Sydney in Australia and the University of Auckland in New Zealand. The authors note a wide disparity in findings from studies that have attempted to resolve the issue of whether higher volume (more sets) programs increase strength more than lower volume (single set) programs. They point out that much of the literature on this topic compares 1-set to 3-set programs, and suggest that comparison of 1-set to 8-set programs might yield a clearer picture. These authors conducted a proper power analysis prior to beginning the study, they designed the training programs and methods to minimize the most obvious sources of bias, and their methods are well-described. Forty-three resistance trained (and fairly strong) young men were recruited; 11 dropped out during the study (a potential source of self-selection bias). These gentlemen were randomized to 1-set, 4-set or 8-set programs. Sets were performed to “volitional exhaustion.” This 6-week program was followed by a four-week “peaking program” that was the same for all participants. Squat strength, contractile rate of force development, quadriceps strength, integrated electromyography, and anthropometric analyses were conducted at appropriate time points during the course of the study. All trainees got stronger. The investigators found that 1RM squat strength demonstrated greater improvement in the 8-set group compared to the 1-set group throughout the training period. Their findings could not support increased neuromuscular adaptation in any group. Results suggested an impairment of explosive force production (decreases in RFD) using all regimens. The authors conclude that multi-set programming is superior to single- or low-set programming for strength acquisition.

Discussion: I've left out a lot of the details here in the interests of brevity, but this is one of the papers I would invite you to pull and read for yourself. There's a lot to learn from the techniques

and methodologies used here, and the paper's discussion is a thought-provoking read. Lest I sound too effusive, I will say there are significant limitations to the study that must be born in mind when interpreting the findings.

- The study subjects were experienced strength trainees, not novices.
- In all three groups, reps were not uniform; rather, sets were performed to failure.
- On my reading, squats were not performed below parallel.
- Obviously, work volume (total reps x weight) was much higher in the 8-set group, and this raises an interesting question—was it the number of sets or the total volume that caused the difference? Yes, this seems like splitting hairs; after all, you say, *volume was what was being studied*. But was it the *volume* or was it the *sets* that led to differences in outcome? After all, it is conceivable to design an experiment in which the total volume (total sets x reps x average weight) was equalized between the two groups. Or was it the *reps*? Individuals in the 1-set group performed more reps per set (an average of about 11) than the 8-set groups (an average of 7). When we remember that *high reps train for different adaptations than low reps*, we have to ask ourselves whether the results obtained here derive from *more sets*, *fewer reps*, or *more volume* overall. This may be something worth arguing about.
- The lack of measured neuromuscular adaptation to the programs described here is easily attributable to the relatively advanced training of the participants—these guys are well past the point in their training where high levels of neuromuscular adaptation are likely to be observed. One has to speculate that novices might have demonstrated a very different pattern.
- The decline in explosive lower-body power is easily attributable to the lack of cleans, snatches, or any sort of loaded lower-body power training (poorly described jump squats don't count as far as I'm concerned).
- Finally, the investigators performed a sub-group analysis of their three groups into high responders and low responders. Any conclusions they draw here are highly suspect, as this sub-grouping was performed in a completely *post-hoc* manner, and the groups so derived were smaller than the minimum numbers identified in the authors' own power analysis.

One more thing I have to point out. In the methods, the authors allow that they did not manipulate bench press sets and reps for any group, because “we believe that resistance-trained males would be unlikely to adhere to a one-set upper body protocol.” Too funny.

Practical Application: Despite its limitations, this paper manages to rise above most of the strength literature. It provides qualified support for the use of high-volume training to drive strength adaptation, while suggesting future avenues of investigation. Again, I recommend that readers interested in strength science take a look at it—and read it with a critical eye. If you're not careful, you might learn something.

4. [Endurance and resistance training lowers C-reactive protein levels in young, healthy females](#). Daray LA, Henagan TM, Zanovec M et al. *Appl Physiol Nutr Metab* (2011) 36:660-70.

Abstract: From the Departments of Kinesiology, Human Ecology, and Exercise Biology of Louisiana State. The authors note that (1) cardiovascular disease (CVD) is a Really Big Deal; (2) there

all kinds of biomarkers for CVD risk; (3) CRP is one of those biomarkers; (4) exercise reduces risk of CVD disease and (4) exercise *may* (read: *may not*) reduce levels of CRP. Coincidence? *They think not*, and so they got 38 young women to do either endurance training (E) or endurance + resistance training (ER). The young ladies who did strength training got (surprise!) stronger, and they also decreased their CRP. Their body composition did not change. The authors conclude that exercise *may* decrease CRP. In young women. After 15 weeks of training. Or...it *may not*.

Discussion: It's just so tiresome, really. *So what?* Even if we accept that CRP sorts with risk of CVD, there's absolutely no good evidence that CRP *causes* CVD. And even if CRP did have some role in the causation of CVD, there's no good evidence that decreasing CRP, with exercise or anything else, would prevent or even slow the development of CVD. This study is just not helpful: wrong hypothesis, wrong population, wrong end points. A classic case of confusing a change in a disease marker with a meaningful benefit...except in this case they even chose a lousy disease marker.

The best that can be said of this paper is that it allows me to highlight a recurring theme:

It doesn't matter whether we're talking about exercise, or a pill, or acupuncture, or yogurt, or any other damn thing. *Changing somebody's test results is not the same as making them healthier.*

Practical Application. None.

5. [Acute loading and aging effects on myostatin pathway biomarkers in human skeletal muscle after three sequential bouts of resistance exercise.](#) J Gerontol A Biol Sci Med Sci (2011) 66(8):855-65.

