

Starting Strength

The Year in Strength Science 2013

by

Jonathon Sullivan MD, PhD, FACEP, SSC

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INTRODUCTION

This is the third of our annual Strength Science reviews, and will of necessity be shorter than last year's...or next year's. Although my traps caught more papers in 2013 than ever before (I'm getting good at this), I had to be more selective. My participation in the development of the Maintenance of Certification Process for the Starting Strength Coaches Association, assembly and startup of the SSCA Science Committee, the first run at SSCA Readings selection for 2014, the establishment of my commercial coaching practice for older adults, and other strength-related projects siphoned away time that I would have otherwise spent reading the vast number of (mostly silly) papers I found in the literature this year. I like to think these other endeavors were important. I make no apologies.

METHODOLOGY

Methodology for paper identification, selection and review was identical to that used for the [2012 review](#). Interested readers are referred to that article. Papers were organized for presentation along the same lines as last year, which is also the architecture of the SSCA library. Some of the categories and subcategories will be empty this year, and in future years, because we won't always find relevant or important literature in a particular category every year.

While we're on the subject of "methodology," I want to go ahead and discuss one of the most important papers to be published in the strength science literature this year, by Travis Beck [1]. It is in fact a methodology paper, entitled *The Importance of A Priori Sample Size Estimation in Strength and Conditioning Research*. As some of you may be aware, this is an issue I've harped on in previous science reviews and other venues. The strength science literature is overwhelmingly composed of small, short studies, and virtually none of them incorporate a sample size estimation, or "power analysis," to ensure us that the number of subjects studied was large enough for us to have confidence in the results, especially when those results appear to confirm the null hypothesis. I would like to think that this paper is a first step toward rectifying this critical defect in the strength science literature. It is a somewhat technical paper, with an overview of power theory and approaches to calculating effect sizes for different types of analysis (comparing means, regression analysis), with a look at the statistical calculations pertinent to these approaches. It also includes a tutorial on the use of [G*Power](#), a freeware statistical power analysis package. The paper should be read by every single investigator in the field, and considered a mandate for the proper planning and execution of exercise science studies. I don't find it reassuring that the author claims that sample size calculations are "required for all research studies submitted to the *Journal of Strength and Conditioning Research*," since the very same is packed with articles that document no such calculations. But, hey, this is a start.

A similar article, on the effect size statistic, is presented in the *Strength and Conditioning Journal*, and will be of interest to exercise scientists, professional coaches, and methodology nerds [2].

I. BIOMECHANICAL ANALYSIS

Biomechanics is simply the analysis of biological systems by mechanical methods. This category examines kinematic/kinetic studies, electromyography studies, and papers focused on what I call "biomechanical squatology." *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. *Electromyography* has been described in previous iterations of this review. Briefly, it measures the electrical activity in muscles as a surrogate marker of muscle activation and contraction. *Squatology* papers may have kinematic-kinetic, electromyographic, physiological or clinical relevance.

IA. Kinetics and kinematics

Once again, exercise simulation wizard Ognjen Arandjelovic [3] has given us a *tour-de-force* of mathematical modeling without any actual physical data. This time, he investigates whether "cheating" a dumbbell exercise by imparting "external momentum" to the load really undercuts the goal of supplying

a hypertrophic stimulus. For the purposes of this paper, Arandjelovic considers “external momentum” to be that supplied by a movement or muscle that is not the target of the exercise itself. The example used here is that of a lateral dumbbell raise, performed with or without external momentum supplied by trunk movement – in other words, leaning to one side to get the load moving. Arandjelovic begins with an anatomical analysis of the exercise that is, itself, worth the price of admission, making a very convincing case that the lateral dumbbell raise is best performed in the scapular plane; that is, at about 30-40 deg anterior to the coronal plane and leaning very slightly forward. He then proceeds to the derivation of a set of differential equations describing the movement, incorporating the masses of the load and the appendage, anthropometric values, the torque at the shoulder joint at various angles and velocities, and the rate of muscular fatigue.

These equations yield a family of solution curves, of which the torque-time curves are particularly interesting. In the initial set of curves, the trainee performs ten repetitions to failure, and the maximal humerothoracic angle, angular velocity and net muscular torque all fall from one rep to the next – no surprise here. When the model incorporates “cheating” – an initial angular momentum of 57.5 deg/s – Arandjelovic finds that the torque-time curves are of slightly less duration (the reps are a bit shorter) but otherwise little changed. In fact, at this particular starting momentum (the sweet spot), higher peak torques are produced for every rep but the first.

