The Year in Strength Science
2012

by
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Introduction

A review of the strength science literature for 2011, the first in this annual series, was conceived in the Autumn of that year, gestated quickly, and got pushed out in early January 2012. This second review has had a year to incubate, and in the interim I’ve tweaked my search parameters and expanded my net. The result is that I’ve been able to capture many more relevant articles for the review. Last year’s review captured about 300 articles, of which 30 were presented, with capsule summaries of some also-rans. This year my literature searches and Citetrack subscriptions captured over 1200 articles, and I am presenting almost 150 papers in this review.

This has forced me to alter my approach. Last year, the review dissected each article individually and in a certain degree of depth, with a considerable amount of focus on methodology, data analysis and conclusions. Using that same format this year would have led to a 200-page article. Upon due reflection, I calculated that an article of such length would appeal to exactly nobody, least of all me. Instead, I will review individual areas of research in the strength science literature and how they were addressed by studies published during the last year. Studies that were particularly important will still get special attention, but overall the style of the article will be, I hope, more readable and less “chunky.” I’ll be looking forward to your feedback. Hopefully this year’s approach will be seen as an improvement and a natural evolutionary step in this project. If not, let me know, and I’ll retool my approach.

I approach the literature using categories similar to those I used last time:

I. Biomechanics
II. Exercise Physiology
III. Big Medicine
IV. Programming
V. Training, Coaching and Performance
VI. Sports Medicine
VII. Nutrition, Supplements and Drugs

Methodology

To find candidate articles, I once again conducted searches in both PubMed and Google Scholar, and set out my nets in Citetrack, using search terms like squat, bench press, power clean, snatch exercise, anthropometry, weightlifting, injury, strength training, resistance training, biomechanical analysis, etc., in various combinations, constraining my searches to papers published in 2012. The voluminous results were subjected to a first-pass triage that eliminated most of the candidate articles. For those that remained, PubMed identified still more, as related citations, which underwent a similar winnowing. Unlike last year, however, I was able to expand my net by following particular journals, most notably the Journal of Strength and Conditioning Research, the Journal of Sports Medicine, the European Journal of Biomechanics, the Journal of Applied Physiology….the Journal of This, the Journal of That, and the Journal of the Other Thing.

Yes. It was mind-numbing. Articles in these journals were also subjected to triage. Finally, there were a few articles posted to the Starting Strength forum over the course of the year that drew my attention. Some of these made it into the mix.
This process yielded about 1200 post-triage candidate studies. These all got a second look, from which I ultimately selected about 150 articles of interest. Once again, this selection was necessarily implicit and practical. Articles that I found unreadable, inaccessible, or uninteresting were discarded. I kept and reviewed the articles that I thought would be relevant and of interest.

Here they are.

I. Biomechanical Analysis

Biomechanics is, to put it most simply, the physics of biological movement. This category examines kinematic/kinetic studies, electromyography studies, and papers focused on what I call “biomechanical squatology.”

IA. Kinetics and kinematics

Throughout this review I will avoid the more inclusive term kinesiology, even though it is correct, because that term has been heinously expropriated from a legitimate academic discipline to include some outrageous New-Agey bullshit quackery and is now contaminated. Kinematics describes movement in rigorous geometric detail, without being particularly concerned about the causal forces involved. Kinetics (or analytical dynamics) brings in the forces that produce or influence motion. For example, a study that uses video capture and motion analysis software to compare the maximal horizontal bar displacements, bar velocities and hip angles in the clean techniques of Bulgarian and Martian weightlifters would be a kinematic, descriptive study. An investigation of peak power outputs correlated with maximal bar displacement in the clean would be both a kinematic (displacement) and a kinetic (power) study. Because barbell training involves moving weights through space, both types of study are clearly relevant, although in practice they often ask the wrong questions, come up with conflicting results, and generally muddy the waters.

For example, while in theory kinematic data (displacement, velocity, joint angles) coupled with force-plate data can be used to generate kinetic values (acceleration, power, torque), in practice there may be significant problems with current approaches, as reported by Lake et al. These authors note that it is increasingly fashionable in the biomechanical sports literature to derive power applied to the center of mass (CM) of the barbell-lifter system by multiplying the velocity of the barbell by the force applied to the CM (that force measurement being the ground reaction force, or GRF, obtained by using force plates). This is called the combined approach, because the calculation combines barbell kinematic parameters (velocity) and a CM kinetic parameter (force applied to the CM). To evaluate the combined approach, the investigators fitted resistance-trained athletes with markers and had them perform squats with high-velocity concentric phases at 60% 1RM while gathering GRF data with a force plate and kinematic data with video cameras for 3D motion analysis (3DMA). The resultant data allowed them to compute the power applied to the CM of barbell-lifter system in 3 different ways: (1) GRF x barbell velocity (from 3DMA); (2) GRF x CM velocity (from 3DMA); and (3) GRF x CM velocity (from the GRF data). The third calculation gives me pause, because it is entirely derived from GRF data, but both 3DMA and GRF gave similar values for bar velocity, so let that stand. The authors found that bar displacement and velocity (as determined by 3DMA) outstripped CM displacement and velocity. Most critically, they found that the combined approach (Bar velocity x GRF) “overestimated” the power applied to the CM by almost 20%. They conclude that it is not appropriate to use barbell
kinematics to compute system power. If these results are replicated by other investigators (and in other lifts, particularly explosive lifts), they may call a good deal of pre-existing research into question. I am compelled to point out that, since the available technology allows calculation of power applied to both the barbell and the CM of the system, a simple, comprehensive and illuminating approach might be to simply report both parameters, as for example in the paper by McBride that was discussed in last year’s review. In any event, this paper will be of concern to those readers who take an interest in the biomechanics of barbell exercises, and serves to illustrate that the field is still undergoing important methodological development, even for the analysis of fundamental mechanical parameters.

An excellent example of the practical potential of kinematic analysis from this year’s literature is the paper by Ikeda et al. These investigators performed a small but powerful kinematic study of the snatch in female weightlifters at the 2008 Asian championships held in Kanazawa, Japan. At this competition, female Japanese weightlifters did not distinguish themselves, and were dominated by Chinese, Thai and Vietnamese athletes on their home turf. That had to burn. In fact, the primary intergroup distinction in this paper is between “Japanese Lifters” (JLs) and “Better Lifters” (BLs). Ouch.

The investigators used high speed cameras and a digitizing system to perform their kinematic analyses, looking at bar path, horizontal and vertical acceleration, and knee and hip angular displacements and velocities. They found that the vertical acceleration imparted to the bar by JLs was essentially the same as that demonstrated by BLs, but that JLs displayed a consistently greater angle of horizontal acceleration, a consistently greater horizontal bar displacement (especially forward of the vertical reference arising from just anterior to the midfoot), and a strong trend toward lower angular velocity of the hip joint in both the second pull (hip extension) and catch (hip flexion). The authors note that the Japanese coaching method for the snatch is to direct the lifter to pull the barbell back into the hips and slam it off the pubis. Based on their results the authors suggest, in Nicespeak, that maybe this approach isn’t working so well.

This biomechanical analysis of the snatch, while purely descriptive, suggests that deviation of the bar path from the vertical and failure to maximize the hip moment during the second pull are detriments to the optimal performance of the Olympic lifts. Moreover, this paper is an excellent demonstration of how an organization (The Japan Weightlifting Association, in this case) can combine modern kinetic-kinematic analysis with dispassionate, pragmatic and critical self-assessment as a first step toward modifying coaching practice and improving performance. One wonders if such an approach could possibly be employed in a systematic and fruitful manner outside of Japan.

A similar kinematic analysis of 7 female weightlifters (gold medal winners from each weight class) during the 2010 World Weightlifting Championship was performed by Akkus. The findings were, again, purely descriptive (there is no hypothesis-testing here). Kinematic-kinetic results were unsurprising (duration of the first pull was longer than the second; power during second pull was higher than the first, etc). Bar paths were all over the place. The author states that the most “optimal” bar paths were to be found in the 53 kg and 69 kg class winners, and these were, in fact, the most vertical bar paths, although a quick look at the actual loads lifted shows that these paths did not sort with a higher relative load (kg bar wt/kg body wt), and there is no data on individual power outputs or other kinetic-kinematic variables.

Akkus also collaborated with Hadi et al on a kinematic analysis of the snatch with the Turkish weightlifting team (n=7) at differing loads. Their results – increased loading, decreased velocity, increased work, and maximized efficiency, etc – are not particularly novel, although it is worth pointing out their finding that maximal loading required a minimization of horizontal work during the second pull.
Another kinematic-kinetic study from 2012 makes a nice follow-up to the study from McBride that I reviewed last year. Both studies evaluated the effect of loading on kinetic and kinematic variables during the clean. This year, Comfort et al investigated the effect of various loads (as a percentage of 1RM power clean) on bar velocity, rate of force development (RFD), peak force (Fz), peak power (W) and impulse (Ns) in the mid-thigh clean pull. For those who may not be familiar with the exercise, this is a highly truncated version of the clean (view YouTube video here) which isolates the second pull, the phase of the clean that requires the highest power generation. As the name suggests, the bar is held at the mid-thigh, and the athlete explodes from there into the full “triple extension” of the second pull, without racking the bar on the shoulders. Sixteen young collegiate athletes with middling power clean 1RM's (averaging about 0.95kg/kg BW) were recruited to spend a little quality time on a force plate. After two sessions of 1RM testing in the power clean, they performed clean pulls at 40%, 60%, 80%, 100%, 120% and 140% of their 1RM clean. The investigators found, to the surprise of exactly nobody in the universe, that bar velocity was highest at the lowest weight, Fz was highest at the highest weight, and peak power was displayed somewhere in between (40% 1RM). Impulse, which is the integral of a force-time curve and relates those two variables to acceleration (for systems of constant mass), was highest at the higher loading.

The study is well-done, and a beautiful illustration of the force velocity relationship. The authors are at pains to point out that their results for peak power (40%) differ from a previous study (60%), but they allow that the previous study used stronger athletes, underscoring that multiple variables are likely to impact which weight produces peak power for any given trainee.

Last year, when I reviewed the McBride, I admitted to being at something of a loss as to the practical implications of their findings. After reading this paper, I'm still not sure how a confirmation of the force-velocity relationship should substantially change our practice. A number of studies like this have made it clear that peak power is generated by an athlete at around half of his 1RM (somewhere between 30% and 80%, depending on who you read). And it appears that as an athlete works from low to high weight while training an explosive lift, he is sequentially emphasizing different attributes: speed, power, force, impulse. And so, again, I suggest that the practical implication of this paper – if any – might be to adopt a training strategy in which the trainee regularly attempts to move his best single (I won’t call it a 1RM) in the explosive lifts forward. But she does most of his volume at lower weight. For example, our lifter's best single for the PC is 150, and his 3x5 max is 130. (In this article, “RxS,” where R and S are integers, always refers to “reps x sets.”) Today he will attempt to nudge his best single to 152.5, and his 3x5 max to 132.5. Since reading the McBride, I have had occasion to experiment with this approach, and I made some progress. That’s strictly anecdotal, and I have no idea whether I would have progressed faster with another approach. At the end of the day we still don’t know the best way to program explosive lifts for athletes of differing abilities in differing sports.

Other kinematic-kinetic studies are likely to be less influential. Bonnet et al, a French-Italian team of investigators, developed a mathematical approach to estimating squat mechanics on the cheap, using some questionable assumptions and a least-squares algorithm that gave them reasonably accurate values using only a single inertial measurement unit (a motion sensor attached to the sacral area) and a single force plate. This setup is apparently less expensive than the spectrophotogrammetric equipment and multiple force plates used in many laboratories. The paper will be of interest to biophysics geeks, developers of new laboratory systems, exercise science PhDs working in the indolent economies of southern Europe, and just about nobody else.
IB. Electromyography.

Electromyography, or EMG, is a mainstay of biomechanical exercise science. If you’re not familiar with EMG, the best way to think of it is that it’s an EKG of the skeletal muscle – in other words, a recording of the electrical activity of the tissue. Nerve, cardiac muscle and skeletal muscle are excitable tissues. They work by pumping ions (charged particles) across their membranes to create an electrochemical gradient – in other words, a voltage. When the time comes to do some work, muscle cells (skeletal or cardiac) open channels that allow the ions to flow back across the membrane, discharging the voltage and generating currents of sodium, potassium and calcium ions. The resultant action potential is worth an article or two in itself, but suffice to say that this electrical activity can be detected by electrodes jammed into the electrically active tissue, or, more mercifully, on the surface of the body overlying that tissue. Hence, the EKG for the cardiac patient, the EEG for epileptics, and the EMG for PE majors hoping to make a few extra bucks by volunteering for tiny studies in the Department of Exercise Science at Miskatonic University.

The EMG is invaluable as a tool for studying muscle activation in exercise science studies. The actual use of EMG in the laboratory, however, is still undergoing evolution and refinement. Signal variability can arise from a number of factors, including movement of muscle fibers outside the range of the electrode during movement, subject motivation and compliance during a particular observation, minor variations in electrode placement, and any inherent difference that might exist between the motor task used to “calibrate” the observation and the motor task actually being studied. For example, an EMG investigation of the squat might use a maximal voluntary contraction during an isometric motor task (i.e., pushing against a dynamometer as hard as you can with a constant knee angle) as the reference for observations of submaximal dynamic motor tasks (i.e., squattage). Balshaw and Hunter12, two promising young investigators from Stirling, Scotland, demonstrated that their well-described dynamic EMG normalization method was superior to the use of isometric standards. In other words, they showed that it’s better to compare apples to apples. If you’re using EMG in your own training or coaching practice, this paper might be helpful to you. The take-home message for us Earthlings, on the other hand, is that there’s a ton of EMG research going on, and it’s not even clear that everybody’s using the equipment properly. It will be interesting to see, in coming years, whether the Balshaw-Hunter approach, or something like it, becomes a research standard.

One of the most illuminating EMG-related papers for this year was not a report of an original observation, but a systematic review by Clark et al13, who evaluated the extant literature on muscle activation in the loaded free barbell squat. This paper underscores the limited information available (only 18 studies suitable for review were found by the authors’ search parameters) not to mention the significant variability in study methodology, rigor and insight that have been brought to bear on the topic. The studies reviewed by the authors all utilized EMG methods to investigate which muscles are activated, and to what degree, in any number of barbell squat variants. The particular muscles studied and the methods of normalizing the EMG data (if any) varied considerably between studies. It is particularly instructive to examine Table 1 of the manuscript, tabulating which muscles were evaluated in the various studies. Quads and hams have been investigated intensely. Anterior and posterior truncal muscles (the fashionable “core” muscles) have attracted more recent interest. But the hips and adductors have been largely ignored, underscoring the widespread and longstanding view that the squat is a “leg exercise.” Indeed, the implicit assumption underlying much of this paper, and the literature it analyzes, seems to be that the purpose of the squat is to overload the “primary movers” (i.e., leg muscles) rather than to recruit the maximal amount of contractile tissue over the largest range of motion.
All that being said, the authors are able to make some tentative conclusions based on the very limited data available. To wit:

1. The optimal execution of the exercise will keep the bar in a vertical path over the middle of the foot.
2. Increasing hip rotation and turning out the feet recruits activation of the adductors and the glutes.
3. The results of studies of squat depth are muddled, in part because they’re not asking the right questions, and in part because of the experimental difficulty of normalizing loads across different squat depths. (I can probably quarter-squat 600 lbs. Full squat? Not so much). However, the data reviewed here, in aggregate, would seem to favor the view that partial squats result in reduced muscle activation and a suboptimal training stimulus.
4. Although the use of weightlifting belts does not appear to affect muscle activation, it does appear to improve “velocity of movement and bar kinematics.”
5. The squat is a more effective activator of trunk stabilizers than other “core stability” exercises.
6. Squatting on unstable surfaces increases leg and trunk muscle activation, but impairs force and power production. The authors don’t come right out and call bosu ball squatting stupid. That would be unprofessional. But you can read between the lines.
8. The highest level of muscle activation during the eccentric phase occurs during the latter third of the descent.

This review is limited by its underlying assumptions, the paucity of data available for analysis, and the significant variability in methods and quality of the studies incorporated in the review. Nevertheless, it suggests that the available evidence is beginning to converge with a model of the squat that may sound strangely familiar to many of the people reading this article.

The final conclusion by Clark et al (#9 above), which I have paraphrased solely for the sake of brevity, was based on the work of Schwanbeck et al\textsuperscript{14}, although Saeterbaken\textsuperscript{15} has found similar results, and a study by Cotterman\textsuperscript{16} might also be used to call the usefulness of the Smith machine into question. That didn’t stop Arandjelovic\textsuperscript{17} from publishing an in-depth, tour-de-force mechanical analysis of common variants of the Smith. On the off chance that you care, two major variants of the original Smith design are the counterweighted Smith and the viscous resistance Smith. Obviously, the mechanical properties of these two systems will differ, and the author kindly provides us with the differential equations governing them, along with results indicating which machine would work best for which training goals. There is no comparison with free barbells. In fact, there is no actual physical data here at all – just results from computer simulations. Turns out that when Sims go to gyms, they should choose the viscous resistance Smith for low-intensity work and the counterweight Smith to get swole with medium intensity work. At high intensities, it just depends on the virtual lifters’ weaknesses. Up to the Matrix, as it were. This paper is actually kind of breathtaking in its focus, brilliance, chutzpah, and complete lack of actual physical data or practical applicability.

IC. Biomechanical squatology.

The discussion of the Clarke article allows us to segue, however inelegantly, into the all-important realm of biomechanical squatology, the science of how to do squats on a force plate with stickers all over you and then pretend you got something meaningful out of it.
While common sense, the universal experience of strength athletes and the available evidence all make it apparent that squats performed to parallel or below activate more muscle tissue than partial squats, it is still not clear to some that deep squats are necessarily superior. Drinkwater et al\textsuperscript{18} compared the kinetics of full to partial squats in a paper that is sure to be waved under the nose of any coach or athlete who advocates full squats to an incredulous public. The authors defined “full range of motion” squats (FROM) as “hips parallel to knees” and partial range of motion squats (PROM) as knees to 120 degrees. We are assured that the squats performed by the subjects were regulated and uniform, but are provided no documentation or exemplars of squat depth. The investigators found that lifters were able to generate a higher mean concentric force with PROM than FROM, which is a fancy way of saying you can partial squat more weight than you can full squat. They also found that you can generate more power if you partial squat, which, surprisingly, they found surprising. Although they don’t tell us where peak power was generated, I would guess it was at the top of the squat, where the “finishing velocity” is highest, and doesn’t vary much between FROM and PROM squats. Since the peak velocity is similar at the top of the squat, we might expect that the higher load generates the higher power. So what? Finally, they found that FROM – in other words, moving weight over a greater distance – generated higher mean concentric work. Shocking. They conclude by suggesting that FROM squats are more beneficial when the athlete seeks to maximize work output and time under tension, which is not exactly a major contribution to coaching wisdom. Conversely, they suggest that PROM squats will be beneficial to athletes who require high velocity or force production through a limited range of motion. Of course, this paper offers no proof of anything of the sort, since it did not evaluate the relative impact of FROM and PROM squats on actual performance. The entire enterprise seems to be predicated on the notion that the selection of a squat variant can be based on the kinetic features of that variant. The usefulness of this doubtful and undemonstrated concept is merely assumed, not addressed, by this paper. In fact, this concept will be dealt a shattering blow by Hartmann et al, as we’ll see shortly.

Bryanton et al\textsuperscript{19} conducted a study of the effect of barbell loading and depth on relative muscular effort (RME, calculated as the ratio of net joint moment to maximum voluntary torque matched for joint angle) for the squat. They report that the knee extensor RME is more affected by the squat depth than the load, while the opposite is true for the ankle plantar flexors. The paper could be cited as an endorsement of deep squats, but there’s no assessment of transfer to performance or practitioner-oriented outcome, and the results are not particularly surprising or exciting in any case.