Abstract: Myostatin is a member of the transforming growth factor-beta (TGF-beta) family of proteins, a product of the *MSTN* gene. As its name suggests, myostatin antagonizes muscle hypertrophy and promotes muscle atrophy. Naturally, the biology of this protein is of profound interest to investigators working on sarcopenia, muscular dystrophies, and other myopathies and wasting diseases. Myostatin binds to an activin-type receptor, which ultimately results in the activation of SMAD-family transcription factors, which bind to DNA and promote the muscle-wasting effects. One may (very simply) think of myostatin as the *Yin* to IGF-1's *Yang*. In this study, the authors compared the effects of very short term resistance training on myostatin mRNA expression in both young (av 28 yo) and "old" (av 68 yo) men. They found that both younger and older men were able to mount a significant down-regulation of myostatin mRNA. They also report that older men demonstrated higher levels of gene products that inhibit myostatin, and lower levels of the phosphorylated form of SMAD-3, a signaling intermediate that promotes the effect of myostatin. The authors conclude that older men demonstrate a paradoxically more favorable myostatin-related response to strength training than younger men, and speculate that this may be part of a compensatory mechanism to preserve muscle mass during aging.

Discussion: Anybody who lives in my demographic would find these results encouraging. Unfortunately, we can only take so much from this study. There are a number of limitations, but on my read two big ones stand out:

- The authors measured *MSTN* mRNA, using PCR of muscle biopsy tissue. Well and good: that tells us that *MSTN* was being *transcribed*. But it doesn't tell us whether, or to what degree, that mRNA was being *translated* into actual myostatin protein. It's the protein, after all, that does the dirty work. The authors took the time to do protein chemistry (Western blots) for phosphorylated SMAD3. They could just have easily done

Westerns for myostatin. It must have occurred to them. If they did do Westerns for myostatin...where are they? If they didn't...well, why the hell not?

- There's no assessment of body composition, muscle hypertrophy, muscle degradation, muscle protein synthesis, fiber shifting, strength, or any other clinically significant endpoint. Yes, I understand that this is a basic science study. But the authors tested to see whether an intervention (RT) changed gene expression (it did), but didn't bother to check whether that change had any effect whatsoever on the target tissue.

Practical Application: We can't take too much from this paper, but at least it's not bad news. The most charitable interpretation is that these authors have given us another data point suggesting that older individuals who engage in strength training can exert profound effects on their biology at multiple levels, including gene expression.

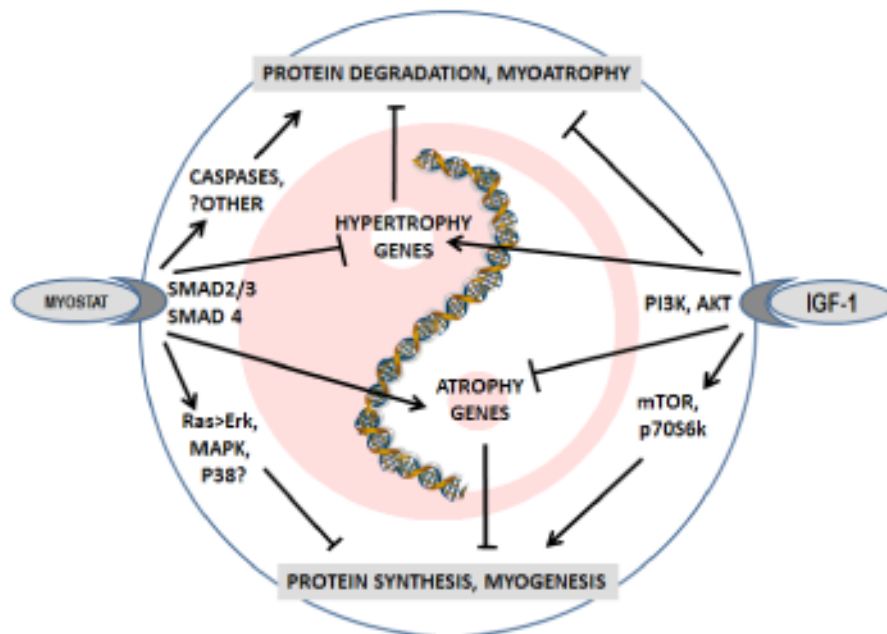


Figure 2: Myostatin vs. IGF-1, an example of antagonistic cell signaling processes. Arrowheads indicate a target process that is promoted, hammerhead lines indicate a target process that is inhibited. IGF-1 binds to a cell-surface growth factor receptor and acts through a number of multi-step signaling pathways to promote myogenic gene expression and protein synthesis. Myostatin binds to a cell-surface avidin receptor and acts through a number of multi-step signaling pathways to suppress myogenic gene expression and suppress protein synthesis. (Illustration by Sullivan.)

6. [The effect of rest interval length on multi- and single-joint exercise performance and perceived exertion.](#) Senna G, Willardson JM, de Salles BF, et al. JSCR (2011) 25(11):3157-62.

Abstract: In this Brazilian-American collaboration, the investigators studied the effect of different rest intervals (1, 3 and 5 minutes) on the performance of a multi-joint (bench press) and several single-joint (machine) exercises. They studied a small cohort of resistance-trained men. The

subjects could perform more reps if they rested longer. This phenomenon was more pronounced for machine (single-joint) exercises than for the bench press.

Discussion: Wow. Turns out you can do more reps if you rest longer. And multi-joint exercises are different from single-joint exercises.

Practical Application: When you catch your breath, recover from the shock and utter surprise at these results, and take a few minutes to think about them, my guess is your training practice won't change very much.

Also Ran:

[Changes in bone mineral density in response to 24 weeks of resistance training in college-age men and women.](#) Almstedt HC, Canepa JA, Ramirez DA, Shoepf TC. JSCR (2011) 25(4): 1098-1103. Another study showing that resistance training increases bone density. No surprises here, and the study was conducted in a population that doesn't have a heavy burden of osteoporosis.

[Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training.](#) Chen JL, Yeh DP, Lee JP, et al. JSCR (2011) 25(6):1546-52. A Taiwanese observational study of heart rate variability and DHEA secretion in weightlifters after a heavy bout of exercise. Interesting read; no surprises.

[Effect of different resistance-training regimens on the WNT-signaling pathway.](#) Leal MC, Lamas L, Marcelo SA, et al. Eur J Appl Physiol (2011) 111:2535-45. Another heavy cell-signaling paper, this one from Brazil. WNT is a signaling molecule that appears to mediate the response to overload in skeletal and cardiac muscle, through mechanisms that are not entirely clear. The authors showed an increase in WNT signaling in 25 subjects on a strength training program.

[Effect of resistance exercise on serum levels of growth factors in humans.](#) Vega R, Knicker A, Hollman W et al. Horm Metab Res (2010) 42(13):982-6. Eleven healthy subjects mounted significant increases in IGF-1 and brain-derived neurotrophic factor after heavy exercise. An interesting contrast to West's paper.

NUTRITION, SUPPLEMENTS AND DRUGS

1. [Nine weeks of supplementation with a multi-nutrient product augments gains in lean mass, strength, and muscular performance in resistance trained men.](#) Schmitz SM, Hofheins JE, Lemieux R. J Internat Soc Sports Nut (2010) 7:40.

Abstract: I made allowances for papers from late 2010 that popped up in my selection process to be included in this maiden voyage; this is one of only two that made the cut. This is a manufacturer-supported trial of a proprietary product. The product in question, SOmaxP, contains carbohydrate, whey and creatine along with proprietary ingredients. The investigators randomized 20 strength-trained men to either this product or a control composed of equal amounts of the core ingredients, but without the propriety substance. The product or control preparation were ingested before and during

workouts on training days only. Strength was assayed by 1RM bench press and repetitions to failure (at some percentage of 1RM that is not clear). Body composition and a laboratory profile that included complete blood cell count, electrolytes, renal and liver function, and serum lipids were also obtained. The investigators report that both groups gained 1RM strength and reps to failure. Body fat decreased in the S_OmaxP group but not the control group. Laboratory profiles were unchanged.

Discussion: Manufacturer-supported studies should always give you the heebie-jeebies. The lead author of this study is a paid consultant for the proprietor, Gaspari nutrition. Articles like this are not science. They're *product testing*. If you want to call it science, I need to be able to read your report and replicate your experiment. I can't do that with Secret Formulas. As near as I can tell from online research and ad copy, the Secret Formula may include pterostilbene, a resveratrol-like substance that acts as an insulin secretagogue. No assessment of insulin secretion or insulin signaling was attempted in this study.

Setting all that aside, there are some big problems with the data. We are given the mean changes for the *groups*, when it would have been quite an easy matter, and more proper, for the authors to have presented both the mean group differences and the *individual changes* for such a small cohort (see the **Summary**). This is important, because as it stands we don't know the extent to which the (rather small) mean differences between these two groups is accounted for by outliers within the groups.

Although the trainees performed a dozen different exercises, including the bench press, chest flies, shoulder presses, curls, pressdowns and leg extensions among others (not a squat to be found), *only data for the bench press is presented*. Why is that? I leave you to speculate.

Practical application: *Caveat emptor.*

2. [Creatine but not betaine supplementation increases muscle phosphoryl-creatine content and strength performance](#). Favero S, Roschel H, Artioli R et al. Amino Acids (2011) epub ahead of print.

Abstract: The ergogenic effect of creatine (Cr) supplementation is well-established in the literature, and in my opinion most serious lifters who don't use it are missing out on an edge (although there is evidence that some people are "non-responders.") The mechanism is probably familiar to most of us, but I'll review it briefly. Creatine, ingested as a supplement or in the diet, is avidly taken up by muscle, brain and renal tissue, where it is phosphorylated by the enzyme creatine kinase, allowing the tissue to maintain a localized high-energy Cr-PCr cycle to mitigate the depletion of ATP. ADP left over from energy consumption can be rapidly and locally re-phosphorylated by transferring phosphate from phosphocreatine (PCr), without the need to be shuttled back to the mitochondrion (in aerobic cells) or await ATP production by glycolysis. This system therefore provides both temporal and spatial buffering, and extends the availability of high-energy phosphate beyond the few seconds that would be possible with ATP alone. Recent data suggests that betaine (Bet), a methylated amino acid (glycine) derivative, also has ergogenic properties, due at least in part to its ability to donate methyl groups for Cr synthesis. Several reports in the last few years have suggested that betaine supplementation increases muscle Cr content and improves performance.

The authors of this study, from the Department of Radiology at the University of Sao Paulo, Brazil, investigated the effects of supplementation with Cr, Bet, a combination regimen (Cr+Bet), or placebo on muscle PCr content and performance in 34 untrained or detrained men. The study was conducted in a placebo-controlled, blinded, randomized manner. Power output (on the bench press), 1RM squat strength (on a Smith machine) and 1RM bench press were assayed before and after a 10-day supplementation protocol, *during which the subjects did not engage in any resistance training*.

Muscle PCr was assayed noninvasively in the calf by magnetic resonance spectroscopy. The authors report increased levels of muscle PCr in subjects taking Cr or Cr+Bet, but not in those taking placebo or Bet alone. They also report improvements in power output and squat/bench 1RM in those taking Cr or Cr+Bet, but not in those taking placebo or Bet alone. Body composition was also assayed and no changes were noted. The authors conclude that betaine does not augment muscle PCr content or improve performance.