Arandjelovic takes this one step further, creating a model of total hypertrophic stimulus, based on the assumption that this stimulus is a function of both load and time under tension. The result indicates that an initial external momentum (“cheating”) maximizes the hypertrophic stimulus, by allowing more reps to be completed for any particular load, maximizing the time under tension.

This is a beautifully realized paper, as far as it goes, and it presents a challenge to the orthodox idea that the strict performance of an exercise is essential to the pursuit of Hugeness. We have recently seen the concept of external momentum imparted to the bar during curls (the hypertrophy-focused exercise *par excellence*) receive a charitable (and more qualitative) treatment by Rippetoe [4]. When considering the results of this paper, however, we should bear these caveats in mind:

1. Arandjelovic is quite explicit that this work is focused on the impact of external momentum on the *hypertrophic* stimulus of a movement, and not on performance, power or strength.
2. We are given no practical guidance in the application of these findings to our training. The 57.5 deg/s value, the sweet spot angular velocity yielded by Arandjelovic’ model, came out of his modeling of a *particular exercise performed by a particular theoretical lifter*. Slightly higher or lower angular velocities did not generate the same returns. Since most of us aren’t cyborgs who get retinal digital readouts from our integrated shoulder goniometers, the practical applications of these findings to hypertrophic training seem unclear. At best, the results suggest that adding a little “cheat” to a movement to get an extra rep and, therefore, more Hugeness, is no big sin. If that’s your Thang.
3. There is no actual clinical data here. *All of this happened in a Sim Gym*. Nobody actually took any living bros, made them do flyes with and without cheating, and looked to see who got more swole.

As Patsy said on the threshold of Camelot, “it’s only a model.” But it’s a damn beautiful one.

Luk et al [5] performed a comparison of muscle strength imbalance in competitive powerlifters and Division III field jumpers. Muscle strength imbalance is a concept much-beloved in The Industry, of particular relevance to physical therapists, certain subgenera of physical trainers, and exponents of the Functional Movement Screen. The claim is often made that limb strength asymmetry predisposes to injury, but on my reading this has not been convincingly demonstrated. In the present paper, the investigators looked at the forces produced by powerlifters and jumpers during single-leg jumps with either the dominant or non-dominant leg and with double-leg jumps. Raw data was used to calculate the Limb Symmetry Index: $LSI = 1 - (NDF/DF) \times 100$.

Where NDF = force produced by the nondominant limb and DF the force produced by the dominant limb. An LSI of 0 indicates complete limb strength symmetry. Although the study is small and its importance debatable, the data is very well presented and clearly indicates that (a) powerlifters produced more raw force than jumpers and (b) powerlifters had considerably less limb asymmetry than jumpers.

So, let's take those results and make the assumption that you're a track and field, fencing, boxing or figure skating coach, and that you're concerned about the potential adverse effect of limb strength asymmetry on injury rates and performance. You read this paper, which would seem to suggest that athletes who perform symmetrical strengthening exercises under the bar not only generate force more symmetrically, but also generate *more total force*. Some of us might say the implications for your training program seem clear.

Comfort's group [6] published a study of the power clean in "inexperienced" female collegiate athletes that purports to show no differences in peak power, rate of force development or peak vertical force between the hang clean, midhigh clean and full power clean. Sixteen collegiate female athletes performed variants of the clean and demonstrated no significant difference in the aforementioned variables at either 60, 70 or 80% of 1RM. Based on these findings, the authors suggest that inexperienced athletes perform different variations of the clean to "ensure all round development and technical competence in each variation of the exercise." They present no performance-relevant data to support this recommendation. Last year, Comfort's group published data showing that different loads in the clean pull actually resulted in *different* kinetics [7], including different peak powers and peak vertical force, and they have also published data showing that variants of the clean produce different kinetic outcomes [8]. Why, then, did the same kinetic variances fail to materialize in this experiment? The authors suggest that it's because the female athletes were inexperienced. But these ladies had all been performing cleans for *6-12 months*, and their average clean 1RM was about 115 lbs or 0.83 lbs/lb BW. I'm sorry, but the clean is just not that complicated – if you can't do a good clean after 6 months, you probably never will, and you probably won't be cleaning close to your body weight in an exercise science lab. So the data presented here seems a bit odd...or would, if we could be confident in the results. But the authors do not provide us with an explicit *a priori* sample size analysis (they state they had adequate statistical power, but don't tell us how or when they made this determination). So we cannot be assured that their small study had the power to detect a difference where one actually existed.