One of the most important papers of the year is that by Hartmann et al\textsuperscript{20}, which transcends the usual shortcomings of the squatological literature. This German study is a serious challenge to the concept of joint angle-specific weight training and a major reinforcement of the concept that strength is a general adaptation. The authors used a longitudinal experimental design involving 10 weeks of periodized maximal strength training to determine whether deep squats (BSQ), front squats (FSQ) or quarter squats (BSQ\textsubscript{1/4}) would lead to (a) higher gains in angle-specific 1RM, (b) higher enhancements in vertical jump performance and (c) greater gains in mean rate of force development (MRFD) and maximal voluntary contraction (MVC) at the 120 deg knee angle (the angle of the quarter squat and countermovement jump). Thirty-six Brüder und 23 Fräuleinwunder were recruited and tested for their countermovement jump (CMJ) height. Based on this parameter, the subjects were parallelized into 1 of 3 groups, training in the FSQ, BSQ or BSQ\textsubscript{1/4} for the duration of a 10-week program that began with a hypertrophy phase and ended with a strength-power phase. Descriptions of the experimental procedures and exercise performance are excellent. After the training period, the subjects underwent testing for 1RM in all the squat variants, and for the CMJ, squat jump (SJ), MRFD and MVC. All groups demonstrated an increase in the strength of their specific squat variant; in other words, front
squatters increased their FSQ, quarter squatters increased their BSQ¼s, and full squatters increased their full squats. But then it gets interesting. Full squatters increased their strength in all the squat variants, whereas quarter-squatters increased only their quarter squat. In other words, increases in full squat strength transferred to increases in all the squat variants, while increases in quarter-squat strength did not transfer to any other squat variant. In fact, quarter-squatters showed a trend toward decline in their FSQ and BSQ strength. Moreover, while both full- and front-squatters increased their CMJ, quarter-squatters demonstrated no improvement in their CMJ – similar to controls who did no exercise whatsoever. And the quarter-squatters demonstrated greater declines in MRFD and MCV values than front- and full squatters. All of this in spite of the fact that the quarter squatters added more weight to their squat variant than full- or front squatters.

In other words, when it came to increasing countermovement jump height, squat jump height, or general squatting strength, the quarter-squat sucked mightily. The study is not ideal – it is, again, small and short. But it is otherwise well-designed and well-controlled, and as far as I’m concerned it should signal the beginning of the end for the idea that quarter-squats add anything to a general strength-training program for any sport. The discussion by the authors is quite long, and almost qualifies as a first-rate literature review on the squat, including a devastating evidence-based deconstruction of the idea that full squats should be avoided because they will damage the knee joint.

I conclude discussion of this very important paper by leaving you with this in-your-face language from the authors’ discussion:

“Based on functional aspects, only a deep squat can be used as an effective general strength training exercise. Solely, deep joint positions provide the required neural and morphological stimuli for the hip and knee extensors to positively influence the acceleration process.…

“Periodized maximal strength training quarter squats elicits significant transfer losses into the isometric maximal and explosive strength behavior of hip and knee extensors in the initial phase of the turning point and does not provide any significant increments of force application into the acceleration process of reactive and concentric speed-strength performances. This refutes the… concept of superior angle specific transfer effects.”

II. EXERCISE PHYSIOLOGY

This category includes studies of the effect of resistance training on molecular and cellular physiology, tissue structure and function, adaptation, hemodynamics, and the like. In the previous review, such studies were coupled with investigation of the specific benefits of resistance training in health and disease under the category of “Big Medicine.” The sheer volume of work to be considered this year mandates separate categories for basic physiology and health intervention.

IIA. Muscle physiology

This subcategory includes papers that investigate tissue-level skeletal muscle responses to resistance training stress.

IIA(1). Eccentric exercise produces a unique and uncomfortable response in muscle tissue that is subject to ongoing inquiry. The ultrastructural, neuromuscular and biochemical characteristics and responses to the “muscle damage” imposed by eccentric work on skeletal muscle remain to be elucidated
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in full detail. While both concentric and eccentric contractions lead to an immediate decrease in strength, eccentric work is thought by many to lead to a more profound acute loss of performance. Beck et al. sought to compare decrements in strength induced by concentric and eccentric work and tease out the relative contributions of neuromuscular and muscle-specific phenomena to each. They tested bro biceps (3 reps) on a dynamometer for isometric, concentric and eccentric force production (peak torque). Then they had them perform volume sets (10x6) under either concentric or eccentric conditions, and then tested for peak torque again. During pre and post-testing, they assayed neuromuscular function with EMG (which picks up action potentials in excitable tissues), and used mechanomyography (MMG, which picks up mechanical vibrations from muscle activity) to evaluate mechanical properties of the muscle. This technically limited approach might have led to some interesting results – if they had found that the eccentric and concentric conditions produced different effects. Unfortunately, they did not. While both the eccentric and concentric conditions produced changes, the experimental approach produced no difference in the magnitude of effect of concentric and eccentric contractions on peak torque, normalized EMG amplitude or frequency, or normalized MMG amplitude or frequency. In other words, their model did not produce the differences they were interested in studying. And yet, they felt free to speculate away about their results, going so far as to claim they had demonstrated a neural component to the strength decrease after both concentric (“fatigue only”) and eccentric (“fatigue and muscle damage”) exercise. This could certainly be true (and it makes sense), but I don’t think they showed anything of the sort, since they performed no specific assays of neural function (nor did they perform any assay of muscle damage). The paper is of some interest for its use of MMG and its review of the extant literature in the introduction and discussion. Its “conclusions” are likely to be cited by others. But the results are not illuminating and have no novel practical implications.

(Science tip: If you want to study why two phenomena are different, it’s really helpful to have an experimental model in which those two phenomena actually display a difference.)

Readers interested in the relationship between exercise-induced muscle damage and hypertrophy-strength acquisition would be much better served by reading the paper by Brad Schoenfeld, even though it is a literature review, not an original observation. It is a remarkably lucid and evenhanded examination of what is known – and, more importantly, not known – about this topic. Of particular interest are his examinations of the literature on inflammation, reactive oxygen species, satellite cell activity, the role of IGF-1, and the AMPK-PKB “switch” that determines catabolic-vs.-anabolic responses to exercise (more on this later). The paper is an excellent overview of where things stand in the field, and should be read by anybody with an interest in exercise myophysiology.

Farup et al. conducted a morphological comparison of muscle adaptation to strength vs. resistance training, focusing on changes in muscle fascicle angle (FA). FA describes the angle between the muscle fibers and the aponeurosis, the broad tendinous connection to the bone. A change in FA has significant mechanical implications. An increase in FA not only changes the angle at which the muscle exerts force on the bone, but also allows for an increase in muscle cross sectional area (CSA), which correlates directly with force production. This study subjected untrained individuals to either an endurance (ET) or strength training (RT) program for ten weeks. FA was assessed by ultrasonography, anatomical CSA by MRI, and VO₂ max and strength by standard testing. Biopsy of vastus lateralis was used for assessment of fiber type and fiber CSA by ATPase histochemistry and microscopy. RT, but not ET, increased FA. The other findings of this study were in line with previous reports, confirming that RT, but not ET, increases anatomical and fiber CSA, and strength. ET but not RT increased VO₂ max (nominally). This paper is the first definitive comparison of the FA adaptation in RT vs. ET.
The findings are not particularly earth-shattering, but they do serve to re-emphasize a key principle: adaptations to ET are primarily metabolic, while RT induces profound structural changes in muscle.

IIA(2). Fiber-type shifting is a critical area of investigation in exercise myophysiology. All skeletal muscles are composed of an assortment of muscle fiber (muscle cell) types, which differ in their biochemical and biophysical properties. “Slow twitch” fibers are aerobic cells, rich in mitochondria. They burn fat and they’ll march all day long. But they have low cross-sectional area (CSA) and they’re weak. “Fast twitch” fibers are, depending on the subtype, anaerobic or mixed aerobic-anaerobic. They use phosphagen and glycogen preferentially, have high CSA, and are stronger but far more subject to fatigue than slow twitch fibers. Table 1 summarizes the attributes of muscle fiber types in humans.

<table>
<thead>
<tr>
<th>THE OBLIGATORY CLIP-N’-SAVE MUSCLE FIBER TYPE TABLE</th>
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<td><strong>PROPERTIES:</strong></td>
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<td>MHC Gene</td>
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<td>Fiber Shifting</td>
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Table 1.
Fiber shifting occurs when the relative proportion of fiber types changes in response to some stimulus. How this occurs, to what degree, under what conditions, and in which directions, are perennial problems in exercise science. Those with an interest in this topic will want to take up two papers published in the last year: the excellent review by Wilson et al24 and the methodology paper by Staron, Herman and Schuenke25.

The functional prevalence of Type I fibers in endurance athletes and Type II fibers in explosive athletes raises a classic chicken-egg problem. Is fiber-type prevalence genetically determined and fixed, or is it subject to training stress and adaptation? The Wilson review addresses the current literature on the topic. It seems clear that Type IIx fibers can convert to Ila and vice-versa in response to training. The outstanding issue is whether training can drive fiber shifting from slow (Type I) to fast (Type II) fibers and vice-versa. Older work suggested that shifting, particularly from slow-to-fast twitch, simply did not occur to any practically relevant extent, but newer work suggests otherwise. In particular, a 2001 study by Paddon-Jones et al found evidence of a shift from Type I to Type IIx in response to high velocity eccentric resistance training26. The authors of the current review point out limitations of the current literature and make excellent suggestions for future research. Their practical recommendations are based on sketchy evidence – as the authors themselves have so ably demonstrated – and should be taken with a grain of salt, although they are rational and would be excellent candidates for interventional studies.

However, those studies, and all previous studies in this area, should probably be viewed with the results of Staron et al in mind. The most popular approaches to fiber typing of muscle tissues are myosin ATPase histochemistry (which preferentially stains fibers with high ATPase activity) and MHC immunohistochemistry (which uses antibodies directed against the fiber-specific MHC isoforms to localize these proteins). Staron’s group showed that either method used in isolation resulted in significant misclassification of Type II fiber subtypes, particularly during a period when fibers were in transition in response to resistance training. Such findings by the Staron group and other investigators27, if verified, would have critical experimental implications for studies addressing the potential of training to alter fiber composition.

Bottom line: there is some tenuous evidence to suggest that resistance training at high intensity might drive a small increase in Type II / Type I fiber type ratio (which would be Good). But we are well-advised to be skeptical, and it will take a lot of solid work to overturn the prevailing view. Stay tuned.

A technically impressive bit of myophysiology, conducted at the tissue level, Figure 1. Muscle fibers in cross section at high magnification, stained for mATPase activity. Darker fibers have more mATPase activity, suggesting Type II (fast twitch). Note also that stained fibers tend to have a larger cross-sectional area. Reproduced with the kind permission of Alan Pestronk MD, of Washington University, St. Louis MO.
ultrastructural and molecular levels in heart failure patients undergoing resistance training, was conducted by Toth et al; it is discussed in the section on Cardiovascular Diseases.

**IIB. Endocrine, metabolic, and cell signaling processes**

This subcategory includes papers investigating endocrinologic, biochemical and cell-signaling responses to strength training. These areas of investigation are incredibly important, and if you’re at all interested in the science behind increasing strength and muscle mass, you’re going to need to get familiar with some of the major characters.

**IIB(1). Overview of cell signaling.** If you meditate upon Figure 2, it will help you wrap your head around some of the papers we’ll be discussing in this and other sections of this review. The figure may look daunting to you, but they don’t call it a *cartoon* for nothing. It’s really not a big deal if you take it piece by piece. The major key to understanding the figure is that an arrowhead means the thing it’s pointing to is *promoted* or enhanced by the thing it’s pointing from. A blunt-ended line means that the thing it’s pointing to is *inhibited* or blocked by the thing it’s pointing from. All of the boxes with

![Figure 2](image-url). Cell signaling pathways involved in the adaptive response of muscle tissue to different exercise stimuli. GF = growth factor. GFR = growth factor receptor. Ca++ = calcium cation. MEKK/MAPK/ERK = elements of the MAP kinase signaling cascade. Figure prepared by the author.

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funny names written on them represent proteins, most of them signaling proteins. Key “switching”
elements are in red and blue. Proteins that regulate gene transcription are in gray. Let’s just say that
the steak is a ribeye.

The left side of the figure tells the story of Joe Weightlifter, who does short, intense workouts
under heavy loading. Through mechanisms that are poorly-described or just plain speculative, this
results in the activation of growth factor signaling, perhaps through an isoform of IGF-1, the mechano-
growth factor (MGF). Autocrine and paracrine release of IGF-1 by the muscle cell itself may play
a role, and other growth factors may also be involved. Activation of the growth factor receptor can
in turn activate the MAP kinase cascade, a signaling system not unlike the relay signal towers of
old, which culminates in a genetic response from the nucleus. Growth factor (GF) activation also
works through PI3K to turn on Akt (also known as PKB), a critical signaling molecule, which in turn
activates mTOR, a key decision point, as we’ll see. The activation of mTOR leads to up-regulation
of the protein synthesis machinery (S6, 4E-BP1, and other factors), resulting in increased protein
synthesis and hypertrophy. Note that some data shows that amino acid ingestion also activates mTOR
and thereby ramps up protein synthesis, either directly and/or through the GF signaling machinery.
GF signaling on this side of the figure culminates in the transcription and synthesis of both anabolic
and anti-catabolic gene products.

The right side of the figure tells the story of Marathon Mike, who’s clocking in serious hours
on the treadmill, after which he’ll have a garden salad (no dressing). This tragic ordeal leads to the
activation of the critical signaling molecule AMPK, which, unlike Akt, turns off mTOR, effectively
shutting down the mTOR-mediated anabolic signaling cascade. This is the so-called AMPK-Akt
switch, which is proposed as a central mechanism underlying Hickson’s famous (and much-ignored)
interference effect. Also pictured here is the accumulation of muscle fiber calcium and the consequent
activation of calcium calmodulin kinase (CaCMK), resulting in the expression of a library of genes
that inhibit hypertrophy, and genes that promote increased mitochondrial mass. The net result of the
right-sided story is the promotion of more catabolic, aerobic phenotype.

It is difficult to put into words just how ridiculously simplified and incomplete my pathetic
little figure is, although I’m sure some people will be all too happy to try. The initiation of protein
synthesis alone involves more than 140 proteins and ribonucleoproteins. My cartoon is not intended
to be comprehensive, or even right (some of this is speculative or based on inconclusive or conflicting
data), but rather to give the reader a road map so that the papers we’ll be discussing here will have some
sort of meaningful context.

IIB(2). Studies of MAPK, AMPK-Akt and mTOR signaling. The MAP kinase (MAPK) cascade
has emerged as an important signaling pathway for cell function in general and for skeletal muscle
hypertrophy in particular. This signal cascade is responsive to exercise stress and growth factor
stimulation. Activation of the growth factor receptor by growth factor binding and phosphorylation
triggers subsequent phosphorylation-activation events on MEKK, MAPK, and ERK, leading finally
to the activation of transcriptional regulators like Elk, Fos and Jun, which mediate gene expression.
Taylor et al sought to investigate whether differences in exercise intensity would differentially activate
this signaling cascade, using a standard bro-bike-biopsy & blood model. Bros did leg extensions at 60%
1RM (20x4) or 85% 1RM (10x4) followed by blood draws and biopsies. The results demonstrated a
strong trend toward increased serum IGF-1 after both forms of exercise, greatest for the high intensity
work. The authors dismiss this finding because it is not statistically significant, even though they provide
no documentation that their study was powered to detect a significant difference, and even though
they detected a significant increase in IGF-1 receptor activation. Moreover, even though biopsies were taken and used for MEKK-ERK-Elk measurements, no effort was made to assay intramuscular IGF-1 levels to account for autocrine/paracrine elaboration of this growth factor. Both forms of exercise resulted in profound but similar increases in MEKK, ERK, and Elk phosphorylation. The authors claim their findings demonstrate that this signaling system is activated by resistance training in an intensity-independent manner.

They demonstrate nothing of the sort. The failure to normalize intensity-volume products between the groups, the use of uncontrolled ELISA assays for phosphorylated proteins without assessment of total protein levels (an important control), the lack of a power analysis, potential problems with biopsy methodology, and the complete lack of any exploration of downstream effects make such conclusions untenable. This paper is an additional data point in favor of MAPK-ERK cascade signaling in response to resistance training, but its value should not be overstated.

The contribution of Galpin et al30 to exercise/MAPK literature is methodologically superior to the previous study, and is a novel evaluation of MAPK-ERK cascade activation in response to a multi-joint power exercise (the clean pull), as opposed to a more hypertrophy-oriented or single-joint stimulus. Trained weightlifters performed clean pulls at 85% 1RM (3x15, 4-minute rests), and biopsies were taken before, during and after the workout. Unlike the vast majority of strength science studies, this paper actually documents the exercise in a figure, and the authors monitored performance kinematics, in part to determine whether differences in power or work sorted with biochemical changes (they didn't). And unlike the Taylor paper, the authors correctly assayed and reported both phosphorylated protein and total protein levels for each species they interrogated. ERK, MAPK (p38), and JNK all underwent significant increases in phosphorylation, although these changes were less robust than is usually reported for hypertrophic stimuli. It would have been nice to see results from a “control” group doing hypertrophy/single-joint work, to determine whether the responses seen in this study were truly less robust. Nevertheless, the results demonstrate that multi-joint, explosive exercises are also able to stimulate the MAPK cascade, and that this response is maintained for the duration of the exercise session.

A potentially important paper by Hellyer et al31 promises to add a little complexity to the model presented in Figure 2 above. They subjected young adolescent rats (3 weeks old) to a program of progressive resistance training, which involved a climbing task. Weight on the tail was increased in a linear fashion over the course of this rat novice progression. Twenty-four hours after the final workout, rats were sacrificed (no progression to Intermediate status, alas). Muscle tissue (flexor hallicus longus) was removed and assayed for hypertrophy, fiber type, total myosin heavy chain (MHC) content, and levels of total and phosphorylated Akt, mTOR and rpS6 (the substrate of S6K, involved in regulation of protein synthesis at the ribosomal level). The biochemical methodology was excellent. A sedentary group of young adolescent rats was used a control. Trained rats developed muscle hypertrophy, as we would expect, with higher CSA than their lazy brethren. Trained rats had higher MHC content and higher proportions of MHC-IIx. But total and phosphorylated levels of the signaling proteins were not different between the groups. The authors conclude that in growing kids, chronic increases in the level and activation of signaling molecules are not necessary for muscle hypertrophy. They also conclude that since these animals displayed hypertrophy at “moderate” loads, maybe coaches shouldn’t have their kids lift so heavy. Replace “kids” with “rats” and all those conclusions might be supported by this data. Still, the findings with regard to cell signaling beg for a study in human adolescents.

On the other hand, decreased mTOR signaling in older muscle has been invoked as a potential mechanism for the blunted anabolic response to resistance training in the elderly. Farnfield et al32
examined the response of Akt, mTOR and downstream signaling molecules in both young and elderly subjects, with or without administration of whey protein isolate (WPI), and in both the untrained and trained state (i.e., after twelve weeks of resistance training). They found that both older and younger trainees exhibited a more marked response of mTOR and its downstream signaling molecules after WPI compared to placebo, but that these signaling responses were blunted in older muscle after twelve weeks of training. This paper suggests that diminished mTOR responsiveness in older muscle may play a role in the blunted anabolic response of elderly trainees to resistance exercise.