Discussion: It may be true that betaine neither increases muscle phosphocreatine nor improves performance, but this paper certainly doesn't prove it. I'm not particularly confident in the NMS assay of muscle PCr content, but let's take that data at face value. The use of 1RM as an assay of strength in untrained subjects is definitely problematic. The authors claim that their data contradicts other reports on betaine supplementation, but those studies typically supplemented betaine for 14 days, not 10... would it have been so difficult for the authors to have matched their study interval to those of previous reports?

But forget all that. The two really big problems here are (1) the *complete absence of training* during the study interval and (2) *the astonishing failure to measure both poles of the Cr-PCr cycle*. These two problems are synergistic. It is trivial to hypothesize that betaine either increased muscle Cr in these subjects, or *would* have if they had trained. But we don't know either way, because the investigators didn't look. Similarly, it's more than reasonable to speculate that, if betaine did increase muscle creatine in these subjects, it did not result in increased phosphocreatine. Because, after all, the subjects didn't *need* increased phosphocreatine, *because they didn't train*. Both of these possibilities would have been ruled out easily with a properly-designed study. The lack of a training group and the lack of a muscle creatine assay are lethal to this study.

Practical applications. None. The information here is so compromised it shouldn't change anybody's practice. I don't think the evidence for betaine is particularly compelling yet, and this paper doesn't add to it either way.

For those of you interested in the creatine phosphagen system, I recommend an excellent review, also published this year in *Amino Acids* (40:1271-96) by Wallimann et al. See also the excellent article by Matt Reynolds, [Death by Prowler](#).

3. [Minimal effect of acute caffeine ingestion on intense resistance training performance.](#) Astorino TA, Martin BJ, Schachtsiek L, et al. JSCR (2011) 25(6):1752-8.

Abstract: This small study from California State University looked at the effect of caffeine on the performance of 14 resistance-trained men who regularly consumed caffeine. These poor bros were asked to *abstain from caffeine intake for 48 hours* prior to testing. Horrible. Real Josef Mengele-type stuff. On the day of testing, they were given either a caffeine tablet (6mg/kg) or a placebo, and their performance (measured as the number of reps they could perform on sequential sets at 70-80% of 1RM) was assayed on the leg press, bench press, lateral row and shoulder press. Salivary caffeine levels were sampled throughout the experiment and assayed by HPLC. The experiment had a crossover design, and subjects returned in one week to repeat the protocol with caffeine or placebo, as appropriate. The investigators found that the effect of caffeine on performance in these individuals was minimal. A *statistically* significant but *practically* insignificant difference in the leg press was observed, and only on the first set. The other exercises did not demonstrate a difference between caffeine and placebo. The authors conclude that any effect of caffeine was minimal.

Discussion: Don't throw out your favorite mug just yet, gang. We've got more problems here than a Bigby's has beans. This small study investigated the effect of a *caffeine tablet* administered to men who were, to use the technical lingo, Java Junkies, shortly before a heavy workout and after two days of caffeine withdrawal. Well...*that's just not natural*, and certainly doesn't model the way actual decent red-blooded human athletes use caffeine. But setting aside the experimental design for a moment, I think the investigators' conclusion really got cooked by the caffeine assays. Here's how: On the day of the experiment, subjects showed up dragging ass from two days of decaffeinated decrepitude, and were given a little pill. They were then asked to sit quietly for 35 minutes. Then they walked a half mile to the gym (their "warmup;" call it another 25 minutes). They then engaged in the prescribed testing protocol, which lasted an hour. Post workout caffeine levels were drawn at 3 and 8 hours. Then, presumably, everybody went to Starbucks.

So – the workouts were all over within two hours of caffeine/placebo ingestion. But salivary caffeine concentrations peaked at *three hours* after ingestion. This may seem like a long time to wait for a jolt, but remember that the subjects took their caffeine in pill form. They weren't drinking a hot, delicious, rapid-delivery caffeine solution like real people do. The point is that, on my reading, caffeine peaked *after the workout was over*. It's just too horrible to even think about.

Practical Application: This paper doesn't convince me. Besides...there's more data on this *critically important topic* coming up next. Let me get a warmup and we'll get right to it...

4. [The effect of caffeine ingestion on mood state and bench press performance to failure](#). Duncan MJ, Oxford SW. JSCR (2011) 25(1): 178-85.

Abstract: This paper comes to us from the Department of Biomolecular and Sports Sciences at Coventry University in the UK. The investigators sought to assay the effect of caffeine on a strength performance task, and also to *get in touch with the lifters' feelings*. The study again had a crossover design. Thirteen college athletes with lifting experience were recruited, and their strength assessed as 1RM bench press. Their dietary intake was monitored and they were to *abstain from caffeine after 6pm the night before testing*, which took place at 9am. Sixty minutes prior to testing, subjects received either a caffeine beverage made of a standardized dose of caffeine (5mg/kg) in artificially sweetened water, or a placebo of sweetened water. Performance was assessed as the number of reps to failure and the total volume lifted. Rating of perceived exertion (RPE), and mood were assessed. Serum lactates were also measured. The investigators report that caffeinated bros completed more reps to failure and lifted significantly greater weight than decaffeinated bros, that their vigor and fatigue scores were more salutary, and that their serum lactates and RPEs were not significantly different. They conclude that caffeine improves performance in resistance exercise. And it *makes you feel good about it, too*.

Discussion: Those of us who have Dedicated Our Lives to the Methylxanthine Sacrament will be tempted to say, "Oh, *hell yeah*." But this study, while less problematic than the Astorino, still has significant shortcomings. The differences in reps and volumes lifted with caffeine may have been *statistically* significant, but their *practical* significance is arguable. Decaffeinated dudes completed an average of 20.4 + 3.4 reps, while in their caffeinated condition they completed 22.4 + 3 reps. I have to say I'm a bit underwhelmed. Total weight lifted looks a little better, but still not great when you look at the confidence intervals: 1039 + 231.7 kg decaff vs. 1147.2 + 261.4 kg caff. The findings on mood and RPE come with all the usual caveats applying to assays of subjective states. At least these investigators didn't drive their subjects into dysfunctional despair by depriving them of life-giving caffeine for two

whole days, and they used a caffeine delivery method that more closely approximated the one used by most sentient life forms.