IB. Electromyography.

Three papers were selected in this subcategory. None of them are particularly great, but all three produced results that challenge the whole idea of performing resistance exercises on unstable surfaces, even if their authors didn't realize it.

Saeterbakken and Fimland [9] looked at the effect of having bros do bench presses on balance cushions, Swiss balls, and standard benches. They provide us with helpful exemplars of the exercises, with pictures of a shirtless (of course!) bro performing what appears to be a 95-lb bench on the three different surfaces. The study was small (n=16), with no power analysis and no long-term clinically relevant strength or performance outcomes. Their principal contribution to this body of literature was to adjust the relative load for each condition, noting that one's 6RM on a safe, stable bench is likely to be greater than one's 6RM while draped over a big stupid rubber ball. With this adjustment in place, the authors found that activation of the prime movers (pecs, triceps) was greatest on a stable bench. They found no relevant advantage of unstable surfaces for activation of the anterior deltoid, biceps, or – this is important – the external obliques or other trunk muscles. They conclude, correctly, that unstable surfaces offer no advantage for training of the prime movers in the bench. I think their data demonstrates no advantage for stabilizing or “core” muscles either.

A second paper by Saeterbakken and Fimland [10] conducted an EMG assay of *isometric* squatting – *squatting without moving* – on unstable surfaces. I won't belabor the methods or design, because, let's face it, the entire idea of trying to get a picture of squat muscle activation by attaching electrodes to a (shirtless!) bro who *isn't actually squatting* is just deeply, deeply silly. Suffice to say that the authors found that performing non-squatting squats on unstable surfaces resulted in similar EMG signatures and less force production than non-squat squatting on, you know, a *floor*. From this, they conclude that squatting on unstable surfaces may be beneficial in rehab and certain periodized training programs. Taking their data at face value, I arrive at exactly the opposite conclusion.

Similarly, Yongming et al [11] found that EMG activity of a number of limb muscles and the erector spinae increased with load during a deep squat, but was not affected by an unstable surface. The study was small and we have no way of knowing whether it was properly powered. The authors conclude that increasing muscle activation is best achieved by increasing the load, rather than by making the working surface more squishy. They make the remarkable and perhaps contentious observation that most sports and daily activities are performed not on marshmallows but on solid surfaces, and appeal to the principle of training specificity to argue that perhaps training should be conducted on similarly firm footing. Food for thought.

IC. Biomechanical squatology.

Squatology is the science of squats. It usually boils down to covering bros with stickers or electrodes and having them squat on a force plate, and then trying to convince everybody that the project resulted in useful data. Take, for example, an investigation of knee joint kinetics in the squat published by Cotter et al [12]. They note that deep squats continue to be controversial, with some “health professionals” indicting squats as bad for the knees. Of particular concern to the authors is the patellofemoral pain syndrome. The authors note that peak femoral joint reaction forces *may* lead to this syndrome by overloading cartilage – their first major assumption. They also note that peak external femoral knee moment *may* correlate with these reaction forces (and it's easier to measure, too). They therefore take the knee moment as a surrogate for reaction force – their second major assumption.

Just so we're clear on the Cast of Characters: The peak reaction force *may* be a surrogate marker for pathological stress on the knee, and the peak knee moment *may* be a surrogate marker for the reaction force.

So they measured the knee moment, by pasting stickers all over some bros and having them squat at various depths, and with various loads, while filming them behind a curtain in a setup that

is described in far more detail than is really necessary. The data was then crunchified in MATLAB to yield the peak external knee moments. The authors found that 1RM declined as squat depth increased – no surprise. At increasing depths and loads, external knee moments increased, and the authors note that these increases in knee moment overwhelmed the effect of decreasing barbell loading at increasing depths. In other words, if you squatted deeper, your load on the bar went down, which tended to decrease the moment on the knee. But the increased depth at that load still resulted in a higher net peak knee moment. Bottom line: squatting lower and heavier produced a greater external torque about the knee.