The paper has serious limitations. The data on Akt is unconvincing to me, both in the way it is presented and the authors’ failure to document both phosphorylation sites (Ser 473 and Thr 308). The data on mTOR shows that all groups, young and old, trained and untrained, increased mTOR phosphorylation, with a trend toward more phosphorylation after WPI. This does not reach statistical significance in the older trained group, but the trend is clearly there, the biological significance of the data is unclear, and in any event we have no idea if this small study was adequately powered to detect a significant difference, since the authors do not seem to have found it worthwhile to conduct a power analysis prior to embarking on their project. The same criticism holds for the analysis of eIF4G and p70S6K. The study design does not account for the possibility that acute changes in signaling seen in younger muscle may not be absent, but merely delayed, in older muscle. The major shortcoming of the study is a common one: the investigators are using low-dose exercise medicine. There’s not a structural exercise in the lot, and it appears the authors actually discontinued progressive overload of the exercises in the second half of the study, holding the intensity at 80% of 1RM for the last 6 weeks. This is a mechanistic study, not a clinical one, and it will be of interest to those wishing to understand the molecular mechanisms underlying age-related declines in anabolic responses to resistance exercise. For trainees and coaches, it is another data point underscoring the importance of protein ingestion in conjunction with training. See also the paper by Symons et al in the section on Nutrition.

The role of AMPK signaling in the regulation of skeletal muscle mass is an exciting area of investigation. Referring to Figure 2 above, you will recall that AMPK plays a critical role in the development of the catabolic, aerobic phenotype of Marathon Mike. Activation of AMPK by depletion of tissue energy (high levels of AMP allosterically activate this enzyme), calcium activated kinases (CaMKK), prolonged vigorous exercise, or by oxidative stress, leads to a fundamental shift in the cell’s use of resources. In muscle tissue, this means enhanced protein breakdown via autophagy and ubiquitination, decreased protein synthesis via suppression of translation initiation and elongation processes, mitochondrial biogenesis, increased lipolysis, decreased glycogen synthesis, and modulation of the glycolytic pathway, primarily via phosphofructokinase and hexokinase. The importance of AMPK, Yin to Akt’s Yang, couldn’t be more manifest to those interested in exercise physiology. The picture is a complicated one, because AMPK activation may have important positive implications as well, including positive cardiovascular and longevity effects. I highly recommend the in-depth and nuanced review of AMPK’s effect on muscle protein turnover published this year by Sanchez et al35.

The study published in Acta Physiologica by Ngo et al34 complicates the issue of cellular switching between endurance/oxidative and resistance/anaerobic muscle phenotypes…and not in a good way. The investigators recruited elderly men and had them do resistance training in the upper body and endurance training in the lower body. Muscle biopsies after 14 weeks showed that resistance-trained exercise enhanced glycolytic enzyme expression, endurance training enhanced lipid burning enzyme activity, and both regimens evoked increased capillarity and citric synthase expression. This is the kind of study that lends itself to misapplication, and is sure to be quoted by fringe fitness gurus with an axe to grind. It’s an awful paper: the endurance training wasn’t really endurance training, and...
In the Year in Strength Science 2012, the resistance training was a bunch of dumbbell thrashing done in the hypertrophy rep range. Training did not extend past the novice interval. Even worse, both training regimens were employed in the same people at the same time. In other words, these guys were subjected to circuit training and then used to describe biochemical differences between “resistance training” and “endurance” training. This is a very poorly-designed study that tells us nothing about the differential response to resistance and endurance exercises. Watch for it in blog posts, mainstream “wellness” stories and the pages of Men’s Health.

A study by Gregory et al adds to the growing body of literature indicating that resistance training increases systemic IGF-1 signaling (which in turn would activate the MAPK and AKT-mTOR pathways). This was a babe-bar-blood study. Women squatted. They gave blood. They bumped their IGF-1. It’s getting to be old news.

IIB(3). Inflammation and inflammatory signaling in response to exercise is a topic that generates a lot of lay interest, not to mention a certain amount of spurious coaching advice. The interactions between exercise stimulus, inflammation, recovery and adaptation are nevertheless important, and still being fleshed out.

Fernandez-Gonzalo et al characterized the effect of eccentric exercise on the TLR4 signaling pathway. This is a pro-inflammatory pathway, particularly well-described for the response to bacterial endotoxins. One of the most remarkable aspects of this paper is subject selection: the investigators found twenty male undergraduate sports science students who had not engaged in strength training for six months. This phenomenon is, in itself, worthy of some intense investigation. So they had these flaccid bros do an acute bout of eccentric squats (performed on a Smith, negative phase only) and then drew their blood. Blood mononuclear cells (immune cells; basically monocytes and lymphocytes) were separated from the blood and subjected to PCR for RNA (gene expression) and Western blot (protein expression) for components of the TLR4 pathway (CD14, MyD88, TLR4, TRAF6, TRIF, pIKK, pIkB, and p65, if you must know). The bros were then divided into two groups, a training group (TG) and a control group (CG). TG bros underwent a 6 week progressive eccentric exercise program at high volume designed for maximum DOMS-like torment. The CG bros went back to studying their sport science textbooks and not doing any strength training. (It just blows my mind.) After the six weeks they all came back for another round of eccentric training, and blood was drawn and tested again. The assay methods appear to be excellent, and the findings were significant and intriguing. Both groups demonstrated increased transcription of pro-inflammatory mRNAs after the first bout, as well as increased expression of the corresponding proteins and phosphoproteins, demonstrating that eccentric exercise had elicited a profound inflammatory response in mononuclear cells (muscle was not assayed, alas). However, after the training interval, TG demonstrated a marked decrease in the expression of inflammatory proteins. Curiously, however, their transcription (mRNA) response remained high. The CG response did not change. The authors conclude – correctly, in my opinion – that eccentric training induces an inflammatory response, mediated in part by TLR4 signaling, but that this response is markedly blunted by continuing training. This has uncomfortable implications for those who would argue that, without inflammation, there can be no strength, no hugeness, no life. I leave the reader to ponder these implications. A major bitch I have with this paper is the failure to present data for NF-κB, a critical end-point of this signaling system, responsible for affecting transcription of the genes involved. Either they assayed NF-κB and didn’t want to show the data, which would be chickenshit, or they didn’t look at it at all, which would be stupid. The discrepancy between transcription (mRNA) and translation (protein expression) in the TG is very intriguing indeed, and has the potential to create an entire new line of investigation. Stay tuned.
Other studies published this year also point to a mixed pro-inflammatory and anti-inflammatory response to eccentric resistance exercise. Much as this idea warms the cockles of my heart, these studies were of uniformly suboptimal quality.

Ye et al\(^\text{37}\) conducted a strange little study in which they took serum from men pre- and post-eccentric exercise. They then showed that a protein factor in post-exercise human serum suppressed lymphocyte proliferation \textit{in mice}. The factor itself was not identified or further characterized, and the significance of these findings, if any, is as yet unclear.

Henagan et al\(^\text{38}\) report that melanocortin receptor expression (which correlates with decreased systemic inflammation) is associated with reduced CRP (also associated with decreased inflammation) in response to resistance training. The data with regard to melanocortin is actually pretty underwhelming, and the clinical significance of the paper’s findings is, to say the least, dubious.

Gokbel et al\(^\text{39}\) conducted a \textit{bro-bike-blood} study looking at the elaboration of IL-6 and TNF-\(\alpha\) (inflammatory mediators known to be produced during exercise) and adiponectin, a signaling molecule released from fat tissue (an “adipokine”), which appears to have anti-inflammatory properties. Bros performed repeated bouts of the Wingate (a test of peak anaerobic power) on an ergometer bicycle, and gave up some blood. The authors report that they found a “marked increase” in IL-6 levels and a “marked decrease” in adiponectin levels. \textit{Baloney}. Adiponectin went from 37.9 ± 2.1 µg/ml before exercise to 36.7 ± 2.2 µg/ml. Setting aside that these levels are a bit higher than what you’d expect to find in the average bro\(^\text{40}\), that’s not what I’d call a “marked” change. I wouldn’t call it a change \textit{at all}, since the \textit{biological} significance of the difference in these values is almost certainly negligible. IL-6 went from 100.9 ± 13 to 133.8 ± 34.1 µg/ml….only slightly less underwhelming. TNF-\(\alpha\) did not change. No assay of actual inflammation was conducted. What this paper actually found was no biologically significant change in anti-inflammatory adiponectin or pro-inflammatory IL-6 or TNF-\(\alpha\) after repeated bouts of exercise. So why didn’t the authors say so? Maybe because they thought that negative results from a poorly-powered study wouldn’t be illuminating, interesting or useful. And they’d be right.

\section*{IIC. Cardiovascular physiology and hemodynamics}

Ozaki et al\(^\text{41}\) provide us with one of the most important papers of the year in this or any other category. This group compared the effect of standard “high intensity resistance training” (HIT) and \textit{blood flow restricted} low-intensity resistance training (LI-BFR) on the hemodynamics of previously sedentary Japanese bros. For those of you not familiar with LI-BFR, well, it’s been around for about twenty years, but is finally starting to emerge from the chrysalis of fringedom into full-winged faddishness. The idea is that, Dude, you perform your supersets at lower intensity, but while restricting the blood supply to the muscle with a special cuff (Kaatsu-Master system, made in Japan). So if you’ve seen bros during curls with purple arms while wearing overpriced straps…there you go. If you haven’t, you will. The data on this is pretty lean, but there is some reason to believe that it \textit{may} be of some use in populations that are physically incapable of performing resistance training at maximum loads, such as extremely frail individuals, astronauts and bodybuilders. Ozaki’s group used bench press only for their two groups, and found that HIT actually produced superior results in strength and CSA (although not by much). But hold on: The HIT group, according to the authors, demonstrated worrisome changes in their hemodynamics at the end of the training interval. HIT for 8 weeks decreased carotid compliance (from 0.14 to 0.11 mm2/mmHg, compared to a change of 0.15 to 0.17 for the LI-BFR group). HIT also increased systolic blood pressure…by about 6mmHg (normal is 120 mmHg). \textit{No abnormal change}
in blood pressure was demonstrated. The authors also found no significant biochemical or endocrine changes in either group.

I told you that this is one of the more important papers of the year. I didn't say it was a good paper. This study has the potential to generate considerable interest, discussion, and complete bullshit hysteria. Somebody out there is going to make money by claiming that Kaatsu training will make you just as strong as regular training, without giving you high blood pressure, heart disease or a stroke, and they're going to point to this study to give their scam a veneer of scientific respectability. But this study showed that Kaatsu didn't make you as strong as conventional training, and there is no demonstration here of any cardiovascular pathology whatsoever. The hemodynamic changes reported are of such small magnitude, and of such questionable physiological and health significance, as to be completely meaningless. I will keep my eye on the Kaatsu literature (and see also the paper by Godawa et al139, discussed under Coaching/Gear). But for now, I have to say that my gut-level bias is that exercising with quasi-ischemic muscles is really kind of nutty. (And where, exactly, am I supposed to strap myself to cut off the blood supply to my squat?)

There is no consensus in the literature regarding resistance training and vascular health42,138, but Japanese strength science researchers really seem to have a hard-on for this arterial compliance business. Miyachi43 conducted a meta-analysis of the effects of resistance training on arterial stiffness (stiffness being the inverse of compliance, as studied in the last paper). He identified only 8 RCTs meeting his inclusion criteria, encompassing a grand total of 193 subjects, of whom roughly half were actually control group subjects. Pooled data analysis revealed an aggregate effect of resistance training on arterial stiffness. The problem is that, again, we have no idea what that effect actually means. The two measures used were carotid beta index (normal about 8-11 AU) and arterial pulse wave velocity (normal about 5-15 m/s). Carotid beta index displayed a pooled absolute increase of 1.12 AU (95% confidence: 0.66-1.58 AU), while pulse wave velocity displayed a pooled absolute increase of 0.72 m/s (95% confidence: -0.03 -1.48 m/s). The clinical significance of these changes is unclear, as the author himself is forced to admit. The study’s problems are manifold: it is a meta-analysis, highly susceptible to the GIGO effect, with less than 200 total subjects. The data was not abstracted in a blinded fashion. The author chose two of his own studies to include. And, once again, the paper is completely devoid of any data on patient-oriented outcomes: death, disability, quality of life, need for medication, development of comorbidities such as hypertension or heart disease… nada. In short, this meta-analysis finds that resistance training may (or may not) increase arterial stiffening to a very minor degree, with unknown clinical effects. This paper is liable to be quoted extensively, primarily by people who either aren’t smart enough to realize that it doesn’t really tell us anything, or not ethical enough to care.

The literature review by DeVan and Seals44 on this same topic is also likely to get some press. This is a non-systematic review, without explicit methodology. Entitled “Vascular health in the aging athlete,” it covers a body of literature suggesting that aging endurance athletes have less arterial stiffness and arterial thickening than Masters lifters and therefore have better “vascular health.” Papers that found discordant results are glossed over, and the authors quote not a single study looking at comparative, long-term, patient-oriented cardiovascular outcomes (MI, stroke, aortic catastrophe, CHF, you name it) in aging lifters and endurance athletes. In other words, this paper, which is supposed to be about vascular health, did not assess a single controlled observation of long term health in aging athletes. This study tells us only one thing: the authors read some papers about vascular stiffening and formed an opinion. They stop short of making any recommendations against resistance training in aging populations, which is wise. Again, watch out for this paper to be quoted widely by folks with axes to grind and money to make.
On the flip side, Nascimento et al\textsuperscript{45} provide us with a systematic review of resistance training and hemostasis suggesting a cardioprotective response. They conclude that there is an indication that resistance training results in an acute fibrinolytic response, meaning that lifting weights makes your blood less likely to clot. This in turn might help prevent heart attack, stroke and other thrombotic complications. Tempted as one might be to crow about these findings, this review suffers from the same problems as the reviews by DeVan and Miyachi: a small number of small studies encompassing a small total number of subjects, and no assessment of long-term, patient-oriented outcomes. It is tempting to speculate in a way that brings all of this stuff together: perhaps the acute fibrinolytic response to high-intensity exercise is an adaptation to protect against the acute (high endothelial shear stress, turbulent flow) and chronic (arterial stiffening and thickening) consequences of training. But it is just that: speculation. This work is in its infancy, and researchers in these areas need to get their acts together and start asking the right questions.

Gurovich and Braith\textsuperscript{46} conducted a highly technical study of intravascular rheology, endothelial stress and flow patterns during both resistance and endurance training. They found that both endothelial shear stress and turbulent flow increase with exercise intensity with either resistance or endurance training, in a dose-dependent manner. The clinical implications were not explored, but the study is likely to be important for those investigating the mechanisms underlying hemodynamic/vascular adaptations to exercise (including the arterial stiffening crowd).

A very important but flawed paper comes to us from Niewiadomski et al\textsuperscript{47}, at the Warsaw University of Technology in Poland. This group investigated the effect of the Valsalva maneuver on the hemodynamic response to resistance exercise, using noninvasive measurements and some liberal biophysical assumptions. Twelve Polish bros (“broskis”) performed concentric and eccentric leg press exercises while their intraoral cavitory pressure (“mouth pressure”) was measured, along with their blood pressure and heart rate. As expected, Valsalva markedly increased systolic and diastolic blood pressures at rest and during exercise, with a higher target mouth pressure (a surrogate marker for a stronger Valsalva) in conjunction with a heavier load producing the largest blood pressure spikes. The authors then derived figures for arterial transmural pressures – that is, the pressure acting across the arterial wall during systole (when pressure is the highest). They found that, in general, Valsalva decreased transmural pressures across the arterial wall, although the responses were highly variable. No broskis died or stroked out during the experiments.

This paper provides a data point in favor of the protective effect of Valsalva in strength training. In this model, the exercise + Valsalva-induced increase in intraluminal arterial pressure is balanced (more or less) by Valsalva-induced increases in cavitary (extraluminal) pressure, in both the thorax and in the cerebrospinal fluid (Hamilton and others have shown that an increase in intrathoracic pressured is transmitted to the CSF). While I think this is right, the paper does suffer from methodological shortcomings, some of them unavoidable. The use of mouth pressure as a surrogate for intrathoracic pressure is not ideal. The alternative, however, is to measure intrathoracic pressure directly, which would mean chest tubes, which is extremely difficult to get past the Human Subjects Committee, even in Poland. The figures for transmural pressure were calculated from the arterial pressure and the mouth pressure, so these critical values were not obtained by direct measurement, but were derivatives of a surrogate value. Finally, the study was small. Nevertheless, it is a not-half-bad stab at a very important topic, and hopefully it will stimulate further conversation and investigation in this area. The review of the literature in the discussion is, in itself, worth the read.

Other studies of interest in this area were limited by their experimental design, methodology and/or lack of novel findings. For example, De Deus et al\textsuperscript{48} used a rat model to investigate cardiac and...
metabolic adaptations of resistance training. Their findings suggest beneficial changes in pancreatic function, insulin sensitivity and autonomic control of heart rate. Boukani et al\textsuperscript{49} reported that concurrent endurance and resistance training elicited cardiac remodeling and ventricular function that tended more toward the endurance-training phenotype.

IID. Aging and atrophy

We begin with the important editorial by Manini and Clark\textsuperscript{50} published in the special issue on sarcopenia and muscle function in the \textit{Journal of Gerontology} in January 2012. The authors contend that dynapenia and sarcopenia are two different things, that they sort differently with mortality, and that they should be assessed separately. They provide a working algorithm for the definition of dynapenia based, by their own admission, on controversial assumptions. They make much of recent reports claiming to show that only strength, not muscle mass, is associated with mortality. They cite three studies in this regard, two from the same group (Visser and Newman et al\textsuperscript{51,52}, with the third being a secondary analysis (data-mining) of the InChianti study\textsuperscript{53}. This latter paper looked at calf muscles in > 65 yo subjects, and in fact found an association with mortality that disappeared after a poorly-described statistical “adjustment for potential confounders.” Setting aside the limitations of all these studies (and they are significant), several observations are pertinent. Mortality is not the only outcome deserving of our attention, or even the most important. Quality of life, hospitalization rates, need for medication, cognitive function, adverse events, etc, are all more meaningful, and necessary for a complete picture of the health of an aging population. The relationship of sarcopenia to such factors is not experimentally addressed in any of the literature discussed here.

Finally, the observation that greater muscle mass does not necessarily translate into greater strength is certainly correct…but also trivial. The authors note correctly that many factors influence the ability to produce force, and muscle mass and cross-sectional area are only part of the equation. All true, but hardly novel. Moreover, the authors do not address any corrective interventions or their relative impact on sarcopenia or dynapenia (“resistance training” does not appear in the manuscript, and “strength training” appears only once, in a tangential context). Strength and adequate muscle mass (which has benefits beyond raw strength) are both important and interdependent. One may certainly have dynapenia without sarcopenia, but it is more common to have both, and quite uncommon to have the latter without the former. Manini and Clark make a reasonable case that it’s important for us to distinguish between sarcopenia and dynapenia, and this has some bearing on epidemiological research. But let’s not forget that, through the miracle of Iron Age technology, we can treat them both at the same time. As an attempt to move the field forward, and as an excellent review of the literature and epidemiology, I do think this paper is worth a quick read.

In a previously-published article, I invoked apoptosis, a type of programmed cell death, as one of the mechanisms underlying aging in general and sarcopenia in particular. I also tried to make clear that the situation was vastly more complicated than all that. Ongoing study implicates a number of regulated processes underlying sarcopenia, including ubiquitin-mediated proteolysis, autophagy, and the down-regulation of muscle protein synthesis, as well as classical apoptosis. Like apoptosis, these processes are all more-or-less subject to modulation by exercise and trophic cell signaling. Readers interested in the biology of skeletal muscle aging and sarcopenia will want to take a look at the review published in the \textit{Journal of Aging Research} by Peterson et al\textsuperscript{54}. These authors focus particular attention on mitochondrial mechanisms underlying declines in skeletal muscle function and mass, including...
oxidative stress, derangement of mitochondrial DNA, mRNA and protein production, changes in mitochondrial dynamics (fusion and fission), mitochondrial biogenesis, and mitochondria-triggered (“intrinsic”) apoptosis. The section on strategies to attenuate mitochondrial aging is a mixed bag, with discussions of exercise (which has been clearly shown to be of benefit in humans) and caloric restriction (which hasn't).