As with the previous study, the real problem here has to do with their serum caffeine measurements. As in: *these guys didn't do any*. Seriously, they popped for serum lactate, but not for serum caffeine levels? It boggles.

Practical Application: The literature to date on caffeine for strength training is conflicted, but on my reading it suggests, on balance, a positive ergogenic effect that most of us should exploit. My advice, based on the flawed data we have, is this: If you use caffeine when you lift, then *for God's sake don't stop*. If you don't, please consider trying it.

5. [The anabolic 500: Characteristics of male users versus nonusers of anabolic-androgen steroids for strength training](#). Ip EJ, Barnett MJ, Tenerowicz MJ, Perry PJ. *Pharmacotherapy* (2011) 31(8):757-66.

Abstract: The authors, from the Touro University College of Pharmacy in California, sought to “contrast the characteristics” of male weightlifters who reported anabolic-androgenic steroid (AAS) use versus those who did not. Respondents were recruited from Internet strength and bodybuilding discussion boards to fill out the Anabolic 500, a web-based survey composed of 99 questions. *The survey questions are not included in the published manuscript*. Of 2380 survey “attempts,” only 1277 could be used. Of these, 506 respondents were AAS users. After milking the resultant data six ways from Sunday, the authors report that most AAS users were recreational athletes, and were more likely to meet criteria for substance dependence disorder, more likely to use cocaine, and more likely to report a history of sexual abuse.

Discussion: All we *really* know from this study is that there are 506 guys who were hanging out on certain unidentified Internet Bro Boards *and* found the recruitment notice *and* completed the survey *and* whose survey got used *and* who claimed to be AAS users. And *those guys* had a slightly higher incidence of meeting the authors' criteria for substance abuse disorder, of claiming to have a history of sexual abuse, and of admitting to cocaine use, when compared to 771 similar bros who *didn't* admit to being AAS users.

I include this paper because it's certain to be used to support any number of extravagant claims: “*Sexual abuse victims grow up to use steroids!*” “*Steroids cause anxiety disorder!*” “*Steroids are a gateway to crack!*” But this paper can't support any such claims, because it can't support *anything at all*. It's an observational study, and a poor one, at that. The use of anabolic steroids is an incredibly important topic, with potentially profound medical implications. It warrants much better study than this.

Practical Application: None.

Also Ran:

[Caffeine intake improves intense intermittent exercise performance and reduces muscle interstitial potassium accumulation](#). Mohr M, Jens JN, Bangsbo J. *J Appl Physiol* (2011) 111:1372-79. For those who just can't get enough Java Science, here's an extra shot. (By the way, this is one of the few papers in the bunch with a proper title – that is, a declarative statement of what the authors actually concluded, rather than a vague and noncommittal description of the topic.)

TRAINING AND COACHING TECHNIQUES AND SPORTS MEDICINE

1. [Spontaneous coronary artery dissection after intense weightlifting](#). El-Sherief K, Rashidian A, Srikanth S. *Catheter Cardiovasc Interv* (2011) 78:223-7.

Abstract: This is a case report from the Department of Cardiology at UCSF Fresno. They describe the case of a 29-year-old male with no previous cardiac history who had recently started weight lifting. During a bout of exercise, he developed *severe* chest pain and associated symptoms. So...he took some Prilosec and waited for it to go away. Next workout it happened again. So...he took some Prilosec and waited for it to go away. Ultimately, he presented to the emergency department, where his physical exam revealed no marked abnormalities. EKG was nondiagnostic. Tox screen was positive for cannabis (shocking!). Ultimately, cardiac cath demonstrated a dissection of the left anterior descending coronary artery.

Discussion: A dissection occurs when the inner lining of an artery develops a defect, allowing a column of blood to dissect into the wall of the artery, creating a false lumen. The dissection can propagate down the artery and lead to any of a number of nasty scenarios, including complete obstruction of the artery (resulting in tissue death) or free rupture of the artery (really, *really* bad news). Our pot-smoking bro required stenting of the artery and antithrombotic management, and was ultimately released from hospital.

Practical Application: The first take-home message is that *this is an exceedingly rare phenomenon*, as the authors themselves are at pains to point out. Of course, this is the exact opposite of what Some People will conclude when they scan the abstract. The rarity of such catastrophes in the weight room is reflected by the fact that they always pop up in case reports, never in large series. Basically, *this kid got struck by lightning*.

The second take-home message is: Don't do what he did, and blow off severe pain that develops during a workout. *Severe* head, neck, or chest pain, and/or any form of physiologic distress (prolonged shortness of breath, loss of consciousness, confusion, parts don't work, etc.), deserves investigation. People *do* get struck by lightning, and other serious injuries are more common than dissection, especially in older lifters. Don't be dumb.

2. [Effect of verbal instruction on muscle activity during the bench press exercise](#). Snyder BJ, Fry WR. *JSCR* (2011) epub ahead of print.

Abstract: This paper comes to us from the University of South Carolina and Shenandoah University. Eleven male Division III football players were subjected to 1RM testing on their bench. They were then brought back to the lab a few days later and performed three sets at 50% 1RM and three sets at 80% 1RM while EMG recordings of the pectoralis major (PM), triceps (TB) and anterior deltoids were recorded. On the second set at each weight, the subjects were instructed to use only their chest muscles. On the third set, they were told to use only their arms. Normalized root mean square EMG activity demonstrated increases of PM activity over the pre-instruction state when subjects were told to lift only with the chest at 50% 1RM, while TB and AD activity remained unchanged. When told to use only their arms, PM activity returned to the baseline determined in the first set, but increased TB activity was noted. At the heavier load, instructions to use the chest only increased

firing of the PM and AD. TB did not change at the heavier weights. The authors conclude that verbal instruction effectively shifts muscle activation, but may be less effective at higher weights.