Since there's a better-than-even chance that Those People will seize on this paper as evidence that deep squats kill knees, it's important to point out a few caveats. First, the largest increases in knee moment were seen in the “below parallel” squat – which was actually an ass-to-grass squat. The “parallel” squat had only slightly higher knee moment than the above-parallel squat, and it was defined as a squat in which the hip crease fell just below the patella. The authors make a big deal of the finding that the greatest increases in peak moment were observed as one transitioned from unloaded squats (where the moments were practically identical) to the 50% 1RM condition, but the clinical relevance of these differences is not at all clear. At 85% 1RM (the highest loading studied) knee moments were again very similar between the above-parallel and “parallel” squats; the ATG squat demonstrated much higher knee moment.

The actual practical import of these findings, given the undemonstrated assumptions upon which the paper is based, are unclear. Even worse, the authors themselves are forced to concede that their assays were confounded by deformation of the markers on the subjects as they squatted.

Moreover, the authors admit that “cocontraction from the hamstrings and gastrocnemius with the quadriceps was not considered.” This is absolutely critical, because such cocontraction, especially of the hams, *would tend to balance the internal forces acting on knee structures without changing the external knee moment, the variable measured here.*

This paper will be waved around by orthopods and PTs who cleave to the mythos of deep squats as knee poison, but these shortcomings render the study almost unserviceable to either side of the argument. The authors use their results to recommend that squat programs should progress from above-parallel to deeper squats before addition of load, and then continue proceeding in the same higher-to-deeper fashion. This is a ridiculous prescription, all the more so because it cannot possibly be supported by the data presented here.

Pereira et al [13] present an EMG study of hip adductor activation as a function of external femoral rotation. They found that external femoral rotation – an action that necessitates hip abduction (“knees out!”) in the squat – promotes hip adductor activation. This does not strike me as a novel observation. The study is limited by squat depth, alas – squats were performed to 90 degrees of knee flexion. The authors recommend that squats should be performed at 30 degrees of external femoral rotation and “at least” 90 degrees of knee flexion. Strictly speaking, I guess I agree.

Esformes and Bampouras [14] present a study of the effect of back squat depth on jumping performance, said effect which they attribute to the mechanisms underlying *postactivation potentiation* (PAP). We've discussed PAP briefly in other reviews; it refers to the well-described phenomenon in which intense muscular activity can, after a brief period of recovery, enhance force development and rate of force development or power. At least three mechanisms are invoked for this phenomenon: enhanced calcium release from sarcoplasmic reticulum leading to increased myosin light chain phosphorylation, enhancement of motor unit recruitment, and optimization of fiber pennation angle. The authors recruited 27 rugby players and measured their countermovement jump and attendant

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biophysical parameters. Subjects were then counterbalance-randomized to perform either quarters squats or parallel squats for 3 reps at their 3RM. After a 5 minute recovery, the athletes again performed countermovement jump. All subjects visited the laboratory for testing twice after their initial testing/familiarization session. The authors found that both squat variants led to improved performance in the jump, but the parallel squat evoked superior improvements in jump height, impulse, flight time and peak power. You will note that the paper is purely clinical: no evaluation of the underlying mechanisms of PAP are undertaken, which is fine. I have quibbles with the authors' failure to regulate and/or document specific joint angles for the squat and the CMJ. Nevertheless, the data looks fairly solid, the effect sizes were moderate, and the implications for training seem clear and probably won't come as a surprise to most readers.

List et al [15] present a study of the kinematics of the trunk and lower extremities during the squat that entirely misses the point. Turns out that if you let your knees slide forward at the bottom of the squat, you don't have to bend over as much. The authors seem to think this is a great idea, because you'll avoid strain on your lower back, you won't bend your upper back so much, and you'll do a better job of strengthening your quads. Like I said: completely missing the point.

Last year, I presented the very important German study by Hartmann et al [16], which attacked and overwhelmed the concept of joint angle-specific training as an argument for quarter- or half-squats, and offered strong evidence that full squats were the preferred training modality for strength and performance. This year Hartmann's *blitzkrieg* continues. His literature review in *Sports Medicine* [17], one of the most important papers of the year, demolishes arguments that deep squatting is more injurious to passive structures – namely, the knees and spine. Hartmann's exhaustive examination of the literature leads to the following conclusions:

- Previous analyses of patellofemoral and tibiofemoral loading in squat variants (based extensively on study of cadavers) have overestimated the risk of full over partial squats by failing to take into account the “wrapping effect” of quadriceps tendon in the intercondylar notch at deep knee angles, which enhances load distribution and transfer in the deep squat.
- Deep squats enhance contact of articulating surfaces and decrease retropatellar compressive forces.
- These differences are made even more acute by the heavier loads lifted in partial squat variants.
- Half squats (80-100 deg) are particularly problematic, because the turning point corresponds to the greatest patellofemoral compressive forces and greatest compressive stresses concomitant with minimal tendofemoral support surface. In other words: more mechanical stress at the moment of least anatomical support.
- Greater forward movement of the knees (“knee slide”) corresponds to high knee shear forces. On the other hand, limitation of knee slide in the full squat implies a more acute back angle and more vertebral shear stress (a pretty clear trade-off).