The relative contributions of apoptosis and other regulated cytolytic processes to sarcopenia are nevertheless ill-defined. The matter is complicated by the suggestion that apoptosis may actually be increased by exercise, to aid in the removal of damaged or senescent cells. This is a profoundly interesting hypothesis, and might suggest a model in which a differential and highly targeted, highly regulated form of apoptosis would be at work in resistance-trained individuals, removing some cells while other cells were actually growing. The relevant study by Sharafi and Rahimi55 completely misses the point and overstates its conclusions. This was another bro-bar-blood study, in which Iranian bros did a variety of resistance exercises and had blood drawn before and after. The authors state that apoptosis markers (p53, caspase-9, caspase-3) were increased immediately after exercise, along with serum lactate and IGF-1. None of the increases were impressive, and their clinical relevance is completely obscure, especially since the authors looked only at circulating levels of these markers. The place to look for apoptosis markers is in the tissue of interest. Would it have been so hard to do vastus lateralis needle biopsies and assayed for myocyte apoptosis?

Ratel et al56 took old guys, trained them in a mixed aerobic/resistance regiment for 16 weeks, and then let them detrain for 8 weeks. What they found was contrary to most coaching experience and published literature: detrained geezers held onto their VO2max fairly well, but completely lost all their precious leg press gains. I won’t belabor the methodology, because it’s not worth the candle. The authors conclude that it’s important to prevent detraining. No kidding.

One of the clear benefits of structural resistance training in older adults is the osteogenic stimulus it provides, resulting in an improvement in bone density and resistance to injury and advanced osteopenia. I was excited to see the title of the paper by Ebben et al57: The Optimal Back Squat Load for Potential Osteogenesis. Excitement gave way to fury when I actually read the article. The authors note that higher ground reaction forces (GRF) probably promote greater osteogenesis, so they measured GRF at various loads of the squat, including 120% of 1RM performed at partial range of motion (in other words, quarter-squats or half-squats). The authors note that it is “difficult” (not impossible58, mind you, just difficult) “to assess the osteogenic potential of exercise in vivo.” So they didn’t. They just showed that if you lift more weight you generated a higher GFR. Voila! For optimal osteogenesis, we therefore should probably do quarter-squats at 120% of our 1RM. You got your exercise prescription, right there.

Just to be clear: there is absolutely no assay of osteogenic adaptation in this paper. That would have been difficult. This paper is complete and utter crap, the very nadir of force-plate hack science, and it’s sure to be quoted by orthopods, PTs and fitness gurus. This paper wasn’t just bad. It actually pissed me off.

Li et al59 investigated AMPK and mTOR signaling (see the relevant discussion under Cell Signaling) in older men and the effect of “long term” resistance exercise on these signaling systems. Their findings are at odds with other published reports, and their data and conclusions are severely limited by their methodology. Muscle biopsies used in their analyses were not collected immediately after exercise and/or nutrient intake, but rather after an overnight fast and four days of detraining. Moreover, while they compared the expression and activities of these enzymes in younger and older men at baseline, the effect of resistance training was studied (or at least reported) only in older men.
The authors concede that resistance training does not have chronic effects on the mTOR pathway, but that its contribution to hypertrophy from resistance training is due to its repetitive activation.

III. BIG MEDICINE

In this section, we will examine papers published in 2012 addressing the impact of resistance training on health and various disease states. Before launching into the subcategories of cardiovascular, pulmonary, endocrine-metabolic and other conditions, however, it is propitious to discuss the important review article by Jan Sundell60. This brief, non-systematic review of the literature makes a very strong argument for resistance training in elderly and frail populations, similar to those I have advanced myself, but in a rather more compact format, and without the liberal use of expletives. The specifics of the exercise prescriptions offered here are arguable, but the general case for resistance training is spot-on. If there is one article from this year’s review you should obtain, this is it. Make a stack of copies to distribute to your friends, family, clients and physicians. This paper should be read widely.

IIIA. Cardiovascular disease

Cardiovascular disease, a spectrum of disorders which includes hypertension, coronary artery disease, peripheral vascular disease, and congestive heart failure, is the major killer of Americans, and there is every reason to believe that lifestyle is a major contributor to this epidemic. This year’s literature reflected the growing interest in the role of resistance training in the management and prevention of cardiovascular disease, with papers on hypertension, heart failure, and risk factors for coronary artery disease.

IIIA(1). Hypertension (“high blood pressure”) is a major public health problem, with untoward implications for every major organ system. Uncontrolled hypertension blasts away at capillary beds year after year, damaging the delicate tissues of the retina, kidney and brain, leading to blindness, renal failure, cerebral atrophy, stroke or even brain hemorrhage. It puts a huge and unrelenting workload on the heart, leading first to maladaptive ventricular hypertrophy and then dilatation and congestive heart failure. It promotes endothelial damage and the development of peripheral vascular disease, which can culminate in amputation, heart attack and stroke. Medical treatment is effective and can ablate much of this fuckery, but it is clear from abundant research that exercise is – or should be – a cornerstone of therapy for established hypertension, and first-line, possibly curative therapy for pre-hypertensive patients. This most emphatically includes resistance exercise.61,62,63

Moraes et al64 investigated the effect of a “chronic conventional” strength training regime on mild hypertension in 15 middle-aged men. The study is remarkable for incorporating a medication washout – most of the subjects were on antihypertensive medications, and these were withdrawn over the course of several weeks prior to the onset of training, resulting in significant elevations of their blood pressure. Also remarkable was the investigators’ decision to observe changes in blood pressure during detraining. “Chronic conventional” strength training, according to the authors, means a 12-week program of crunches, leg presses, leg curls, chest presses, lat pulldowns, biceps curls and triceps extensions. Subjects were instructed to breathe in on the eccentric phase and breathe out on the concentric; the Deadly Valsalva was forbidden. Blood pressure returned to pre-washout values: in other words, resistance training reduced blood pressure to the same near-normal values that the
subjects had demonstrated prior to medication withdrawal. Moreover, this benefit persisted after 4 weeks of detraining. No changes in vasoactive factors were observed, so the study provides us no insight into the underlying mechanisms for the effect. The study is small, its design and its methods are imperfect, it uses “low-dose” resistance training, and it tells us nothing about long-term patient-oriented outcomes. It’s not time to throw out your lisinopril just yet. But the potential importance of this line of investigation is obvious.

IIIA(2). Congestive heart failure (CHF) is one of the many unfortunate end-points of uncontrolled hypertension. It’s a pervasive disease in developed countries, and it’s a bastard. It comes in a wide variety of noxious flavors, and is caused by anything that leads to the inability of the cardiac pump to meet demand: ischemia, uncontrolled hypertension, infection, endocrine-metabolic or toxic insults, valve disease, you name it. Its clinical manifestations vary, but the most common avatar of this pestilence is a progressive decline in exercise tolerance, increasing shortness of breath, fluid retention and swelling, congestion of the lungs and liver, painful swelling of the extremities, decreased resistance to injury and illness, and finally death, often by a mercifully sudden lethal arrhythmia. In other words, you get weaker for years as you drown slowly in your own fluids until one day you just drop dead. This horror is treated with antihypertensives, nitrates, antiarrhythmics, diuretics and other poisons, which can prolong and improve life somewhat but cannot cure the disease (nothing short of a heart transplant will correct established heart failure). I was brought up to believe that the exercise intolerance of heart failure patients was fixed, and that subjecting these people to vigorous exercise lacked both benefit and safety.

Like much of what I was taught in med school, this isn’t true, and we see accumulating evidence that patients with chronic heart failure can train safely and derive benefits. Case-in-point is the systematic literature review on this subject published by Chen et al56 in the International Journal of Cardiology in July 2012, which found that combined endurance-resistance training improved exercise capacity in patients with congestive heart failure. Their findings are in accord with everything else we’ve seen on this topic, but are unfortunately limited by the small number of randomized controlled trials suitable for pooled analysis. The authors call for larger, more definitive studies of exercise in CHF. Everybody always calls for better studies, but nobody ever does them.

Sao Paulo in Brazil continues to be a hotbed of strength-science research, of varying quality. Servantes et al⁶⁶ produced an important study of the effect of aerobic and mixed aerobic-strength regimes on physiologic parameters and quality of life in patients with both moderately severe heart failure (New York Class II-III; left ventricular ejection fraction < 40%) and sleep apnea. Unfortunately, heart failure and sleep apnea are often comorbid with each other, and sleep apnea significantly exacerbates the pathophysiology of heart failure. Fifty subjects were randomized into three groups: aerobic training (walking, basically), aerobic-strength training (walking and free weights), and no training. The real hook is that the two training regimes were home-based, with frequent contact by the investigators to provide moral support and program compliance. Both training groups showed improvements in aerobic fitness, strength, sleep parameters, and quality of life (as measured by the Minnesota Questionnaire). Most parameters showed better improvement when strength training was part of the mix. The non-treatment group continued to dwindle, showing actual deterioration in these same parameters. The study is limited by its rigorous exclusion criteria, as the authors are at pains to point out. There were a number of clinical events: one death in the aerobic training group, one heart attack in the aerobic-strength group, and one death and two strokes in the sedentary group. The authors insist they were non-training-related, but they give us no details. The take-home points for
III. Coronary artery disease is a subset of peripheral vascular disease (PVD), which your grandma used to call “hardening of the arteries.” This is a complex pathology which can precipitate myocardial infarction (heart attack), limb ischemia (dead leg) or stroke (stroke). The risk factors for CAD and PVD are diverse, and are thought by most physicians and researchers to include hypercholesterolemia and hyperlipidemia, truncal obesity, decreased insulin sensitivity and diabetes, genetic factors, etc. Ho et al conducted a small study of resistance training on a number of cardiovascular risk factors, particularly cholesterol and serum lipids. The effects they observed were statistically significant, but of dubious clinical significance. The study used pathetically low-dose exercise prescriptions and made no observations of long-term, patient-oriented outcomes. This paper falls on the side of resistance training, but it suffers from the same flaws as many papers that do not. It may have the beneficial effect of spurring some doctors into writing a more appropriate exercise prescription, and more patients into complying with such a prescription. But as science it is mediocre, and it doesn’t really tell us anything important.

IIIB. Endocrine and metabolic pathologies

This subcategory encompass a vast array of disease states, including acquired and genetic abnormalities, with varying degrees of responsiveness to medical interventions, including exercise interventions.

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future reviews, strength-related studies of thyroid, adrenal, pituitary, pancreatic, and some hepatic and renal diseases will fall into this category. Most of the literature relevant to resistance training, however, is focused on the so-called metabolic syndrome and the insulin-insensitivity states that culminate in Type 2 diabetes.

Diabetes is a leading cause of death and health care expenditure in the United States, due in very large part to the growing prevalence of obesity and inactivity. The sedentary, super-sized lifestyle endemic to industrialized nations leads to a fatbody phenotype with insulin resistance and low glucose tolerance that ultimately culminates in the down-regulation of insulin receptors and other glucoregulatory proteins and the development of Type 2 diabetes. Exercise has long been known to improve glucose tolerance and insulin sensitivity, and a growing body of data shows that resistance exercise performs at least as well if not better in this regard, with the added benefits of increasing strength and reducing muscle and bone atrophy.

The beneficial effect of resistance training on glucose tolerance is underscored by the paper from Reed et al, demonstrating that normoglycemic women demonstrated these effects with even moderate intensity exercise after only two bouts. The authors note that higher volume work produced the most marked results, but that their results show that even frail and elderly individuals without the capacity to produce high volume-intensity products can produce beneficial glucoregulatory effects from resistance training, and quickly. This paper, however, undertakes no evaluation of long-term, patient-oriented benefits, and its results should not be overstated. The study by Hansen et al shows similar benefits in overweight individuals with prediabetic derangements in glucose tolerance and serum insulin levels. Again, the study is small and short, and the effect sizes were nothing to write home about. (The investigators were actually more focused on comparing the relative benefits of two different resistance exercise regimes, but the study is too small to make any such discriminations.)

Moving further along the spectrum of disease, Moreira et al produced similar findings in type-2 diabetics, with small effect sizes and no long-term outcomes. Finally, a poorly-powered short-term study of elderly Icelanders by Geirsdottir et al found no beneficial impact on fasting glucose or hemoglobin A1c levels (a marker of prolonged hyperglycemia).

I didn’t delve too deeply here into the methodology of these papers, because I don’t have to. If you have brain waves, you’ve noticed the pattern by now yourself. All of these studies, whether they claimed to show a benefit or not, were small and short, and focused exclusively on whether training could manifest a beneficial impact on laboratory values. Those values were and are pertinent and important, but unless you can show me that resistance training prevents progression of disease, improves quality of life, decreases the need for hyperglycemic medications and insulin, decreases hospitalizations, minimizes health care costs, and reduces the incidence of diabetes-related events and morbidity, I’m not impressed. It’s as if taking a dozen fat diabetics, putting them on the machines and showing some small improvement (or lack thereof) in their glucose tolerance is a sure ticket to publication in *JSCR* or *Joe’s Journal of Aging*, with the attendant completion of your summer research rotation or padding of your CV.

*Enough already.* There’s already abundant literature, including a large Cochrane review, showing that resistance training has a physiologic effect on metabolic syndrome and diabetes. It’s time to get busy with a big, randomized, placebo-controlled trial focused on long-term, patient-oriented outcomes that matter.
IIIC. Pulmonary Disease

This subcategory encompasses a broad spectrum of disorders. Obstructive pulmonary diseases are the most prevalent, including asthma and the chronic obstructive pulmonary disorders (COPDs) loosely differentiated as chronic bronchitis and emphysema. The obstructive pulmonary disorders are characterized by obstruction to outflow. In the case of asthma, this is primarily due to bronchospasm and edema of the small airways, while in COPD it is due to structural changes that cause small airways to collapse on expiration, often with superimposed bronchospasm. Except in the advanced stages, patients suffer not so much from an inability to oxygenate as an inability to ventilate, leading to CO₂ retention and respiratory acidosis. Pulmonary-ventilatory disorders have, historically, received more attention in the literature on aerobic exercise interventions. This might make sense on the basis of crude physiologic reasoning: when you’re working in an anaerobic energy system (as in high-intensity resistance training), ventilation is less likely to be the physiologic bottleneck limiting your ability to perform the exercise task. As usual, though, things are rather more complicated than all that, and resistance training in the setting of pulmonary disease is receiving increased attention.

The single paper in this subcategory for this year, the study by Vonbank et al., is not just a case in point, but also happens to be one of the better-done papers of the year. This is a randomized clinical trial with a larger number of subjects than most studies. The authors even go so far as to document that their study is adequately powered. Outcome measures included not only measures of strength, VO₂ max, pulmonary function, lactate clearance and the like, but also assessment of quality of life – an honest-to-God patient-oriented outcome. Patients with COPD were randomized to endurance training (ET), strength training (ST), or combination (C-EST), although no attempt was made to control for the increased work volume in the C-EST group (the paper isn’t perfect). Training was conducted for twelve weeks. All three groups showed increased exercise capacity, VO₂ max, and strength. In contrast to some previous work, the authors are able to conclude based on their data that progressive strength training, without associated endurance training, will increase not only muscle strength but also exercise capacity and quality of life. There are some minor problems here, but as an investigation of the potential health benefits of resistance training, this study stands out for its methods and scope.

IIID. Orthopedics and Rheumatology.

This subcategory will probably be split in later reviews, but I found relatively little literature in either field worth discussing, including a single original observation. Orthopedics is the surgical discipline concerned with structural disorders of, and trauma to, the musculoskeletal system, while rheumatology is an ill-named subspecialty of internal medicine, which one may loosely describe as being concerned with diseases of connective tissue. This means rheumatologists are involved in the investigation and care of a variety of particularly cruel collagen-vascular, autoimmune, and atraumatic joint diseases, including a number of devastating arthritides, such as rheumatoid arthritis and psoriatic arthritis.

IIID(1). Arthroplasty is the replacement of a diseased native joint with a prosthetic (“artificial joint”). Practitioners who are interested in coaching trainees post-arthroplasty, or trainees who have arthroplasies themselves, may be interested in the informative and comprehensive review by Marie Westby on rehabilitation for patients with total joint replacements. It’s a long read, and it doesn’t cover much data on resistance training…because there isn’t much data on resistance training. But the body of evidence is growing, and it clearly suggests that progressive resistance training has an important
role in promoting bone growth, securing the prosthesis, restoring function, and combating the atrophy and weakness associated with total joint replacement procedures, especially those involving the hip and knee. Post-arthroplasty patients are a rapidly growing population and their need for strength training is acute. Coaches need to be educated about and comfortable with this population. I hope to see more literature on this topic in the future.

IIID(2). Osteoarthritis (OA), or “wear and tear” arthritis, is the most common form of degenerative joint disease, and is almost universal in aging populations. Resistance training has a well-established role in the rehabilitation and management of this condition. The study published in Arthritis Care and Research by Sayers et al\textsuperscript{83} compared the benefits of slow-speed leg press (SSLP) and high-speed leg press (HSLP) in older subjects with OA. Both training programs resulted in improved pain and function, to similar degrees. Only high-speed training increased “power,” which is just about the most unsurprising observation anybody made in the literature all year. The paper is a data point in favor of resistance training for OA, but there’s nothing novel here.

IIID(3). Rheumatoid arthritis (RA) is an altogether different creature from OA. It is a vicious, crippling disease in which the body’s own immune system attacks various connective tissues, including the synovial membranes surrounding the joints. It is a polyarticular (multi-joint) arthropathy that is particularly cruel to the small joints of the hand, wrist, feet and cervical spine. Synovial joints are ultimately disfigured and destroyed by this disease, and disability can obviously be severe, promoted not only by the disease itself but also by the understandable inclination of RA patients to become more inactive over time, leading to sarcopenia, weakness, and accelerated loss of function.

The role of resistance training in RA remains controversial, with only a small number of randomized controlled trials (RCTs) addressing patient-oriented outcomes for this intervention. Baillet et al\textsuperscript{84} published a very well-conducted systematic review and meta-analysis in the January 2012 issue of Rheumatology, using the well-regarded Cochrane approach to pooling and analyzing data. Their inclusion criteria admitted ten RCTs, encompassing 547 patients, roughly half of whom were controls. Outcomes analyzed were tender and swollen joint counts, pain, disability, strength, and functional capacity (all patient-oriented), as well as erythrocyte sedimentation rate (not so much). Pooled data demonstrated positive differences in all parameters evaluated, although the small number of studies and the heterogeneity and quality of the data rendered the clinical relevance of these findings uncertain, except for clear benefits to functional capacity and disability. Subgroup analysis (the weakest kind of data) showed a trend toward more benefit from higher-intensity training. Importantly, and as one might expect, there was no evidence of an impact of resistance training, positive or negative, on structural damage. There were no adverse consequences for patient safety. So, again: \textit{Enough already.} We’ve done the small studies, and the pooled results are promising. It’s time for the Big Kahuna: a big, adequately-powered, well-controlled, multicenter, longitudinal RCT with high-dose resistance training for RA. But don’t hold your breath. In the meantime, patients with RA and the doctors who care for them are running out of excuses.

IIIE. Neurologic, Neuromuscular and Myopathic Disorders.

This subcategory is reserved for papers that investigate resistance training in populations with stroke and cerebrovascular disease, neurodegenerative disorders such as Parkinson’s and multiple sclerosis, neuromuscular diseases such a myasthenia gravis, and assorted myopathies and muscular dystrophies.
IIIE(1). Neurodegenerative Disease. These disorders are characterized by the loss or damage of neuronal tissues in a progressive manner, resulting in loss of motor and/or cognitive function. Such disorders include Parkinson’s disease, amyotrophic lateral sclerosis (ALS), Huntington’s chorea, Alzheimer’s disease, multiple sclerosis and spongiform encephalopathies.