Discussion: Compared to most of the strength literature, this paper is well-executed, and is consistent with previous findings. The authors should not, of course, be surprised that the ability to shift muscle firing patterns is less pronounced at higher weights; a heavier lift requires more input from all muscle groups. I'd like to see similar work done with the other lifts, and with both novice and experienced lifters.

Practical Application: Basically, this paper is about the effect of *cues*, and reminds us of their power and importance. It's a demonstration of the tremendous effect that coaching cues can have on the biomechanics of a lift. Anybody who's ever attended a seminar is aware of the great importance the SS model places on proper use of verbal, visual and tactile cues, for precisely this reason. When we cue a lifter, we are focusing her motor attention, and altering the way her nervous system activates her motor units, with profound implications for which muscles get involved, to what degree, and when. Of course, this is a power that can be used for either Niceness or E-vil. Learn, and cue wisely.

3. [Relationship between functional movement screen and athletic performance](#). Parchmann CJ, McBride JM. JSCR (2011) 25(12):3378-84.

Abstract. The Functional Movement Screen (FMS) is a seven-item test developed by Lee Burton and Gray Cook in the mid 90s. It's supposed to identify deficits in athletic performance and the potential for injury. Some authors, in particular Okada *et al*, have reported that the FMS correlates with athletic performance. Many more studies have shown that 1RM back squat also correlates with athletic performance. The authors of this study compared the correlation with athletic performance of both the squat and the FMS. Twenty-five young NCAA Division I golfers (15 men, 10 women) were subjected to 1RM squat and FMS testing. Athletic performance was assayed by vertical jump, 10- and 20 m sprint protocols, and the agility T-test. Sport-specific performance was assayed by computer-assisted measurement of maximal club head swing velocity. Squat 1RM demonstrated excellent linear correlation with sprint times, vertical jump, agility T-test, and club head swing velocity. FMS, on the other hand, produced "scattergrams," with *extremely* poor correlation coefficients. The authors conclude that FMS is "not an adequate field test," and note that their findings confirm those of other investigators that 1RM squat is a good indicator of athletic performance.

Discussion: The FMS is much-beloved of PTs and RTs, and it's a cash-cow, too. To see for yourself, just use The Google, or point your browser to <http://www.functionalmovement.com> or any of the myriad other sites where you can learn about the FMS, register for an FMS workshop, set up an FMS consultation (Visa and MasterCard accepted), get certified to give the FMS, etc. At a recent SS seminar, the issue came up during the Q&A, whereupon an animated discussion ensued at length. In fact, the point at issue in that conversation was not entirely dissimilar to the question addressed in this paper: how to screen trainees for the ability to follow the SS program? One party felt that the FMS would be an ideal screening instrument. Another party, whose name rhymes incongruously with Tip-Toe, argued that the ideal screen for the ability to perform squats and deadlifts would be to have the trainee perform squats and deadlifts. The present results would seem to lend support to the latter view.

It is important to note, however, that the study has several limitations. No power analysis is documented. The data set is small, and is limited to healthy young athletes, all of whom were engaged in a strength training program being administered by one of the investigators – a potential source of bias. The FMS in this study was administered by a single investigator – another source of

bias. A more robust and proper approach would have been to have multiple investigators administer the test and demonstrate that the FMS scores so produced were reproducible. Finally, while vertical jump and sprint times are generally applicable surrogate markers of athletic performance, and while the T-test seems reasonable, the findings for club head swing velocity limit conclusions about sport-specific performance.

Practical Application: Despite its limitations, the study is definitely a black eye for the FMS. The most important take-home point is the robust correlation of squat strength with several measures of athletic performance. Now it's time for somebody to compare 5RM squat to 1RM squat as a performance screen, the hypothesis being that 5RM would be just as robust, but more generally applicable – that is, useful in both trained and untrained populations.

Also Ran:

[Injuries and overuse syndromes in powerlifting](#), Siewe J, Rudat J, Rollinghoff M, et al. In J Sports Med (2011) 32(9):703-11. Another survey-based study, with all the major caveats that pertain. The most common injuries were to the shoulder, low back and knee. In this study, belts seemed to increase the rate of injury, but the overall injury rate was extremely low.

ORTS AND LEAVINGS

1. [Effects of self-selected music on strength, explosiveness and mood](#). Biagini MS, Brown LE, Coburn JW, et al. JSCR (2011) epub ahead of print.

Abstract: The authors compared the effect of self-selected music (SSM) vs. no music (NM) on mood and performance of the bench press and squat jump. Subjects were resistance-trained college males who were tested under both SSM and NM conditions. They reported their profile of mood state (POMS) and rating of perceived exertion (RPE) before and after three sets to failure on the bench press and 3 reps of the squat jump at 30% 1RM. Bench press performance and RPE were not affected by SSM, but performance on the explosive exercise and POMS were both influenced positively. The authors conclude that SSM might be beneficial for acute power performance.

Discussion. The study is small, with a very specific population, but the methods and analysis here are acceptable. This is not the first paper on this subject. The authors cite multiple previous studies supporting the use of music as an ergogenic “supplement” – and some that do not. The current study is hardly dispositive – they conclude that SSM might be beneficial, and this conclusion necessarily encompasses the alternative – it might not be.

Me? I'm *still* waiting for the peer-reviewed, randomized, controlled, large cohort, NIH-funded long-term longitudinal study that compares symphonic Beethoven, Power Metal, and Country Rap.