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- The preponderance of evidence strongly indicates that strength training in the full range of motion results in functional adaptations of cartilage and meniscal tissue.
- *In vitro* experimental models used to support the contention that squats will damage menisci and lead to chondrocyte death are highly flawed, and at odds with observations of cartilage-cartilage interactions and investigations of joint loading in living persons.
- Mechanical analyses based on cadaver studies and measurement of tendon tensile properties indicate that deep squats produce a balance of posterior and anterior forces around the knee that is not observed in partial squat variants. In *properly performed* deep squats, neither anterior nor posterior shear forces will pass the threshold necessary to damage an intact anterior or posterior cruciate ligament.
- Tendinopathies of the patellar, patellofemoral and Achilles tendons have not been shown to have an increased incidence in weightlifters or to be attributable to any squat variant.
- Spinal structures are subject to adaptive responses, including increased bone mineral density in spinal vertebral end plates and salutary modifications in intervertebral discs. No epidemiological data links weightlifting to an increased incidence of *clinically* relevant back pathology when compared either to other athletes or the general public – in fact, there are indications that strength training is protective in this regard.

This is a *devastating* analysis. Once again, Hartmann has shifted the burden of proof. Nevertheless, the paper is not ironclad. The quantity and quality of epidemiologic data that Hartmann can draw upon is not great (although that's sauce for the goose, I guess). And the paper relies heavily on cadaver studies and biomechanical analyses, rather than *in vivo* measurement of joint forces. Minimally invasive technology for such measurements may soon be at hand (bionanosensors will change everything), but for now I believe that full squats are in possession of the field, and digging in. Partial squat advocates are in disarray. And that, I guess, will do for the military analogies.

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.

IIA. Muscle physiology

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

Fiber-type shifting is a critical area of investigation in exercise myophysiology, and the basic principles were reviewed extensively last year. Nilwik et al [18] present data suggesting that the decline in muscle mass with aging is almost entirely due to a reduction in Type II muscle fiber size – but not number. This is a potentially important result, at least from an academic perspective. The authors took 25 healthy bros and 25 healthy geezers and determined the volume of their quads using computed

tomography (CT). Muscle biopsies from the vastus lateralis were used for fiber typing. The authors found that geezer quads were smaller than bro quads, and that the difference in size could be attributed almost entirely to the smaller size of geezer Type II fibers (Type I fibers also tended to be smaller in the older group). Geezers then underwent a 6 month RT program with the usual machine-based exercises at 3x/week. Their quads got swole, and the investigators report that the concomitant increase in vastus Type II fiber size completely accounted for this change. The finding of decreased fiber size, not number, is at odds with those of other studies, but it's a happy thought, because making atrophic Type II fibers larger with training is a rather more tractable prospect than replacing lost fibers...which probably doesn't happen to any clinically relevant degree. A potential problem with this paper is that the investigators used the volume of the entire quadriceps as one of their outcome measures, but only took biopsies from the vastus and extrapolated type II fiber populations from this muscle to the entire group.

In a related study, Verdijk [19] et al looked at satellite cells in human muscle in 165 subjects ranging in age from < 18 to 86 years old. Muscle biopsies were assayed for fiber-type distribution and the presence of satellite cells, which are a type of stem cell specific to muscle tissue. Upon an appropriate stimulus, these stem cells can proliferate and generate new muscle cell nuclei, critical for the process of muscle cell hypertrophy. The authors found that as the age of subjects increased, they demonstrated massive decreases in muscle cell size without major changes in satellite cell number, and very little in the way of fiber-type shifting. As in the Nilwik, older subjects demonstrated Type II fiber atrophy. This was accompanied by a decrease in Type II fiber-associated satellite cells. Twelve weeks of strength training increased Type II fiber muscle size and satellite cell content. These two important papers, taken together, underscore the power of resistance exercise in older populations, the relative susceptibility of Type II fibers (the strong/powerful fibers) to aging and atrophy, and the responsiveness of these fibers to even very brief periods of strength training.