Although it is not a peer-reviewed publication, I am compelled to bring your attention to this document, an outline for a scholarly presentation given in January 2013. It is an excellent resource on the extant literature on exercise interventions aimed at promoting neuroplasticity and combating neurodegenerative disease.

Parkinson’s is characterized by the loss of dopaminergic neurons in the brainstem, critical for the regulation of movement. Symptoms include spasticity, rigidity, bradykinesia (slow movement), and progressive motor and cognitive degeneration. Gait impairment, including loss of the ability to even initiate walking, is a common morbidity in this disease. Hass et al85 contributed to a growing body of literature on the role of strength training in Parkinson’s disease with a study focused on the clinically relevant endpoint of gait initiation. The investigators demonstrated that a progressive resistance training program improved measures of gait initiation, as well as stride length and velocity. A non-contact control group showed no improvements. The findings are in line with earlier reports showing that resistance training is safe and beneficial for patients with mild-to-moderate Parkinsonism.

Multiple sclerosis (MS) is a disease in which neurons of the central nervous system (brain and spinal cord) undergo patchy demyelination, a loss of the fatty “insulation” that surrounds the axon. The irregular distribution, temporally and spatially, of demyelinated plaques can lead to a wide range of symptoms and illness severity. There is a considerable body of research on exercise in MS. Kjohelde et al86 conducted a systematic review and found evidence of a beneficial effect of progressive resistance training. They also say they found weak evidence of an effect on underlying mechanisms, but I’m skeptical. This analysis excluded all but 16 small studies. These studies were nevertheless of low-to-middling quality and considerable methodological heterogeneity.

A small (15 patients) original observation by Huisanga et al87 found evidence for an improvement in postural control in MS patients subjected to a progressive program incorporating machines, dumbbells, Swiss balls and balance boards.

Neither of these studies does anything to substantively challenge or advance current thinking about the role of resistance exercise in multiple sclerosis. My bias is to suspect that some cases MS will respond positively to tailored progressive resistance training, but it will take a large, longitudinal RCT to tell us more than we already know. I wouldn’t expect large effect sizes or arrest of the disease process.

IIIE(2). Stroke. There now exists a body of small studies showing that resistance training can improve function after ischemic stroke. Hill et al88 conducted a study of resistance training in stroke survivors that, once again, boils down to same-old-same-old. Take a very small cohort of stroke survivors. Train them. Watch them get stronger, wow! Show that some metrics of gait, function and quality of life improved and others did not. Spin your results positively and call for larger studies. This study is yet another suggesting that resistance training has value after stroke. Hooray. It’s too easy to point to data like this and claim that it supports barbell medicine. But it doesn’t. It’s a small, non-randomized, poorly controlled, improperly powered bit of well-meaning slop, using short-term, low-dose resistance training. Exciting topic. Cruddy data.

IIIE(3). Myopathy. Studies of resistance training in progressive myopathies such as muscular dystrophy are still pretty scarce, in part because of the concern over the potential of training to promote damage
that the atrophic muscle cannot repair. These concerns may be well-founded, but a case report from this year's literature reminds us that, well, You Never Know. Garcia-Benitez et al describe the case of a young man with Mc Ardle disease who underwent resistance training without ill effect, in defiance of traditional medical wisdom. Mc Ardle's is a rare autosomal recessive condition in which both copies of the gene that encodes muscle glycogen phosphorylase are mutated, resulting in an inability to burn muscle glycogen. As you might imagine, this has profound implications for work in the anaerobic energy regime. Patients with Mc Ardle's are known for exercise intolerance and a tendency to go into rhabdomyolysis at the drop of a hat. As I have pointed out elsewhere, rhabdo is no joke. It reflects an untoward acuity of muscle damage, and the consequences for kidney function can be severe. This kid, however, undertook a progressive resistance training program and did just fine, with an actual decline in his post-exercise CPK, and no evidence of rhabdo or myoglobinuria (muscle pigment in the urine).

Now, this is a case report. It's one kid, and a kid with a rare disease (the next case I see will be my first). I include this paper not because we're going to have Mc Ardle patients lining up to be coached, but to illustrate that conventional medical thinking about contraindications to exercise is usually just that: conventional, conservative, and not evidence-based. Prudence is a virtue, and special populations are well-advised to work closely with their physicians. But it seems that just about every time we look at what happens when people with medical issues start picking up barbells, we observe that it somehow fails to kill them. Food for thought.

IIIF. Psychosocial Health and Disease.

As in all the other subcategories we have surveyed, a growing body of data suggests that resistance training confers benefits to psychosocial health and is at least tolerated by those with psychological and neuropsychiatric disorders.

A very large study of Spanish children and adolescents by Padilla-Moledo suggesting an association between muscular strength, psychological health and somatic health risk factors. The study points to a correlation, but cannot, as designed, demonstrate causation.

A relatively large (n= 122) study by Hauer et al showed that a modified resistance training regime was tolerated and beneficial in frail, demented seniors. It did not incorporate barbells or structural exercises (i.e., it was a low-dose study), and was not designed to investigate an impact on cognitive function or quality of life.

One of the most important papers published in this subcategory in 2012 was the review by Chang et al. In this Taiwanese paper, the authors review the available randomized controlled trials on the effect of resistance training on cognitive function in older adults, elucidate the limitations of the research done so far, and discuss potential mechanisms for cognitive benefit from weight training. They find, not surprisingly, that the evidence is mixed and of variable quality. However, on balance there is evidence for a positive effect of strength training on cognitive function in aging. Moreover, there is data to suggest that strength training may have a more profound effect on geriatric cognition than aerobic exercise, and that benefit from resistance training on cognitive parameters may be obtained in a dose-response manner. The authors note, correctly, that our enthusiasm for these findings should be leavened with an appreciation of the varying methods, duration, exercise prescriptions, and outcome measures of the studies, not to mention a few trials that have failed to demonstrate benefit. Discussion of mechanisms focuses heavily on the exercise-induced elaboration of growth factors, particularly IGF-1.

Chang's piece is a literature review, not a meta-analysis. Indeed, I believe the few RCTs available are far too diverse in their methods and outcome measures to permit a useful meta-analysis. It is a short
and very readable little paper, and the authors are dispassionate and balanced in their assessment of the literature. The tables provide a useful overview of the better studies on this topic, and a brief perusal of them will reveal what you have probably already guessed: these studies tend to be short and small, and they incorporate exercises virtually guaranteed to elicit suboptimal endocrine and neurotrophic responses. This literature encompasses a virtually complete absence of structural exercise. Barbells are, as usual, underrepresented. Leg presses, calf raises, seated rows and single-joint exercises abound. This paper summarizes the available experimental evidence on the topic in a way that highlights both its promises and deficiencies. If the authors fall down on the job, it is their failure to note these deficiencies in light of their invocation of IGF-1 as a prime candidate mechanism for cognitive benefit after resistance exercise and their laudable acknowledgement of a dose-response effect. The largest systemic responses are elicited by multi-joint structural exercises that recruit the largest volume of contractile tissue over the largest range of motion. Why do the authors fail to note that the lack of such exercises in the literature represents a failure to study this form of exercise medicine in its optimal dosing range?

IIIG. Obstetrics and Gynecology

The systematic review by Nascimento et al93 began with an explicit PubMed and ISI search, yielding nearly 2400 articles for triage. Their inclusion criteria (randomized controlled trials in healthy women that assessed maternal or fetal outcomes) yielded only 19 papers suitable for analysis. The literature indicates that the prevalence of exercise during pregnancy is low, particularly in the United States, Ireland and Brazil. Physical exercise prescriptions, while more enlightened than in previous decades, continue to be vague and not particularly evidence-based. Most recommendations emphasize aerobic and pelvic floor conditioning, although resistance training is increasingly recognized as having a role. “Light strength training” (whatever that is) and meticulous avoidance of the dreaded Valsava maneuver (based on very poor evidence) are the order of the day. The literature seems to indicate that exercise during pregnancy, including strength training, is safe for the mother and fetus and improves maternal outcomes. Control of gestational weight gain, gestational diabetes, low back pain, and urinary incontinence have all been observed, although the effect sizes appear to be small. There is no observed association between exercise during pregnancy and low birth weight, preterm birth weight, or neonatal well-being.

This is a relatively important paper, and deserves to be in your files. Unfortunately, it underscores that the literature on this topic remains woefully limited. The quality of most of the studies is poor, specific studies focused entirely on rationally-programmed strength training are virtually nonexistent, and some of the most important maternal and fetal outcomes (perineal laceration, pre-eclampsia, HELP syndrome, fetal distress and anoxic encephalopathy) have received very little study.

LeCheminant et al94 conducted an 18-week study of postpartum women, comparing a machine-based RT program with flexibility training. The RT group showed greater strength gains and decreases in sedentary time. So now we know women can work out while they’re pregnant and after they’re pregnant. All that remains is a study of the snatch during parturition.

IV. PROGRAMMING

This category brings in papers that address issues of programming, periodization, exercise selection and set design. There aren’t as many papers in this section as I’d like, because quite frankly there’s
not much quality research (and the papers presented here are of middling quality, as we'll see). Most programming studies are, like those in the Big Medicine categories, too short and too small to generate dispositive data, and use outcome metrics that do not correlate well with desired real-world effects.

IVA. Periodization, intensity and volume

This subcategory examines papers that address the manipulation of intensity, volume, exercise selection and other variables over time to achieve training goals. I emphasize “over time” because programming is conducted along the temporal dimension, which means that the standard six-to-twelve-week sports science study is very poorly suited to the elucidation of optimum programming variables. This is why, in general, you’re probably better off looking to an experienced coach for programming advice than to an exercise science journal.

The study by McDonald et al. is a case in point. These investigators compared standard resistance training, plyometric training and a combination regimen in 30 recreationally active bros. The conceptual basis for this study is that two biomechanically similar exercises, one at high intensity/low power and the other at low intensity/high power, form a complex pair. An example might be a set of heavy back squats followed after a very brief rest by a set of box jumps (a resistance-plyometric complex, as studied here), or a set of front squats followed by a set of full-squat cleans. The high intensity exercise induces a state of postactivation potentiation, which “primes” the athlete for the lower intensity, high power exercise. Postactivation potentiation is an intriguing concept and the focus of much study, and this attempt to investigate ways to exploit the phenomenon is laudable. The resistance-plyometric pairings used by the authors were back squat/lateral jump, RDL/depth jump, and standing calf-raise/box jump. I leave it to the reader to reflect upon the rationale for these pairings, which were used only for the combination training (CT) group; the resistance training (RT) group did only squats, RDLs and calf raises, while the plyometric training (PT) group just jumped around. The study was very short (6 weeks), there was no attempt to correct for the higher training volume of the CT group, and there is no reason to assume that the study is adequately powered. To their credit, the authors are explicit in pointing out that, while they found statistically significant differences for a number of outcomes, no practically significant differences in strength or anthropometry were observed. So far, the literature does not show us convincing evidence of the superiority of complex training, but the idea of training complex pairs deserves a definitive investigation.

So here’s a modest proposal: take two large cohorts of weightlifters (with n determined by an actual power analysis). Train one cohort with standard programming, and train the other using complexes: front squat-clean and back squat-snatch performed in combined sets. Train them six months or, better yet, for a year, taking care to keep their work volume and other relevant variables closely matched. See which group shows the most improvement in their lifts (a very straightforward and appropriate metric for such a population). This study would get you an actual answer. Science may not be easy, but it can be very simple. I’m not holding my breath.

Another investigation with relevance to complex programming, the paper by Voelzke et al, is discussed under Coaching/Sport-Specific/Volleyball.

Simao et al. attempted to compare linear and nonlinear periodized resistance training in a small, 12-week study. Those familiar with the model of linear progression outlined by Rippetoe may not recognize the linear periodization model presented here, which is a different beast, more akin to the classic Matveyev model, in which each training mesocycle is directed at a particular goal (hypertrophy, strength, power, etc). The linear periodization model in this study consisted of 4 weeks of “local
muscular endurance” (12x2 @ 15RM), followed by 4 weeks of hypertrophy training (8x3 @ 10RM), followed by 4 weeks of strength training (3x4 @ 5RM). The nonlinear model included exactly the same progression, but limited to 2 weeks each (in other words, six weeks of linear periodization), followed by 6 weeks of workout-to-workout variation (“daily undulating periodization”) between these modalities. No structural exercises were included; the program consisted entirely of upper-body exercises (lat pulldowns, bench presses, triceps extensions, and curls). The authors claim their results showed that nonlinear periodized resistance training led to greater gains in 1RM and muscle thickness over a 12-week training period.

That’s not how I see it. On my reading, their data shows slightly better gains in bench press strength and trivial differences in triceps muscle thickness (measured by ultrasound). No other practical differences were realized. The paper is bound to be quoted as support for doing somebody’s strange new program, or for not doing a linear progression during the novice phase. The results give us another beautiful illustration of the novice effect, but tell us virtually nothing about the best way to manipulate training variables over time. It’s just another addition to the incoherent literature comparing linear and nonlinear programming modes. If you took all of the papers on this topic and graphed the results, you’d get a shotgun scattergram.

Robbins et al\textsuperscript{101} conducted an investigation of the impact of volume on squat strength, very similar to a paper by the same group that I discussed in last year’s review. Previously trained, relatively strong bros (1RM squat strength > 130% of bodyweight) were randomized to 1-set, 4-set or 8-set groups. The authors observed strength improvements in all three groups, with the greatest squat strength increases in the 8-set group, and no advantage of 1-set over 4-set training after the first few weeks. The differences are not trivial, but there’s a big problem here: \textit{all three groups trained at 80\% 1RM}. So we’re not just talking about a difference in volume; we’re talking about a difference in the \textit{volume-intensity product}. And the number of reps performed per set varied across the groups as well. What would we have seen if the 1-set group had performed their routines at 95\% 1RM, the 4-set group at 85\% and the 8-set group at 75\%, with the number of reps in each set fixed? I think that might have been rather more illuminating.

Overall, my objections to this paper (published in \textit{JSCR}) are similar to those of the study on 8-set volume this same group published last year (in the \textit{European Journal of Applied Physiology}). And that’s entirely reasonable, because, when I look closely, I find that both papers studied the same cohort (43 initial subjects, 11 dropouts), using slightly different outcome measures. In other words, the authors double-dipped. Nevertheless, this study underscores the prime importance of volume as a target variable for programming manipulation, although it is hardly dispositive in telling us exactly how we should manipulate it. To their credit, the authors acknowledge the limitations of their findings and are quite careful not to overstate their conclusions.

The paper by Godawa et al, which addresses the role of compressive gear in the training of powerlifters, includes a detailed discussion of a 10-week powerlifting training progression that resulted in increased performance in both experimental groups. This paper is analyzed in the Coaching/Gear section.

If you want to read about the science of strength programming, the literature from 2012 offers nothing better than the state-of-the-art review by Kraemer and Szivak\textsuperscript{102}, \textit{Strength Training for the Warfighter}, published in a supplement of \textit{JSCR} in July. Kraemer is a world-class exercise physiologist who has made seminal contributions to the literature. Although this brief review is directed at educating military commanders and others responsible for the physical preparedness of American warriors, it should be read by everyone with an interest in strength and fitness. The lucid presentations
of the size principle and programming variables such as sets, reps, exercise selection, volume and intensity are required reading. The authors recommend that military trainers use a “flexible nonlinear programming” approach, which I wish had been presented with a bit more clarity and detail. Such a program is probably more elaborate than most civilian practitioners would need anyway, but might be of interest to high-intermediate, advanced and elite athletes, or those who are trying to balance strength training with intense sport-specific training or complex occupational demands.

In their concluding remarks, the authors throw down the gauntlet:

“In the modern era of the “anaerobic battlefield,” **one does not run into battle; rather, strength, power and functional capabilities play vital roles in the warrior’s success**….A fast 10 mile run or completing a marathon is still viewed as a benchmark of military fitness as opposed to a 40-inch vertical jump or a squat at 2.5 times body mass. **The ultimate paradox is that these performances are not compatible within the same training program** and so one must choose what the modern warfighter will look like physically. The influence of the lay press and various commercial entities have also further exacerbated fundamentals of workout design and training.” *(Emphasis added.)*

If this paper isn't being passed around at the Pentagon, battalion HQs, mess halls, barracks and military gyms all over the world, it should be.

**IVB. Set Design and Interset Recovery.**

Balsamo et al\(^{103}\) found that exercise order affects training volume and rating of perceived exertion for bros doing reps to volitional failure in a superset alternating ham curls and quad presses. Turns out that **these twelve bros in these particular sessions** could generate more volume, with lower perceived exertion, if they blasted their hams before their quads, rather than the other way around. The differences were not small; they were quite real, being both statistically and practically significant, if you consider doing supersets of ham curls and leg extensions “practical.” I must confess, my eyes sort of glazed over about halfway through this one, although I dutifully slogged it out to the end. As a scientist, a coach, a physician and a human being, I really had trouble caring. This data will not change your practice. Or at least it shouldn’t.

Marshall et al\(^{104}\) investigated a “rest-pause” method of set design, based on the idea that increasing the total number of failure sets will increase both the anabolic stimulus and the total volume of exercise performed. This is because, although the total number of completed reps would be the same as with a standard protocol, the number of failed reps will increase, and fatigue will be maximized. The authors speculate that such a design will collapse prescribed volume into as short a time as possible, while increasing the stimulus for growth and strength development. Think of it as an alternative to the standard Texas Method volume day. The idea of increasing the number of failed reps goes against my grain, but one has to **admit** the idea is intriguing. Bros performed one of three protocols, all of which incorporated a total of 20 squat reps at 80% 1RM. Protocol A was 4x5 with 3 min interset rest; Protocol B was 5x4 with 20 sec interset rest, and Protocol RP was a rest-pause protocol, with an initial set to failure with subsequent sets performed to failure with a 20 sec interset rest, again to a total of 20 reps. Unfortunately, bros performed all three protocols on different days, a very important potential confounder. Squats were performed to 90 degrees of knee flexion (partial squats), and EMG was used to evaluate muscle activation in the quads, erectors, glutes, and hams (no measurement of adductor activation). To assess the magnitude of fatigue induced by the various protocols, the investigators measured maximal isometric squat strength and rate of force development before and after exercise, which doesn’t thrill me.
Long story short: the authors report increased motor unit recruitment for all muscle groups using the rest-pause method. Decrement in strength and rate of force development were the same across the different set designs. Again, I have some problems with the study design, and I have a problem with programming built around the idea that failing reps is somehow salutary. The results can only be applied to a model of the squat that is clearly inferior. And there is no measurement of relevant outcomes: strength acquisition, hypertrophy, performance, etc. We’ll keep an eye open for papers that investigate the clinical benefit of this approach.

Two papers from McBride’s group\(^\text{105,106}\) investigated the effect of interrepetition rest on the power clean (PC).

The first paper took ten bros (mean age 23.6 years, mean mass 80.4 kg, mean PC 1RM/mass 1.39) and had them do power clean sets (3x6) on a force plate, with rest intervals of either 0, 20 or 40 seconds. You’ll never guess what they found: bros generated more power, more force, and more velocity to the bar with longer rest periods.

The second paper took ten bros (mean age 23.6 years, mean mass 80.4 kg, mean PC 1RM/mass 1.39) and had them do power clean sets (3x6), with rest intervals of either 0, 20 or 40 seconds. You’ll never guess what they found: longer rest periods resulted in lower levels of perceived exertion.