Practical Application. Come on. *Music helps*. We all know that. Now you have the Blessings of Science. Crank it.

SUMMARY

It's time for a major disclaimer: I'm new to strength science. I am a working scientist and an academic physician, and I know how to read the biomedical literature, but my own professional disciplines encompass different knowledge bases and skill sets than the ones covered in the foregoing papers. I have learned much in preparing this review, not least about my own ignorance.

That all being said, I believe that anybody can and should learn to read the biomedical literature critically. It doesn't take a degree. All it takes is intelligence, knowing how to ask the right questions, a willingness to learn, and a healthy dose of skepticism.

On my reading, strength science is in an odd situation. On the one hand, the importance of strength is more universally recognized than ever, and investigators have access to an arsenal of incredibly sophisticated and sensitive tools, not to mention a modern picture of living matter that allows integrated modeling of the organism's response to strength training at multiple levels, from gross biomechanical descriptions to systemic and tissue-level responses to subtle alterations in cell signaling and gene expression.

And yet, the literature is still dominated by small studies with little power, addressing poorly focused questions with little practical relevance, often with sloppy experimental designs and statistical methods. Excellent work is being published, but is more the exception than the rule. I am optimistic about the long-term outlook for this field, but its present problems are great.

For example, a cursory survey of the literature on strength in humans reveals that there is no generally accepted or uniform method for assaying strength. One group uses bench press, another uses some variant of the squat. Very many just opt for the ubiquitous leg press. Some use 5RM, some use reps to "volitional exhaustion," and a great many use 1RM, even in studies with novice trainees, where it is almost certainly inappropriate. In other words, strength scientists *haven't even settled on a uniform way to measure the subject under study*. This difficulty is not unique to strength science, but it is a difficulty, nonetheless. Another difficulty is the lack of documentation of exercise performance across all types of studies. There's no excuse for this in 2011. It is now very common for investigators in other fields to make ancillary data available online, including video data. Here's [an example](#). Here's [another](#). But the exercise science literature doesn't seem to have caught on to this idea. There are more exercise videos (form checks and whatnot) *on the Starting Strength Forum* than I encountered *in my entire survey of the literature for 2011*. When the authors of a paper on, say, the kinetics of the power clean or a comparison of squats to leg presses don't include representative documentation of *exactly* how those exercises were performed, and what constituted acceptable vs. non-acceptable repetitions, then as far as I'm concerned they're withholding data.

A great many studies are published suggesting that a particular exercise or programming variation will change some measure of strength or performance, but there is virtually no data showing how such interventions actually affect performance in the field. Yes, obtaining this sort of data will be difficult, and it will begin with the very tough but necessary task of establishing meaningful sport-specific performance metrics for each sport. Science is *supposed* to be hard.

A related problem, one that came up repeatedly in this review, is the literature's stubborn focus on disease-oriented outcomes or biological markers instead of practical measures. It's all very good that having old ladies squat decreases their CRP, or bumps their IGF-1, or whatever. This information is important, as far as it goes. But the real question is whether squatting improves their quality of life, decreases their risk of falls or hospitalization rates, makes old lady sex better, you name it. Again, this

data is much more difficult to acquire, because you can't get it from studying a dozen golden gals for three months. Well, *too bad*. Science is *supposed* to be hard.

If I had to put my finger on the single biggest problem with the science of strength, it would be that studies are overwhelmingly *small* and *short*. The typical study enrolls 10-20 subjects and studies them for 12-16 weeks. This means that most studies are poorly powered to detect subtle effects, and that they cannot possibly evaluate the effects of long-term, rational programming. Three or four months wouldn't be enough time to do a half-decent observational study of the *novice progression*.

Small studies are not completely without value, but the data has to be interpreted with caution and presented correctly. One very common genus of strength science paper takes a small group of subjects and imposes a particular intervention, an RT program or a drug, and looks at how it changed the subjects. For example, let's say we do our own study. We recruit 12 guys, measure their 1RM bench, and randomize them into two groups. We put both groups on a two-week exercise program. Group 1 gets a placebo, while Group 2 gets Sullydawg's Power And Mass Extreme! (SPAM-X!™) supplement. At the end of the study we get a bunch of data: 1RM, 5RM, body composition, reps to failure at 60%1RM, you name it. Most of this data shows no change whatsoever, but the results at 60% 1RM look impressive, so that's what we publish. The negative data, mysteriously, doesn't make it into our paper.

Our published article contains the following graphical presentation of the data:

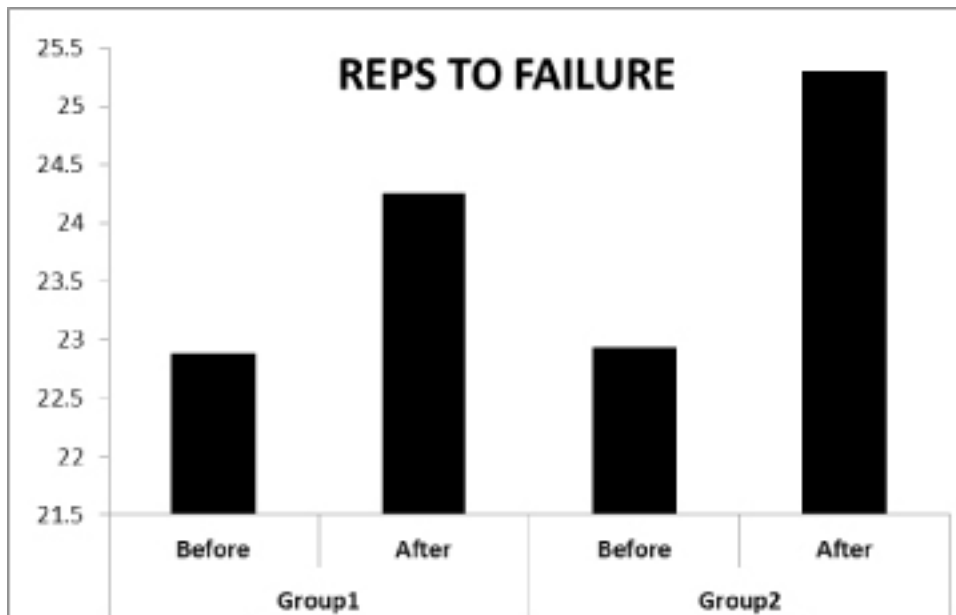


Figure 3: SPAM-X!™ markedly improves repetitions to volitional exhaustion (RTF) on the 60%1RM bench press compared to placebo. Shown are mean RTFs before and after ten days of training.