IIB. Endocrine, metabolic, and cell signaling processes

This subcategory includes papers investigating endocrine, 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 should have some familiarity with the field.

IIB(1): Cell Signaling – General. Myophysiology geeks will want to get their hands on the state-of-the-art literature review by Egan and Zierath [20], *Exercise metabolism and the molecular regulation of skeletal muscle adaptation*, published in the February 2013 issue of *Cell Metabolism*. The paper covers too much ground to be neatly summarized here. This 22-page, in-depth review of the molecular and genetic mechanisms of adaptation to training is a primer on current thinking about the cellular physiology of muscle. After consideration of muscle bioenergetics and fiber-type-specific substrate utilization, the authors turn their attention to recent findings in *excitation-transcription coupling* – in other words, the pathways that lead from muscle fiber contraction to adaptive gene expression. This discussion encompasses current thinking on calcium-mediated signaling, mechanoreceptor and MAPK signaling, and the role of redox potential, NAD⁺/NADH ratio and sirtuin signaling in promoting the appropriate gene response to training. Their examination of the differential role of mTOR isoforms and Akt has led me to other readings that have caused me to refine (although not substantially change) my own views on these central signaling elements. Regulation of skeletal muscle protein synthesis and

degradation and the role of satellite cells are also discussed. The section on skeletal muscle as a secretory organ and the role of *myokines*, or muscle-derived cytokines (signaling molecules) is particularly exciting, and points to a wide-open area of basic science and exercise physiology research. This paper, with its 200 or so references, could serve as a syllabus for a graduate-level course in the cellular biology of exercise, and deserves a preferred position in your literature files. A must read.

Mitchell et al [21] continue the attack on a rapidly eroding model of muscle hypertrophy in which circulating trophic factors stimulate muscle growth. Like other investigators, they found little correlation between circulating hormones (testosterone, IGF-1) and muscle protein accretion. In line with a growing consensus, they conclude that muscle responses to training are dominated by paracrine and autocrine effects – within the tissue itself, rather than from factors released into the circulation.

I think that's probably right, although this paper doesn't prove it. But I also think it glosses over an important set of questions. Circulating trophic factors may very well not be directly responsible for muscle protein accretion after exercise. *But circulating levels of those factors are nevertheless increased by resistance exercise.* Why? And to what purpose? When we work out, we are increasing the androgen receptor content and trophic peptide content of muscle tissue, and the trophic signaling within muscle