If these two papers sound just a bit similar, it’s because they’re both from the same experiment. Look at the bro data in the parentheses above. Same bros, same methods, same experiment…different papers. One paper was published in *JSCR*, the other in the *European Journal of Applied Physiology*. Why publish one paper when you can publish two? Having experienced the publish-or-perish pressures of academia myself, I wouldn’t bitch about it, if I hadn’t read the same paper twice just to find out something that every coach in the galaxy already knows. I expect better from McBride.

Mohamad et al\(^\text{107}\) addressed the issue of how to maximize the training impact of interset rest during resistance training. This is an area that has drawn a fair amount of attention in the literature during the last two decades, with seminal papers by Hannie et al\(^\text{108}\), Ahtiainen and Kraemer et al\(^\text{109}\) and Corder et al\(^\text{110}\), among others. The authors investigated the effect of active (aerobic) rest on kinematic and kinetic variables and lactate clearance. This is yet another underpowered study: 12 trained young men were tested to 1RM squat (to parallel), then assigned to one of 4 exercise regimens: 35% 1RM sets with active or passive recovery and 70% 1RM sets with active or passive recovery. Rest intervals were 90s. Sets and reps were allocated so as to equalize resistance training volume between the groups. The study had a randomized within-subject crossover design, so the bros came back after about 72 hours to participate in another group until the study was completed. The authors found no significant differences in lactate clearance or any kinetic or kinematic variable. The authors seemed astonished to find that even though there was no difference in lactate clearance with the two rest regimens, and even though volume was equalized for the 70% and 35% 1RM regimes, the guys who lifted heavier weights accumulated more lactate. Who’da thunk it?

The use of active rest between sets is an area of ongoing investigation. It appears to be a particularly crucial topic for those interested in hypertrophy\(^\text{109}\). The real issues requiring study aren’t addressed here: do we always want to clear lactate between sets? What is the effect of doing so on strength, power and mass accumulation? How does active rest impact long-term gains? And so on. This study is so small, its endpoints so narrow, and its findings so limited that it cannot be used to support any change in training practice.

Scott et al\(^\text{111}\) attempted to evaluate the effect of time-under tension and different repetition cadences on total energy expenditure and its components. Ten bros were tested for 1RM on the bench press (performed on a Smith for simpler collection of physical work data). On consecutive visits to the
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In the lab, the bros were then randomly assigned to one of three cadences for bench sets (5x3) at 70% of 1RM. The cadences were: 1.5 sec down-1.5 up (15 sec per set); 4 sec down-1 up (25 sec per set); 1 sec down-4 up (25 sec per set). Work and rest periods were held constant. Using a metabolic cart and blood lactate measurements, the investigators then measured or derived values for anaerobic energy expenditure, aerobic energy expenditure, and excess post exercise oxygen consumption (EPOC). Total energy expenditure was calculated as the sum of aerobic energy expenditure, anaerobic energy expenditure, and EPOC (converted to joules). The authors found that the energy components (aerobic, anaerobic and EPOC) differed between protocols. The 1.5/1.5 cadence had the lowest aerobic, anaerobic and EPOC energy expenditures. Anaerobic energy expenditure was highest for the 1/4 cadence, aerobic energy expenditure was highest for the 4/1 cadence, and EPOC was the same for the 4/1 and 1/4 cadences. The differential impact of 4/1 and 1/4 cadence on total energy expenditure was minimal.

Total energy expenditure correlated best with total time under tension, which revelation is not exactly a mind-ripper.

Now, the practical strength programming implications of this work, if any, are completely obscure to me, because to my mind programming is about manipulating variables to get stronger, not to consume more energy. All sets were performed at 70% 1RM…in other words, warmup weight. This was done to eliminate the confounding influence of fatigue, and was also crucial because it was this deloading that actually allowed the manipulation of time under tension. Think about it: when you’re going for a new 5RM, the last thing on your mind is whether You Got Rhythm. The manipulation of cadence studied here may put some readers in mind of dynamic effort sets used in the Westside method and performed at similar percentages of 1RM. But it is my understanding that DE sets are performed as rapidly as possible – minimizing the time under tension. The effect of such explosive performance was not investigated here. Furthermore, we have no idea how the different energy components would be affected by sets performed at these cadences using higher percentages of 1RM, or at 5RM, because the authors didn’t look. They also didn’t look at any practitioner-oriented, long-term, functional outcome. That’s all understandable, because this is really just a basic exercise physiology study, and a very focused and limited one at that. This paper almost went into the exercise physiology category, but it ended up here because of its potential to be misquoted and misused for programming at the level of set-rest design. “Whoa, Dude, you should do your sets at 1/4 cadence, because it, like, maxes out your lactate. Get ya ripped, brah. It’s published.” Of course, this paper tells us nothing of the sort. I include it in this year’s review so that you’ll be aware of its limitations, and also because exercise physiology geeks will find it a worthwhile read for its methodology, which is not half-bad. The Procedures section may be profitably perused alongside Chapter 10 of the essential text by Brooks.112

IVC. Time of day.

Two studies addressed the time of day programming variable for training. Chtourou et al.113 hypothesized that training at the same time of day would mitigate diurnal fluctuations in performance as measured by the Wingate, the vertical jump, 1RM leg extension, leg curl and squat after 8 weeks. Thirty Tunisian bros were recruited and completed a self-assessment instrument that supposedly determined whether they were “morning bros” or not. Most of them, being human, were not. It was all rather beside the point, since they were then randomized to a morning group (MTG), an evening group (ETG), or a control group (CG). Before the experiment, they all underwent testing for the outcome parameters in both the evening and morning as baseline measures. All groups showed poorer baseline morning performance compared to evening. MTG and ETG trained for eight weeks in either the morning or
the evening, respectively, and post-testing of the same parameters took place. Control bros slept in and did not train. Their performance, regardless of time of day, did not change. ETG improved their evening performance, but their morning performance still sucked. MTG showed marked improvement in their morning performance, but also improved their evening performance. The authors conclude that adaptation to strength training at a particular time of day will improve performance at that time of day, and that morning training will improve both morning and evening performance. Combined with studies of strength training with caffeine (lots of caffeine), this may or may not change your practice. This paper might be of special interest to competitors preparing for a meet, provided they know well enough ahead of time when they’ll be taking the platform.

V. Training, Coaching, and PERFORMANCE

VA. Metrics, biomarkers and benchmarks.

This is an important subcategory, bringing in studies that evaluate metrics of performance and progress. In this author’s opinion, the field is desperately in need of well-characterized, clearly-defined, readily obtainable metrics that can be used by practitioners, coaches and investigators to determine whether or not a particular intervention has produced a desirable and relevant outcome. It’s all very well to show that two different interventions produce two different outcomes in leg press 1RM in football players or nursing home residents. But what’s really needed is a metric or set of metrics that can be used as reliable markers for better performance on the football field or the Old Folks’ Home.

Unfortunately, as I indicated in the first review, this is not an easy undertaking. A great deal of very careful work will be required to come up with putative situation-specific metrics, validate them, and refine them. Suitable metrics for health-related strength interventions (mortality, hospitalization rates, need for medications, adverse events, quality of life) are in place, but they are seldom used by strength investigators, in part because studies have to be larger and longer to adequately power the evaluation of such metrics. Athletic performance metrics are in even worse shape. It may be especially difficult to develop meaningful universal metrics for “broad domain” sports like football and rugby. And I don’t see that happening yet, although the literature on volleyball is beginning to light the way.

Gearheart et al did a study to determine whether it was safe to use the OMNI resistance exercise scale to determine the 1RM in older men and women. They took forty-nine men and women of about 64 years of age and acquainted them with the OMNI, a rating-of-perceived-exertion (RPE) metric. The OMNI utilizes cute little hieroglyphics of people working out at different intensities. In this sense, it’s a type of visual analog scale (VAS). After indoctrination in the OMNI, they put the geezers on the usual shiny machines for a couple of weeks to work out at an RPE of 4 (out of 10). Then, ostensibly using this scale to guide them, they took these geezers to their actual 1RM. The authors are delighted to report that they did so without injuring or killing anybody. What they are unable to report is how, exactly, their method was substantially different from just adding weight until the geezers couldn’t lift it anymore, which, on my reading, is pretty much exactly what they did. Nor could they report why, exactly, we really need to measure the actual 1RM for a machine exercise in any 64 year-old novice trainee. But there you go: I include this paper because we should all know there are such things
as the OMNI scale (and the more venerable and well-characterized Borg scales) out there. What we are
to do with them is a mystery to me.

Similarly, Faigenbaum et al\textsuperscript{116} investigated the reliability of the one-rep max power clean test
in adolescent athletes who already had at least one year of experience with the exercise. Kids of about
14-16 years old underwent power clean 1RM testing. After a warmup, progressively smaller weights
were added to the bar until the kid couldn't clean it anymore. Remarkably enough, this procedure
was conducted without the use of any sort of scale or score or hieroglyphics. The kids were brought
back a few days later and the procedure was repeated. The 1RMs were more-or-less the same. None
of the kids were killed. The statistical analysis employed by the authors indicated that the typical
type of error to be expected with 1RM power clean between test measures was just under 3 kg, and that a
difference of >8kg would be required to indicate an actual change in power performance between test
measurements. The conclusions of this study are limited to an adolescent population that is already
very familiar with the exercise, and tells us nothing about what we are actually supposed to do with a
1RM power clean measurement.

Row et al\textsuperscript{117} were a bit more rational in their application of an RPE scale: they wanted geezers
to be able to determine the optimal load for an explosive exercise (leg press, alas) \textit{without determining
a 1RM}. So their hearts were in the right place, although a 1RM was actually determined in the study
for scientific reasons, for 21 males > 65 years old. These geezers also performed high-velocity leg presses
at various loads in random order and reported RPEs. Regression analysis showed high correlation of
the maximal RPE with measured 1RM (no surprise there), which, according to the authors, allowed
for the prediction of an optimal load for explosive resistance training (ERT) with the same exercise.
There was no actual demonstration whatsoever that an ERT load so predicted actually turned out to be
optimal for explosive geezer training. Of course, establishing the yardstick for any such demonstration
would be a neat trick indeed. It is \textit{impossible} to do a heavy power clean non-explosively, even if your
technique sucks. When somebody racks a clean at any but the most trivial weight, you know they
just performed an explosive movement. But I rather fancy that the threshold between an “explosive”
leg press and a “regular” leg press is just a Big Fuzzy Gray Zone. Maybe somebody should come up
with a subjective RPEX (rating of perceived explosiveness) scale. Then you could combine the entirely
subjective RPE with the entirely subjective RPEX. That would just \textit{clear things right up}.

Two papers looked at salivary hormone analysis. Caruso et al\textsuperscript{118} showed that salivary testosterone
levels rose after a high-speed isokinetic resistance exercise bout in men, but not so much in women,
when measured at a single time point after exercise. Cortisol levels did not change at this time point.
No blood assays were done, so we have no idea how this correlates with serum testosterone values, the
temporal signature of the response, or exactly how we're actually supposed to use this information to
make people stronger.

Crewther et al\textsuperscript{119} tried to show a relationship between baseline strength and salivary testosterone
in a practical coaching context. In my opinion, they failed. Ten professional athletes (rugby, mostly)
were divided into two groups: “good squatters” (>2.0 BW) or “average squatters” (<1.9 BW). The
authors report that \textit{the good squatters were stronger than the average squatters!} This is just amazing: it's
like saying you divided your fruit basket into apples and oranges and then reporting that the apple
group had more apples. Unbelievable. On 10 separate occasions over a forty day period, these guys did
squats and sprints, and had salivary testosterone and cortisol concentrations measured. Both groups
actually increased their salivary testosterone concentrations over the forty days – the most interesting
and important finding of the study. This is not what interests the authors, however. What interests
them is the difference between these two groups. And there they have a problem, because the two
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curves (Figure 3 of the manuscript) lie right on top of each other. In every case, the difference between the two groups is no more than a few pg/ml. In other words, looking at the raw data, a child could see that there was no meaningful difference in testosterone elaboration between these two groups. But through the magic of statistical legerdemain, the authors are able to conclude exactly the opposite: pooled testosterone correlations were strong and significant in the good squatters, but not for the average squatters. They claim that this information can be of use to coaches and exercise scientists, and that salivary testosterone measurement will allow us to predict strength and properly allocate athletes into performance and measurement groups. I suppose that's possible. But this data doesn't show it. Their study is so small, so vulnerable to confounding, so poorly controlled at the level of training regimen, the differences observed so lacking in apparent biological significance, and the conclusions so heavily based on derivative statistics, that the findings can have no immediate practical implications.

Other papers with relevance to the Metrics subcategory are discussed in other sections. These include papers by Daniel Robbins and by Mann et al, under Sport-Specific/Football, and by Sheppard et al under Sport-Specific/Volleyball.

VB. General coaching practice and concepts.

Although not a peer-reviewed paper, I would be remiss if I did not point out the remarkable presentation by Dr. Stef Bradford at the Starting Strength Coaches' Association Conference in October 2012. A scholarly and thought-provoking examination of how coaching affects and is affected by the motor and perceptual maps of both the trainer and the trainee, it should be viewed by anyone with an interest in strength training. Some of the ideas presented here mandate further investigation in the research setting.

The article by Alex Walsh in the April issue of the Strength and Conditioning Journal is not a research paper or systematic review, but it is a well-researched and well-presented exposition of a philosophy of coaching that in some respects might fit well with the SS model. Walsh emphasizes the importance of not overwhelming the trainee with cues or feedback, the importance of setting discrete, specific goals (for example, focusing on a particular technical aspect of the lift during a particular rep, set or workout), fostering the development of an integrated cognitive-motor map of performance that will allow the trainee to “self-regulate” and require progressively less cueing and feedback, and the use of ritualistic behaviors (the “obsessive-compulsive style of self-regulating”) to lock in positive habits and minimize form creep. There’s a lot to think about here.

Less useful, I’m afraid, is the “study” by Tod et al on the professional development of coaches. It’s not science, it’s an analysis of loosely-structured interviews of 15 coaches, with an average of 11 years of experience. The authors report that coaches got more mature and confident and caring about their clients as they got older, and less “narcissistic” about their control over athletes and their competitive results. The study is too implicit in its analysis and far too small in its scope to provide any real guidance or hard data. Its inclusion here tells you something about the current state of the literature on coaching practice, professional development, and coaching technique. We have a long way to go.

Two other papers made it into this category for lack of any other clear repository.

Pinto et al looked at the effect of range of motion on muscle strength and fitness. They looked at strength increases and muscle hypertrophy in the elbow flexors of subjects who engaged in 10 weeks of preacher curls at either full range of motion or partial range of motion, and found that both outcomes were enhanced by training through a full range of motion. This paper’s value, such as it is,
lies in its illustration of the growing use of ultrasound in evaluation of muscle hypertrophy. I confess to some astonishment that anybody would consider the results of this study novel or illuminating.

Crow et al. compared three different warmup protocols on the explosive power output of Australian Rules Football players. They compared no warmup (control), a 45s whole-body vibration protocol, and a 5-minute protocol consisting of a series of gluteal activation exercises, evaluating their impact on the athletes’ performance of a weighted countermovement jump on a Smith machine. Experimental procedures were conducted about 1 hour after football practice. You don’t have to be a scientist or a coach to shoot holes in this study with the information I just gave you: these athletes weren’t exactly “cold,” comparing 45 seconds on a vibrator to a 5 minute butt muscle routine smacks of apples and oranges; using no warmup at all as a control group is just silly; and using the Smith Machine countermovement jump at a single load (20 kg) isn’t exactly a state-of-the-art approach to measuring explosive performance. But it doesn’t matter: the differences, while statistically significant, are of no practical significance. Buttcrunch, Vibrator, and No Warmup protocols resulted in 4565 ± 634, 4267 ± 658 and 4374 ± 659 W peak power, respectively. There can be little doubt that the gluteal complex is critical to explosive power development, and that warmups are good for you. And there’s some reason to believe that postactivation potentiation (not invoked as a mechanism here) can optimize performance. But this study proves none of that.

**VC. Sport-specific studies.**

This section examines studies with sport-specific relevance or, in some cases, studies with sport-specific populations. Some of these studies have significant crossover with other categories and subcategories, and in some cases their assignment here rather than there will appear somewhat arbitrary, because it was. Hopefully, this section will get bigger in the future, as improvements in the data quality in general and sport-specific metrics in particular improve. As it stands, not too many studies made the cut, and even those that did were not of the highest caliber, as we shall see.

**VC(1). Football.** The two papers that fell into this section could just as easily have been assigned to the Metrics subcategory, and neither one is liable to have much of an impact on the average strength coach; their interest is primarily academic. Daniel Robbins conducted a study of a very large dataset of American football players (N=1136; data from the NFL’s annual combine) to determine the relationship between body mass and performance outcomes. The conceptual driver here is allometric scaling, which aims to assign an exponent to the body mass variable to maximize correlation with the performance variable in the equation:

\[ NP = P/M^b \]

Where NP is the normalized performance metric, P is the actual measured performance, M is body mass, and b is the allometric exponent derived by analysis of a large data set. If \( b = 0 \), then \( P/M^0 = P/1 = P \), which means that the normalized performance is independent of body mass. There are suggestions in the literature that this is the case for explosive movements. The author of this study analyzed the data for 3 different sprint intervals (9.1, 18.3 and 36.6 m), vertical and horizontal jumps, the 18.3m shuttle, and the cone drill. He found nonzero allometric exponents for the sprinting and jumping tasks. Exponents for the agility tasks, however, approached zero value. The author concludes that normalization-for-mass of explosive movements in populations with large variation in body mass are
indicated, but not normalization of agility tasks. No data on Olympic-style movements or nonexplosive strength tasks is provided.

Mann et al.\textsuperscript{125} investigated the correlation between the NFL-225 test and 1RM bench press strength in NCAA Division I football players. The NFL-225 test assesses how many times the player can bench press 225 lbs. The authors found that this test produced a high degree of correlation with the actual 1RM. The methods here are solid, and the data set is large (N=289), but the results boil down to this: the stronger you are, the more times you can lift 225 lbs. The authors claim that the ability of the NFL-225 to predict actual bench 1RM means that coaches can (1) dispense with the 1RM test and its potential for injury and (2) use a predicted 1RM to guide periodized strength programming. I agree with the first part.

\textbf{VC(2). Volleyball.} The paper by Sheppard et al.\textsuperscript{126} is a pleasant surprise. Performance metrics for this sport are fairly well-developed, and include squat strength, jump squat, countermovement jump, jump squat, loaded jump squat and power clean. These metrics, along with body composition, were followed in sixteen bros (aged 18.5 \pm 1.5 years) followed for a two year longitudinal study. During this period, all these guys successfully transitioned from junior national to senior national team competition and scored professional contracts. Their training year is well-described. The subjects improved their performance in all variables, including body composition. Power clean and squat performances were not at all what competitive strength athletes would consider impressive, but then these aren’t competitive strength athletes, and the authors provide a reasonable sport-specific argument for maximizing lean body mass ratios (i.e., decreasing body fat) in volleyball players. This paper is similar to the paper on rugby players by Applebee\textsuperscript{127} discussed in last year’s review. It demonstrates that, despite the constraints of competition, sport-specific body composition parameters, the demands of skill and tactical and team training outside the weight room, it is possible and in fact imperative for athletes to improve their strength and explosive performance, using a rational, periodized approach. This paper uses relevant performance benchmarks in athletes who actually achieved something during the course of the study. It will be a worthwhile read not only for volleyball coaches, but for athletes and coaches generally.

Not so much the paper by Voelzke et al.\textsuperscript{128} These investigators compared two complex programs in elite volleyball players: resistance training + plyometrics (RT+P) and electromyostimulation + plyometrics (EMS+P). EMS is the approach of strapping electrodes to the bro and stimulating the muscles to contract while the bro does something else worthwhile, like watch \textit{Sons of Anarchy} or read another fine issue of \textit{Juggs}. At the end of a 5 week training period, the authors tested isometric leg extensor strength, squat jump, countermovement jump, and drop jump. They report some minor differences, some favoring EMS+P, some favoring RT+P. They recommend that coaches integrate both approaches into their training program. But the differences reported here are so small, the time window of the investigation so short, and the experimental groups so tiny that I don’t think the study is very illuminating at all. More importantly, the study is poorly controlled: there are no RT-, EMS-, or P-only groups.