Now, this looks impressive. Y'all had better go out and get some SPAM-X!™ right away, because I want a new Jag and this product is clearly the best thing to happen to strength training since the invention of the EZ-Curl Bar.

Except it's not. Here's another way of presenting exactly the same data, showing what actually happened to each participant in the study:

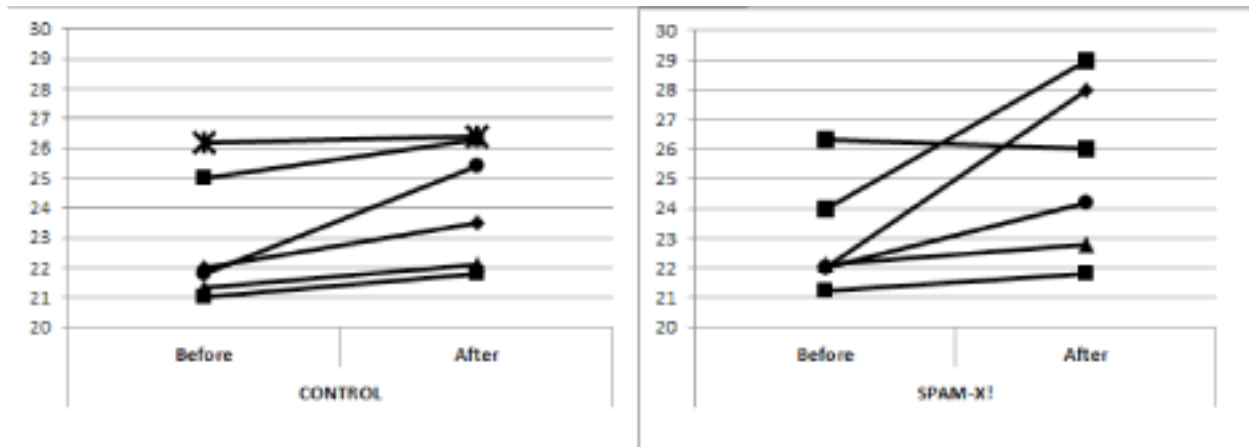


Figure 4: SPAM-X!TM markedly improves repetitions to volitional exhaustion (RTF) on the 60%1RM bench press compared to placebo. Shown are reps for each subject before and after 10 days of training, supplemented by placebo (control) or SPAM-X!TM.

This tells a different story. We can see that both groups got stronger, and more importantly that *all the difference between the two groups at the end could be accounted for by two individuals, and that the only guy who actually got weaker was in the SPAM-X!TM group*. This is the kind of stuff that gets buried in the tried-and-true bar graph. Of course, sorting out skews and biases in the data is where statistics are *supposed* to help us. But that's a whole other steaming pit of malfeasance and dirty little secrets. Maybe we'll talk about it next year.

(The astute reader will have noticed from the legends in Figures 3 and 4 that, despite very different pictures of the data, the conclusions didn't change. The authors can count on the fact that *most* readers will skim the abstract, *maybe* read the figure legends and just...swallow.)

Many of the small studies of the type that predominate in the strength literature are ideal for this second kind of paired data presentation...but they seldom use it. *Why is that?* I leave you to speculate.

Because strength studies tend to be small and short-term, and because there's so much emphasis on observational research and biological end-points, the really important questions aren't being answered:

- What kinds of exercises and programming methodologies drive *long-term* increases in strength, performance and health?
- What are the long-term benefits and complications of resistance training?
- How can strength training be best applied to special populations?
- What are the most sensitive biomarkers for adaptation, the supercompensation "window," and overtraining?
- For any given sport, what are the most meaningful metrics for sport-specific performance, and how are they affected by changes in training and programming variables?

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- What factors influence training compliance? Why do people start training? Why do people quit training? What variables, if any, can be manipulated to improve long-term compliance with training regimens?

I'm sure all of you can think of more.

So, that's my sampling of strength science in 2011. I cannot and do not claim that my sampling was unbiased or comprehensive – it was naturally limited by time and length and reflects my implicit selection process. Again, this was an experiment, a trial run to see if this sort of thing has value to the community. If it does, we'll have a year's lead time heading into the next one, which will allow us to take a bigger and better cross-section of the field. In the meantime, your feedback is welcome, and if you come across papers that might deserve a spot in next year's review, please forward them to me via personal message. I look forward to hearing from you. Have a Strong and Happy New Year.

Jonathon Sullivan is Attending Physician and Associate Director of the Cerebral Resuscitation Laboratory in the Department of Emergency Medicine at Wayne State University/Detroit Receiving Hospital. His research focus is on molecular mechanisms of neuroprotection with growth factors and hypothermia. He is a graduate of the University of Arizona (MD) and Wayne State University (PhD, Physiology). He is a Starting Strength Coach and *Krav Maga* practitioner, and holds a Third Degree Black Belt in *Tang Soo Do*. He started lifting seriously at the age of 50, to save some muscle for retirement.

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