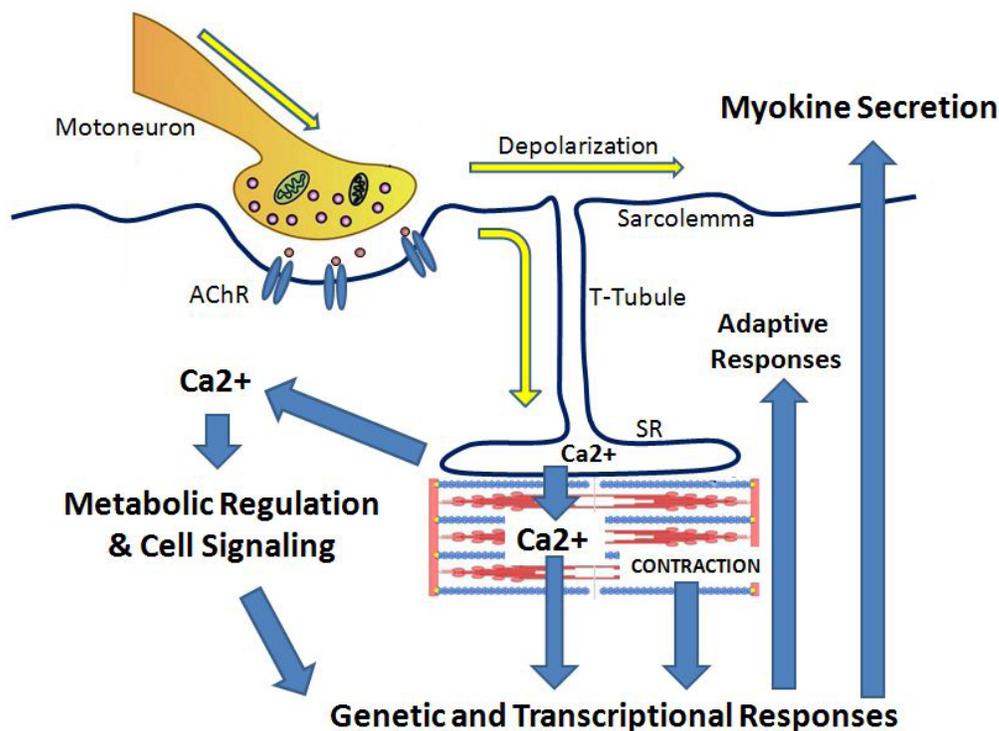


Figure 1. Excitation and contraction influence the muscle both as a contractile and secretory organ. Yellow arrows indicate depolarization of membranes. Depolarization (“firing”) of the motor neuron results in the release of acetylcholine (ACh) at the neuromuscular junction. Binding of ACh to its receptors induces depolarization of the muscle membrane (sarcolemma). This depolarization (action potential) spreads across the muscle cell and down the t-tubule, resulting in depolarization of the sarcoplasmic reticulum (SR). The SR consequently releases calcium into the cytoplasm (sarcoplasm), causing actomyosin binding and sarcomere contraction. Calcium release and the contraction itself appear to activate cell signaling processes, some of which culminate in genetic responses. Genetic and transcriptional responses modulate the cell’s adaptation to the contractile stimulus and mediate the release of muscle-derived hormones or cytokines (myokines). These myokines may have profound systemic effects on the organism. [Image prepared by the author using elements from Wikipedia in accordance with Creative Commons Distribution License.]

cells (through GF receptors, Akt, mTOR, p70S6K, etc) in a *paracrine* (autoendocrine) fashion. But we are also spilling IGF-1, BDNF, IL-6, testosterone, GH, and other factors *into the circulation*. I have to believe this has an effect, and if that effect is not to increase muscle protein accretion...then what is it? As it happens, [I have some ideas](#) about that.

IIB(2). Transcriptional response to resistance exercise is an important if not yet practically applicable area of investigation. Agergaard et al [22] investigated the response of a rather large repertoire of mRNA transcripts to both heavy and light resistance loading, and the effect of feeding on this transcriptional response.

As you will recall from your high school biology, *transcription* refers to the production of messenger RNA (mRNA). This RNA message is transcribed from chromosomal DNA to RNA in the nucleus, in response to some stimulus or signal. It's called "transcription" because the information in both the DNA and RNA is in the same "language" – the language of nucleotide base pairs. The mRNA is then transported to the cytoplasm, where amazing nano-robots called ribosomes translate the message from the nucleotide language into the language of amino acid sequences. *Translation* in cellular biology is roughly synonymous with the process of protein synthesis.

In this study, the authors took sedentary bros, biopsied their muscles, and examined mRNA responses for heavy and light training, both in the short term and after 12 weeks of training, and with

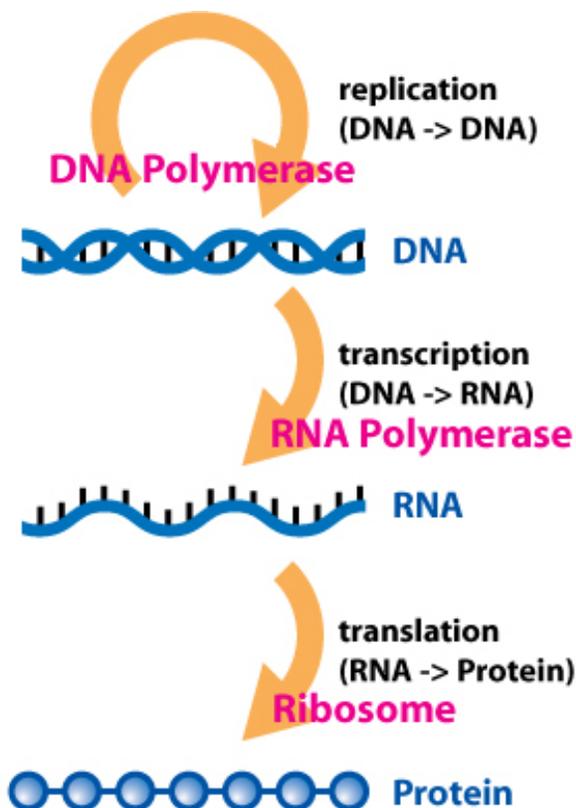


Figure 2. The flow of information in living systems. In the specific case of protein synthesis, information is transcribed from DNA to mRNA, which is then translated into protein. [Image reproduced from Wikimedia under Creative Commons attribution license.]