\textbf{VC(3). Other Sport-Specific Studies.} For all you cycling enthusiasts, we have the paper by Louis et al.\textsuperscript{129}, who examined the effect of strength training on the cycling efficiency of both younger and older athletes. Nine geezers and 8 bros were tested for maximal knee extensor torque, cycling efficiency and VO\textsubscript{2}max. They were all then put on a program of “strength training” using – you guessed it – leg presses. For three weeks. After the program, post-training revealed a statistically significant improvement in cycling efficiency for the geezers, but not the bros. The authors wave their hands
about the physiologic bases for these observations, including some very well-taken speculation that the increased force production capability resulted in a reduction of the relative torque needed for each movement cycle. Of course, that doesn’t explain why both groups didn’t improve their efficiency. But there’s no use worrying about it: this is another study that is just too small and too short, observing differences of unclear clinical significance (despite the statistical significance). It’s almost certainly true that stronger cyclists have better cycling efficiency, and that strength training will help Masters athletes maximize their performance. But this study doesn’t prove that.

Lawton et al130 looked at the effect of an off-season strength-training program on the strength and endurance of elite rowers. A total of 22 rowers were randomized into two groups: one group performed a program of on-water rowing in which the volume was progressively increased, while the other engaged in a progressive, 8-week resistance program. A decent attempt was made to normalize total training volume between the two groups. Pre- and post-intervention testing assessed body mass (an important variable for rowers), aerobic condition (using a braked-ergometer-based protocol and lactate threshold testing); upper body strength (by 6RM bench pull, a kind of prone rowing exercise); lower body strength (isometric pull on a force plate), lower body endurance (30R leg press) and upper body endurance (60R seated arm pull). The authors report that differences in body mass pre- and post- were trivial between the two groups, as was the difference in aerobic conditioning – both groups improved their overall endurance. Both groups got stronger, although the resistance-trained group demonstrated superior increases in lower body strength. The authors claim that gains in upper body strength were similar for the two groups, which gives me a headache, because when I look at the data I see that the improvement in the 6RM bench pull, the measure of upper body strength, was higher in the resistance training group. The study is of limited value, but it does suggest that off-season resistance training will not be detrimental in elite rowers. And that’s important because, as the authors point out, recent work by Gee et al has called into question the value of resistance training in elite and professional rowing athletes131,132.

Nimphius et al133 did a very nice little study of female softball players who performed a periodized RT program during the competitive season. They showed that improvements in strength, power and performance can continue during the competitive season and that these improvements correlate with performance parameters and the expected changes in muscle architecture.

Finally, Argus et al134 examined 112 Union Rugby players at various stages of development and found that more advanced rugby athletes were stronger and more powerful than their high-school, amateur or semipro counterparts. A solid analysis, but not exactly a revelation.

VD. Profession-specific studies

The literature on strength training in the workplace unfortunately seems to be dominated by studies for the “manly” professions: soldiers, cops, firefighters and the like. And, as a rule, they’re not that great.

For example, Pryor et al135 studied the fitness and body composition of a suburban SWAT team. This was a strictly observational study, in which eleven male SWAT operators were observed in the performance of their duties. Based on these observations, a physical therapist decided, implicitly, what the physical demands of SWAT operations were. The operators were then subjected to tests of strength, power, flexibility, core strength and body composition. Some of these assays were questionable, but we’ll set that aside. VO2max was above average, although apparently not good enough for the investigators, who proclaimed that these guys needed more aerobic capacity. Body fat was a bit above average, strength (measured by bench press) was way above average, and mean vertical jump was right
at the mean for the general population. I implore readers to stop and reflect on this last finding for a moment.

Based on this and other data, the investigators conclude that these SWAT operators need more core strength and aerobic conditioning. But they don’t tell us exactly why. Their reasoning seems to be simply that these measures were not as high as they thought they should be. The authors report no actual detriments in performance among these operators. Nor is there any description of an intervention with dependent variables. The investigators developed a personalized fitness routine for each subject, but as payment for participation in the study, not as an intervention to be evaluated. The best that can be said of the study is that the investigators attempted to derive performance metrics for SWAT operators. But they didn’t study how those metrics correlated with actual performance, nor did they test improvement in either those metrics or performance with a studied intervention. Disappointing.

Rossomanno et al\textsuperscript{136} took 175 St. Louis cops and made them do aerobics and bodyweight strength exercises for six months. They observed fairly trivial decreases in BMI and unsurprising improvements in fitness. The calisthenics were chosen to improve “muscular strength and endurance specific to police officers:” pushups, pullups, dips, situps, lunges, standing side leg lifts, bodyweight squats, and back extensions. How these exercises were selected or how they are specific to police work escapes me. We are given no indication that the intervention had any impact on officer performance. Again, disappointing.

Knapik et al\textsuperscript{137} conducted a systematic review and meta-analysis of the literature on the effects of physical training on load carriage performance in soldiers. The study is limited by the paucity of good data on the topic, and the variations in the methodology and quality of the studies selected for analysis. The authors attempted to normalize the data by using effect sizes (the Cohen’s $d$-statistic). They found that aerobic or resistance training alone provided the least benefit in load carriage performance, that combined aerobic-resistance training provided more robust improvement, and that a program incorporating progressive load-carriage training provided the largest effect sizes in improvement. As such, the paper is a beautiful illustration of the principle of training specificity.

The use of resistance training in the workplace was examined by Zavanela et al\textsuperscript{138}. Brazilian bus drivers (Sao Paulo again) were randomized to training or no training for 24 weeks. The program is well-described, and did include progressive programming, although the prescribed exercises may appear silly to some of the readers of this article. The investigators report improvements in strength, muscular endurance, blood pressure, body composition, flexibility, pain incidence, and absenteeism. The discussion is worth reading, and the results comport with what we know about training. Unfortunately, the entire paper is tainted by some strange hijinks in the methods section. The study started with 132 men randomized into training ($n=60$) and control ($n=72$) groups. But the study ended up with both groups at $n=48$, “because of noncompliance with the study protocols and unforeseen injuries.” What noncompliance? What unforeseen injuries? How were the subjects reallocated into groups? Was the randomization violated? What the hell happened? The authors don’t tell us, and I for one would really like to know. Employers thinking about a workplace training program are likely to be curious, too.

Pertinent to this category, and more valuable than any of the papers actually discussed under this heading, is the paper by Kraemer and Szivak, \textit{Strength Training for the Warfighter}, discussed under Programming.

\textbf{VE. Gear.}

We now turn our attention to studies addressing the use of gear in strength training. The study that might have been of the most interest to competitive lifters is, alas, too flawed to be of much use. Godawa
et al\textsuperscript{139} looked at the influence of compressive gear on powerlifting performance, ostensibly in light of the effect of blood flow restriction training (which would have given it some crossover interest with the Kaatsu paper discussed previously\textsuperscript{41}). They studied 18 collegiate powerlifters, who were randomized into two groups: those who trained with (CG) and those who trained without (NON) compressive gear. The two groups differed quite a bit in their baseline strength, which was measured by holding an impromptu little powerlifting meet. We are not explicitly told whether the meet was performed geared or raw. The lifters then underwent 10 weeks of training, with or without gear according to their group allocation. The description of this training program, unlike the study results, will be of interest to powerlifters, and is the real reason to get and read the paper. After 10 weeks, the subjects participated in a local powerlifting meet. Again, we are not told whether the meet was geared or raw. Both groups significantly improved their performance. There were no significant differences in change in performance between the CG and NON groups, although the authors make much of the trend toward increased performance in the CG group. Of course, if they had properly powered their study (no power analysis is provided), they might not have to wave their hands about so much. There’s also some speculation about the role of blood flow restriction in the better performance of the CG group (which better performance, as we just noted, was not actually observed). And it’s just that: speculation, since the authors did not actually design the experiment to look at this mechanism.

“Knee wraps should not be worn during the strength and condition (sic) process.” This is the conclusion reached by Lake et al\textsuperscript{140} in their study of the effect of wraps on mechanical output and performance characteristics of the back squat. Ten bros did back squats on force plates, with and without wraps, and were videotaped for kinematic analysis. The findings showed that knee wraps definitely conferred a mechanical advantage, and resulted in an unexpected decrease in horizontal displacement of the bar. No direct measurement of mechanical energy deposition into or released from the knee wraps was undertaken. No measurement of forces in and around the knee joint, or of the long-term clinical health of the knee joint was undertaken. Nevertheless, the authors conclude that (a) the mechanical advantage they observed resulted from the storage and release of mechanical energy in the knee wraps; and (b) that wraps should not be worn because they could lead to damage of the knee joint. Neither of these conclusions can possibly be supported by this data. All that remains is to wait for word to get out that it’s been scientifically proven that wraps will destroy your knees. I recommend you print out this paper, read it, note my objections and yours in the margins, and keep it with you…so you can roll it up and use it to bitch-slap the first person who says this to you.

Sato et al\textsuperscript{141} examined the differences in squat kinematics between lifters who wore weightlifting shoes and those who wore running shoes. They note the objections to shock-absorbing footwear (citing, among others, Rippetoe and Kilgore). They also note the antediluvian NSCA position statement on squat technique, released in 1991 and based on literature up to the 1980s, which states that the torso must be held as close to the vertical as possible during the entire lift. The authors had 25 subjects squat (incorrectly) in front of a camera with segment markers with both types of shoes, and demonstrated (or so they claim) that weightlifting shoes help you squat with a more vertical back. Therefore weightlifting shoes are good. After I threw this paper at the wall, I wondered if it might be useful in getting more people to wear squat shoes. But of course, that argument has already been made elsewhere\textsuperscript{142}, and better.

Dunn-Lewis et al\textsuperscript{143} examined the effects of using an over-the-counter mouth guard on lifting performance. The report improved performance with the mouth guards of upper-body lifts in men and women, and lower-body exercises in men, although they are at a loss to explain the gender difference. The discussion of the mechanism leading from chomping at the bit to increasing your power under
the bar is worth the candle. I won’t belabor the methods, which are so-so, because the findings are more-or-less in line with previous literature, and this is one of those things you can try for yourself at minimal cost and risk. I wouldn’t expect it to add twenty pounds to your squat.

VI. SPORTS MEDICINE

This section has the potential to expand significantly in future iterations of this project, but this year’s selections were held at a minimum. Many papers that might have been included here were not specific to strength training, or were covered in other categories, or just weren’t interesting enough to merit inclusion. Very little sports medicine literature is devoted specifically to strength training populations, although I think we may expect that to change with time. Moreover, I have recently written an article dealing with the use of anti-inflammatory therapy, including NSAIDs and cryotherapy, which is generally applicable to all forms of exercise, including strength training.

Paoloni et al144 looked at both long-term and short term impact of a treatment regimen for patellofemoral pain syndrome (PFPS), a very common cause of knee pain, particularly in younger athletes. It is generally believed (although not definitively proven) to be due to a “tracking error” of the patella over the groove in the distal femur. This tracking error is thought by some to be, in turn, due to a discrepancy in the timing of activation of the vastus medius obliquus (VMO) and vastus lateralis (VL) during knee extension. The authors selected 44 Italian bros and babes suffering from PFPS and measured their pain on a VAS, characterized the temporal activation of the VMO and VL by EMG, and assessed knee extensor strength on a machine and a half-assed isometric loaded squat. The subjects received two weeks of patellar taping, which applied tension to the patella in a lateral-to-medial direction. The patients then underwent twelve months of physiotherapy. All subjects performed the same exercises, which were supposed to improve VMO activation. Short-term and long-term primary outcomes (pain, disability, strength) were markedly improved. VMO and VL activation were reported as improved, but the differences, while statistically significant, were of unclear clinical significance. The study is strengthened by an appropriate sample size calculation and long-term functional assessments. The lack of a control group and the failure to compare different physiotherapeutic regimens limits its conclusions.

Owing to personal bias, two papers were selected on the topic of the greater trochanteric pain syndrome (GTPS), a common cause of hip pain in the elderly and in athletes, and an important entity to distinguish from osteoarthritis. The condition arises from inflammation of the trochanteric bursa, resulting in lateral hip pain (over the trochanter), pain on flexion-abduction-external rotation (FABER maneuver), and pain when lying on the affected side. It can be a sonofabitch, and wreak total hell on one’s programming. Ask me how I know.

Fearon et al145 attempted to derive a clinical approach to diagnosis that would differentiate GTPS from OA. They took 41 adults with GTPS (diagnosed clinically) and 23 with clinical and radiographically confirmed OA and had them evaluated by a physiotherapist to derive classification trees by recursive partitioning. The resultant clinical instruments are reasonable, but the study is hampered by gold-standard issues: they used patients with a clinical diagnosis of GTPS to derive clinical criteria for GTPS. Even so, their best decision instrument had sensitivity and specificity of about 80% each – not so great. This is not entirely their fault: there are no reliable radiographic or laboratory markers for GTPS.
Del Buono et al\textsuperscript{146} conducted a systematic review of management of GTPS and found pretty much what I expected they'd find: nobody knows the best way to treat this bastard, and the systematic review doesn't clear things up, because the source data is far too limited in scope and quality. Therapeutic options range from conservative (anti-inflammatory, stretching, exercise) to silly-but-lucrative (injection of the effected area with vile potions) to extreme (division of the iliotibial band and resection of the bursa). There is an indication that repetitive low-energy radial shock wave therapy and home training may be the best long-term option over the long term...and you get to keep the bursa.

Although too extensive to discuss in detail here, the review by Maffuli et al\textsuperscript{147} on outcomes of sports injuries is a good one to have in your file as a general reference. Long story short: athletes accumulate injuries and tend to develop osteoarthritis in the spine and weight-bearing joints at a rate that may exceed that of the general population. What is not clear (to the authors, at least) is whether the benefits that accrue from exercise outweigh the detriments of degenerative joint disease in active individuals over time. This question seems to me to hinge at least partly on one's values, and as such may prove to be difficult to resolve with the scientific method.

VII. Nutrition, Supplements and Drugs

VIIA. Anabolics and hormones.

Macaluso et al\textsuperscript{148} present an interesting though flawed study of conjugated linoleic acid (CLA) supplementation that combines \textit{in vitro} and \textit{in vivo} data. CLA is a group of isomers of linoleic acid, an essential omega-6 fatty acid. CLA has drawn considerable interest for its ability (in animal studies, at least) to improve body composition, minimize inflammation, improve VO\textsubscript{2}max, and possibly even upregulate tumor suppressor genes and exert anticancer effects. Data in humans is rather more conflicted and controversial. The authors proceed from observations of the effect of CLA on perilipin, a protein in adipocytes. Perlipin is an important regulator of fat storage. The authors note that this protein is also present in Leydig cells, and hypothesize (rather breezily) that CLA supplementation may mediate an increase in testosterone synthesis. They supplemented cultured rat Leydig cells with CLA, and observed an increase in testosterone production, but only after 48 hours. They then performed a crossover study of 10 Sicilian bros, randomizing them to either placebo (sunflower oil) or CLA (6g/day). Their results suggest a bump in testosterone after a resistance bout in CLA-supplemented bros that is only slightly greater than that observed in placebo-treated bros. Overall, intriguing, but not at all convincing. There are problems here, including the use of a wimpy exercise bout, a very small sample size, the use of young men in springtime (already swimming in T), and the crossover design, which I thought inappropriate to a study like this. But we definitely want to keep our eyes on this topic. Stay tuned.

Hakansson et al\textsuperscript{149} performed a survey of the Swedish population to get a picture of drug and steroid abuse. The response rate was horrible (<40%) and their telephone-based methods to adjust for it are unconvincing to this reader. They report, like several other investigators before them (see the horrible paper by Ip et al from last year's review), that there is an \textit{association} between illicit drug use and anabolic steroid use. In Swedes. Swedes who responded to the survey. Swedes who responded and told the truth. Seriously, we can't be too surprised by this association, which admittedly now has a lot of evidence (most of it of dubious quality) behind it. But there's no demonstrated \textit{causal} mechanism
working in either direction, and at the end of the day all these findings mean is that people who abuse substances are more likely to...abuse substances. Stop the presses.

Dieli-Conwright et al\textsuperscript{150} looked at the effect of resistance exercise and hormonal therapy on the expression of myostatin and related gene products. We discussed myostatin in last year's review, in the context of the paper by Dalbo et al\textsuperscript{151}. As I'm sure you'll all remember quite clearly, 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. In other words, myostatin \textit{is not your friend}. It is assisted in its nefarious muscle-wasting work by ActRIIb (another unfriendly gene product) and antagonized by your allies in strength, the proteins follistatin, FLRG, FSTL3 and GASP-1.

The authors took 14 postmenopausal golden gals, eight of whom were on hormone therapy (HT) and 6 of whom were not, and took muscle biopsies. After an appropriate interval, all the geezettes got a serious workout of 10 sets of 10 maximal eccentric repetitions of leg extensions on a dynamometer. After four hours, muscle biopsies were again taken. Biopsies were subjected to RNA extraction and reverse transcription to a cDNA library, and then taken to qualitative real-time polymerase chain reaction for determination of relative gene expression levels for myostatin, ActRIIb, follistatin, FSTL3, FLRG, and GASP-1, using the “housekeeping” gene for glyceraldehyde 3-phosphate dehydrogenase as an internal standard. The authors report that controls (women not taking HT) demonstrated an approximately four-fold decrease in myostatin and ActRIIb gene expression over baseline, and about a 2-fold increase in follistatin, FSTL3, FLRG and GASP-1 over baseline – in other words, a trophic response. But women who were on HT mounted a nearly 20-fold decrease in myostatin, and a nearly 10-fold decrease in ActRIIb, with similarly dramatic increases in muscle-friendly genes over their non-HT sistahs. In short, in this study, resistance training induced a trophic response in the expression of myostatin and myostatin-related genes in all subjects, and \textit{estrogen and synthetic HT seemed to function as anabolic agents.}

But hold on. As with the Dalbo paper, there are problems. This study is small, and the groups studied were not as homogeneous as we would like. The women on HT were not all on the same HT – some were on estrogen only, while others were on estrogen-progesterone supplementation. There was significant variation in the BMI of these ladies – important, since obesity appears to have an impact on myostatin regulation. As in the Dalbo paper, the authors looked only at gene expression (mRNA, reverse-transcribed to cDNA), \textit{but not at the actual gene products}, the proteins that actually do the work. Finally, as is too often the case, there was no correlation of these findings with actual changes in muscle mass or strength, so the suggestion that HT exerts an anabolic effect through myostatin and related genes seems premature based on this data. A fascinating area of investigation, though: the down-regulation of myostatin by exercise, and possibly by pharmacologic supplementation, is a tantalizing target for Old Farts who want to stay strong.

\textbf{VIIB. Ergogenics.}

\textit{Ergogenic} is a term that can be loosely applied to any performance-enhancing drug. Like certain other authors, I limit its use to those substances or interventions that act as acute stimulants or that promote the generation of force or power through metabolic, non-anabolic means. The two most broadly used ergogenics in strength training are creatine, which is supposed to improve the regeneration of high-energy nucleoside triphosphates in skeletal muscle, and Caffeine, the Most Holiest of Holies, which, as we all know, restores the Life Essence Itself.
We covered some research on caffeine last year, and this year we have three more papers to discuss, all of which confirm that caffeine is proof God loves us and wants us to work out. Del Coso et al\textsuperscript{152} showed that an energy dosed at 3mg of caffeine per kg of bodyweight was more effective at increasing power in the half squat (losers) and the bench than a drink containing only 1mg/kg of caffeine. Duh.

Mora-Rodriguez et al\textsuperscript{153} performed a battery of neuromuscular tests at different times of the day on 12 Spanish bros. They measured dynamic muscle strength and power output in the squat, bench, knee extension and arm grip, and also assayed catecholamine levels. Bros who used caffeine jacked up their catechol (norepinephrine) levels and overcame the morning blues better than bros who did not, and their morning performance was closer to their afternoon performance. This prompts the question of why the investigators left out a critically important experimental group: wide-awake Spanish bros working out \textit{in the afternoon} with caffeine. Best of both worlds. Cooking with gas.

Finally, Duncan et al\textsuperscript{154} showed that caffeine had a salutary effect on various loosey-goosey subjective parameters like rating of perceived exertion and readiness to invest mental effort, in addition to performance. These findings comport with those of other investigators, including the paper by the same investigator that we discussed last year. Drink java. Lift more. Feel good about yourself. \textit{Be somebody}, baby.

In terms of quality, none of these studies are particularly good or particularly bad. They're small and short, as usual, but not particularly contentious. And they all add to a growing body of literature suggesting what you already know: the vital first step in lifting weights is to brew a pot of Joe.

We now turn our attention to the three papers investigating the role of creatine. The principle mechanism for the ergogenic effect of creatine is the rapid replacement of high-energy phosphate in metabolically active tissue. In such tissue, creatine is maintained in its phosphorylated form, as creatine phosphate (CrP). As the tissue performs biochemical and mechanical work, ATP undergoes hydrolysis to ADP, giving up the high-energy terminal phosphate. Without CrP on hand, the tissue would have to rely on glycolysis in the cytoplasm (anaerobic) or oxidative phosphorylation in the mitochondria (aerobic) to maintain cellular ATP concentration and cellular energy levels. Both of these mechanisms are much slower than the transfer of high-energy phosphate from CrP to ADP, catalyzed by the enzyme creatine kinase. So the presence of CrP allows the cell to maintain ATP concentration in the face of extremely high energy demands for just a little longer than would otherwise be possible. Other mechanisms, such as the buffering of hydrogen ion (acid), improved delivery of phosphate to myosin heads, and stimulation of phosphofructokinase, a key glycolytic enzyme, may also play a role in the ergogenic effects of creatine.

The upshot of all this biochemistry is that \textit{creatine itself does not make you stronger or more powerful}. It doesn't make sarcomeres contract harder or faster. But it may help your muscles work longer, just a bit longer, before they run out of gas. This is underscored by the study published by Zuniga et al\textsuperscript{155}. They performed a placebo-controlled trial of 22 bros randomized to either creatine (20g/day for 7 days) or maltodextrose placebo. After 7 days, both groups showed similar (small) increases in 1RM leg extension, bench press, mean power and peak power. Only the change in mean power was significantly different between the two groups. In short, the study showed that creatine supplementation resulted in an increased capacity to perform anaerobic work – but not an increase in strength. The paper by Spradley et al\textsuperscript{156} supports this view, although their study is muddied by the use of a supplement stew containing not only creatine but B-vitamins, caffeine and amino acids.

These studies, along with others in the literature and our knowledge of the biochemical mechanism of creatine action, support a model of creatine use in strength training in which the trainee
does not rely on creatine to make her stronger. Rather, the trainee uses creatine to increase anaerobic work capacity – in other words, the capacity to grind out that one last rep. And over time, that last rep should make her stronger. This makes an awful lot of sense, and I think it’s probably right… but it hasn’t actually been shown yet. We’re too busy doing silly studies showing that seven days of creatine loading won’t increase your bench 1RM.

A nice little paper by Jagim et al demonstrated that a buffered form of creatine did not produce higher muscle creatine levels than a standard (and probably less expensive) creatine monohydrate preparation. Caveat emptor.

VIIC. Analgesics and anti-inflammatories.

Although neither a peer-reviewed publication or systematic analysis, Sullivan presented an informal review of the literature on anti-inflammatory therapy and strength training on the Starting Strength website that may be of interest to some readers. In my self-serving opinion, it is the most definitive review of the topic available from the perspective of strength training. The article includes a review of the pathophysiology and pharmacology of inflammation and anti-inflammatory therapy, and contains 47 references and summary tables for the relevant literature. Geek out.

VIID. Other supplements.

In 2013, the three papers that fell out for this catch-all subcategory included two related to supplements targeted at enhancing nitric oxide (NO) generation, and a paper on telmisartan and doping.

The production of NO is a critical biological signaling event with diverse and powerful effects. NO produces vasodilation, which gives it an important role in modulating blood flow, regulating blood pressure, and Getting A Woody. Sildenafil (ViagraTM) and related pharmaceuticals potentiate the effect of NO by acting on second messenger systems (at the level of cyclic GMP). NO also functions as a bactericidal agent, neurotransmitter, and regulator of smooth muscle function. In certain disease states, NO can be transformed into peroxynitrite (ONOO-), a viciously reactive and toxic anion that contributes to oxidative molecular damage and cell death.

Manipulation of NO by supplements is thought by many to improve muscle blood flow via vasodilation, increasing nutrient delivery and waste removal from muscle beds, and perhaps improve muscle function by other, as-yet-unclear mechanisms (“perhaps at the level of cGMP” – always an impressive and plausible-sounding speculation when you have no idea how the hell something works). L-arginine, an amino acid, is the substrate for a family of enzymes collectively known as nitric oxide synthase, or NOS. In the presence of NADPH, an essential reducing agent commonly found in anabolic reactions, NOS converts L-arginine into citrulline and NO:

\[
\text{L-arginine} + \frac{3}{2} \text{NADPH} + \text{H}^+ + 2 \text{O}_2 \rightarrow \text{citrulline} + \text{nitric oxide} + \frac{3}{2} \text{NADP}^+
\]

Hence the rationale for the use of L-arginine as a supplement: it provides increased substrate for NOS, which increases NO production, which increases muscle blood flow, which allows you to get your pump on, which gets you swole.

Unfortunately, the literature on L-arginine is mixed, and the paper by Wax et al doesn’t clear things up, because they studied a cocktail of L-arginine and alpha-ketoglutarate, a citric acid cycle intermediate which is also supposed to make you strong like bull, but through a completely different
mechanism. The authors took 8 trained and 8 untrained bros, gave them the L-arg/AKG cocktail or a placebo, tested them for 1RM on the bench and leg press, made them do volume sets of both at 60%, and let them go home. The bros came back a week later, switched treatments (crossover design) and went for the same testing regimen. The authors report no effect of L-arg/AKG, and they claim their sample size calculations had the power to detect a difference. But the study is hopelessly flawed. The crossover design, the use of a gmish of trained and untrained bros, the study of acute dosing only (especially in light of literature indicating that loading with L-arg over time produces better results), the lack of any practical outcome measures (strength, hugeness, Woody quality), all render the findings essentially meaningless from any practical perspective.

Bloomer et al\textsuperscript{160} conducted a study of 2-nitrooxy ethyl 2-amino 3-methybutanoate gel, which is supposed to increase circulating NO levels, through a mechanism that is not clear (the molecule looks like it could be a NOS substrate). The authors noted no difference between the gel and placebo for any outcome measure, including the nitrate/nitrite surrogate marker (NO was not directly measured). However, the authors point out that some of the bros did get a pump on with the gel. In a completely brazen post hoc manner, these bros were labeled as “responders.” The authors then report that responders responded more than non-responders, and state in their conclusions that “self-experimentation is needed.” Translation: our little study didn’t show a difference, but you should buy the product and try it anyway. You might be a responder! This study was funded by a company called Advanced Oral Technologies. Guess what they make?

Sanchis-Gomar and Lippi\textsuperscript{161} present a short editorial review on the status of telmisartan as a doping agent. They note that AICAR and GW1516, agents that may have similar biochemical effects, have recently been reclassified by the World Antidoping Agency as “hormone and metabolic modulators.” To put it simply, these compounds work on the AMPK switch we examined in the myophysiology section. In so doing, they optimize the “long-distance running” phenotype (which the authors seem to regard as optimal). This paper is of interest for its elaboration of the mechanisms by which the AMPK switch can be manipulated with supplements, and for its indictment of telmisartan as having similar properties. This is important, because telmisartan belongs to a class of antihypertensives known as angiotensin receptor blockers, or ARBs. Like their cousins, the ACE inhibitors, these agents would seem to work better for strength trainees who require antihypertensive therapy than more traditional agents like thiazides, beta blockers and calcium channel blockers. If, however, ARBs activate AMPK to any clinically relevant degree, they would act at cross-purposes to strength training, pulling the athlete away from the Joe Weightlifter phenotype and toward the Marathon Mike phenotype. However, the AMPK-modulating effect has not been observed for the ARBs losartan and candesartan, and the long-term effect of antihypertensives of any type on training is woefully understudied. This paper is of primarily academic interest, but the topic warrants further study.

VIIE. Nutrition.

Symons et al\textsuperscript{162}, from the University of Texas Medical Branch in Galveston, note that evidence of blunted anabolic response to resistance training may be due in part to suboptimal protein ingestion. In this study, a protein-rich meal was administered to both young and older adults (active, but not athletically trained) prior to a bout of resistance exercise (isotonic knee extensions). Prior to the meal, they had been infused with radiolabeled phenylalanine to permit analysis of post-absorptive fractional protein synthetic rate (FSR) performed in vastus lateralis muscle biopsies. Plasma insulin response was
also measured. The authors report that both groups (young and old) displayed a similar increase in mixed muscle FSR.

This is a potentially important finding, but the paper has significant limitations. We have no long-term, trainee-oriented outcome measures: strength, hypertrophy and health-related outcomes were not measured, although to be fair this was a mechanistic study, not a clinical one. We have no idea which proteins were being produced in these two populations, and have to allow that it is possible that younger trainees produce a different library of proteins (more focused on strength and hypertrophy, say) than older trainees (perhaps more directed to repair and recovery). There was no control for the protein meal; that is, the study did not incorporate a group that ate nothing prior to the workout, or a protein-poor meal. And although the authors invoke a dose-response effect for protein ingestion and strength training, they did not test a range of protein doses in this study.

That all being said, the study is another valuable data point indicating that older trainees can mount robust anabolic responses to a resistance exercise stimulus in conjunction with adequate nutrition. In particular, this paper argues for the ingestion of a mixed protein-rich meal prior to training, followed by high protein intake for at least the next 24 hours. In the same vein, Yang et al\textsuperscript{163} found that older men were able to mount enhanced myofibrillar protein synthesis in response to resistance exercise and whey ingestion. Remember: If you don't eat your meat, you can't have any pudding.

Most of the literature on dietary protein intake in the context of resistance training tends to point in this direction. However, there are discrepancies in this literature, and not all studies of protein supplementation support improvement in strength or lean mass acquisition. Bosse and Dixon\textsuperscript{164} conducted a literature review of this topic through the lens of their protein spread and protein change theories, which they have already applied to the existing literature on protein supplementation in weight loss programs.

Simply put, protein spread theory states that for a study comparing high and low protein supplementation to show a difference between the groups in muscle hypertrophy or strength gain, there must be a sufficient spread or difference in the daily protein intake. Protein change theory states that for a study of the effect of protein supplementation to show a beneficial impact, there must be a sufficient change in daily protein intake over baseline. The authors performed keyword searches in PubMed, Cochrane and CINAHL databases to pull down relevant articles. Seventeen papers met their inclusion criteria. The authors extracted figures for total energy intake and protein intake, along with other data, from each paper. They calculated spread and change as follows:

\[
\text{Between group \% spread} = \frac{\text{High prot group intake} - \text{control intake}}{\text{control intake}} \times 100
\]

\[
\text{\% Change} = \frac{\text{study protein intake} - \text{baseline protein intake}}{\text{baseline protein intake}} \times 100
\]

where all “intake” was calculated in g protein/kg body weight/day.

Applying spread and change calculations to the 17 papers of interest, the authors found that studies reporting a benefit of higher protein intake had a mean protein intake spread between groups of 66.1\%, compared with a 10.2\% spread for those studies reporting no benefit. They found an average change from baseline intake of 59.5\% for studies showing benefit of increased protein, compared to a 6.5\% change from baseline intake for studies that did not show a benefit.

This study is limited by the shortcomings of all the source studies included in the analysis (like any other systematic review or meta-analysis), and in particular the data on change theory should be regarded with caution, since many studies do not report baseline protein intake (and even if they...
did, we probably couldn’t trust their numbers). Nevertheless, this paper supports the use of protein supplementation in resistance training programs, and gives us a putative tool for evaluating extant and future papers on the topic.

For example, the study by Weinheimer et al^65 found that whey protein supplementation did not effect exercise-induced changes in body composition and indices of metabolic syndrome in middle-aged obese adults. There are a number of problems with this study, including its narrow end points, the use of low-dose exercise, mixed aerobic and resistance training, poor compliance and high dropout rates, and the use of a free-feeding design. The authors, in their introduction, acknowledge previous findings that men who consumed > 2g protein/kg/day increased lean body mass, while those who consumed 1g/kg/day did not. Then they turn right around and report their results using 0.2, 0.4, and 0.6 g/kg/day (assuming an mean BW of 100kg in their study population). If one were unkind, one might wonder whether they set poor old whey protein up to fail.

Nevertheless, the Weinheimer paper has several strengths that make it stand out in the resistance training literature: it is a large study (n=337) and followed subjects for 9 months. It uses a double-blind, randomized, placebo-controlled, intention-to-treat design. But if we calculate the spread as per Bosse and Dixon (assuming an average BW of 100kg) we get a value of about 40%, which falls below the 66% threshold. The authors of this study don't give us hard data on pre-study protein intake, so the change ratio could not be calculated; I would guess this ratio wouldn't have cleared the Bosse-Dixon threshold, either, given the low rates of whey protein supplementation. This study, on its own, argues that low-dose whey protein supplementation in obese adults with metabolic syndrome in conjunction with a low-dose resistance training regimen subject to interference effects from aerobic training isn't going to do anybody much good. Take that information at face value, and do with it what you must.

Summary

That's my review of the Strength Science literature for 2012. I think it's better and (obviously) more comprehensive than last year's, but it's still a process in evolution. I've probably missed some good papers. I apologize in advance for any errors or omissions in this work: I'm sure I've made a goof or three along the way. When you screen >2000 abstracts and analyze >150 papers, at about some 2000 pages of scientific material, that's going to happen. But I think I got the gist of it.

And the gist of it is that the literature on resistance training is a mountain of crapola shot through with the rarest vein of pure gold. Last year I harped on some of the unwholesome patterns I saw in the literature. This year I'm going to do it again, focusing on three recurring problems.

Power Donut Hole

Whenever you read a study that finds no difference between two groups, and therefore concludes that whatever intervention under scrutiny did not have an effect on some variable, ask yourself: was the study adequately powered to detect a difference? This is an easy question to answer if the authors have provided us with a sample size determination (sometimes called a “power analysis”). If they haven't, it's because (a) they didn't do one or (b) they don't want you to know that the study is inadequately powered. An inadequately powered study is subject to a Type II statistical error, or “error of the second kind.” This occurs when we fail to reject an incorrect null hypothesis. In other words, the investigation
fails to detect a difference when a difference actually exists. Since a fair amount of literature amounts to “we didn't see a difference,” this is a big deal. Sample size determination is a critical component of experimental design, especially when the effect size you're looking for is likely to be small.

The vast majority of the papers I looked at this year had no sample size determinations reported whatsoever. There were exceptions, like the paper by Paoloni et al:

“Sample size, calculated by assuming a 2±3-cm difference between the pre- and posttreatment values on a 10-cm visual analog scale (VAS), and a possible 10% withdrawal, indicated that 44 participants were required to have an alpha type I error of 0.01 and a beta type II error of 0.05.”

Actually, there are some problems with this particular example, but at least it’s there, and gives some justification for the way the authors designed their experiment. Sadly, that puts them a cut above most of their colleagues in the exercise science literature. I’m not a statistician, and I have to run to one of my stats gurus when it’s time to do my own power analyses and post-hoc testing, but I know enough to look for it in any clinical investigation, especially one that claims to show no difference between conditions. And so should you. For those of you interested in this topic, a quick search of the web will yield a ton of soporific reading, not to mention statistical calculators you can use to calculate the sample size for your own twisted experiments.

When we drill down on this issue of underpowered studies, we come back to the central lesion in the exercise science literature. I know I sound like a broken record, here, but most studies are small and short, and therefore incapable of detecting small-to-moderate effect sizes. Most studies of resistance training in the literature use fewer than twenty subjects. The typical study has 8 bros in the study group and 8 bros in the control. This means that if the number-needed-to-treat (NNT) is 15, there’s an excellent chance you’re going to miss the effect. An NNT of 15 means you need to treat 15 subjects to get one positive outcome. To put this in perspective, many drugs in current use have NNTs in the 20s, 30s or even the hundreds.

In addition to being too small, most studies are too short, at about 10 weeks long – an interval dominated by novice-level adaptations, and suspiciously similar to the length of an average semester or summer project. I’ll just leave that there.

The abundance of meta-analyses, systematic reviews and literature reviews chosen for this year’s survey is a clear indication of this problem. Since most studies are very small, academics, physicians, coaches and practitioners who want to know something about a particular question are better off reading or conducting such a pooled analysis instead of diving into yet another small, short, inadequately powered study.

“The Dose Makes the Poison.”

Once upon a time there was This Dude named Philippus Aureolus Theophrastus Bombastus von Hohenheim. Even 16th Century German people thought this was a little much, and he was called Paracelsus for short. Paracelsus was an alchemist, magician, botanist, philosopher, and physician. He stands at the threshold between the medieval and the modern medical mind, and he coined the phrase “the dose makes the poison.” In other words, a substance that is harmless in small amounts may have a mild benefit in larger doses, a toxic effect at a higher dose, and – whoops! – kill you dead at a still higher dose.

This early recognition of the dose-response relationship (which was not novel even in Paracelsus’ day) is still apparently beyond the ken of some strength science researchers. The literature strongly
indicates that exercise stimulates adaptation in a dose-response manner\textsuperscript{166,167,168,169,170}, and this most decidedly includes resistance training. A higher intensity-volume product will impose a greater adaptive stress than a lower one, and multijoint, full range-of-motion exercises will induce a greater systemic response than bicep curls or leg presses. This kind of dose-response relationship for exercise medicine is observed in the review by Chang et al, and the paper by Reed et al, which demonstrated that higher volume work produced more marked results on glucose tolerance than low dose exercise. Yet the literature is still dominated by low-dose, low-duration exercise medicine.

A related problem is the very poor description, in some studies, of exactly which form of the medicine was administered. In other words, the resistance training program is often poorly described. It is as if all resistance training were the same. It is not. There’s a huge difference between doing a Nautilus circuit and doing a barbell program with deep squats and heavy deads, every bit as real as the difference between using an NSAID and a narcotic. I can say I administered “resistance training” or “analgesia” in both cases, but that hardly tells the tale.

Oh, POO.

If you’ve read this whole article…you’re a nerd, and probably very lonely and isolated and pathetic. And you’ve probably already guessed what POO stands for: \textit{patient-oriented outcome}. For our purposes, we could say the P stands for Person or Practitioner or Player, or whatever you like. A POO is something that actually matters in real life, like strength acquisition, performance, pain, morbidity, mortality, cardiac events, quality-of-life, hypertrophy and the like. It is often the case that knowing what’s going on with blood lactate or CPK or mean torque or peak GRF is important to understanding the biology or mechanics of the effect you’re studying. But you can’t claim that one particular exercise prescription, supplement, program or nutritional regimen is superior to another until you’ve shown that it actually makes a difference in POO — that is, it has to make us healthier, happier and/or stronger. If you can’t show that, you may be doing good science, but you can’t overstate the practical implications for athletes, coaches and patients.

It’s important to know what’s important.

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