and without peri-workout feeding. Heavy and light muscle contractions were verified with and correlated by EMG recordings. I won't dwell on the methods – the paper is highly technical, its practical relevance is not yet clear, and its results don't seem to me to particularly controversial, in part because this field of investigation is still relatively new. The authors found that heavy loading produced a greater expression of mRNA for myogenic proteins than light loading. Myostatin mRNA, as we have seen previously, was suppressed by resistance exercise. The authors found no differences in response to heavy vs. light training between the feeding and fasting conditions, and they found no changes in IGF-1 or atrophy-related gene transcripts.

Although the finding that heavy training induces a greater myogenic transcriptional response than light training probably doesn't strike you as earth-shattering, this paper is important for its exploration of the transcriptional response to training and the role of intensity in modulating that response. It contributes to the growing body of research into just exactly *how* mechanical muscle work signals for an adaptive response at the molecular and genetic levels. The important caveat to keep in mind when reading a paper like

this, especially with regard to the feeding and IGF-1/atrogin results, is that a *transcriptional* response does not necessarily correlate to actual *translational* or *phenotypic* response. These guys measured mRNA, which codes for protein – not the proteins themselves.

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 (see [Stopping the Spread of Misinflammation](#)).

Trappe and Liu contributed a very important review of the biology and pharmacology of prostaglandins, inflammation, and anti-inflammatory therapies; it is reviewed under Sports Medicine.

IIB(4). Metabolism and Bioenergetics. Abboud et al [23] present a study of excess post-exercise oxygen consumption (EPOC) after resistance training. A number of recent investigations make it clear that EPOC – a measure of energy utilization – is elevated for up to 48 hours after a resistance training bout in both trained and untrained individuals. Abboud's paper, a very small study with an unconvincing power analysis, reports no elevation in resting metabolic rate (RMR) or energy utilization for up to 36 hours after either a 10,000 kg load-volume set or a 20,000 kg load-volume set in eight trained young men. In point of fact, the authors did observe an increase in evening resting metabolic rate and energy expenditure, which by my quick back-of-the-envelope calculations comes out to an increased energy expenditure of several thousand kcal over a 4-day period...but it wasn't *statistically significant* (bearing in mind that the study population consisted of *eight* bros). The authors conclude that, while resistance training may certainly increase energy utilization in untrained individuals (and thereby promote fat loss), it will not do so in trained individuals, who are adapted to resistance training. However, it is *exactly this speculation* on the part of the authors that raises the question of whether these trained individuals have a higher resting metabolic rate *to begin with*. No comparison of the RMR of these bros with untrained bros was undertaken, and it is critical to note that the baseline metabolic rate assay was taken as little as 4 days after an initial 1RM testing session. I think the authors may well be right that trained bros see a lower bump in their EPOC than untrained bros, but I don't think this paper proves it. A similar paper by Kelley et al [24], with similar limitations, reached similar conclusions. For an alternative view, the reader is referred to a short review by Koziris, focused particularly on the results of recent work done by Hazell et al documenting prolonged EPOC after interval sprint training [25].

Shing et al [26] investigated another potential mechanism for fat loss in the setting of high-intensity interval training (HIIT). They found that bros who engaged in a HIIT protocol lost more fat and mounted larger increases in adiponectin, a protein hormone from fat (*adipokine*) that regulates energy metabolism, insulin sensitivity and fatty acid breakdown. This is intriguing, but the magnitude of the differences reported here is dubious (a classic example of statistical significance with doubtful biological significance).

IIC. Cardiovascular physiology and hemodynamics

Utomi et al [27] address the perennial question of differences in cardiac adaptation in resistance-trained and endurance-trained athletes. The fundamental concept here is that of the athletic heart, characterized by non-pathological increases in mass, wall thickness and function. The prevailing and oft-cited view is that endurance athletes demonstrate *eccentric hypertrophy* (balanced increases in chamber and wall dimensions), while resistance athletes demonstrate *concentric hypertrophy* (increases

in wall thickness with disproportionately less increase in chamber volume). The readers of this series will not be surprised to learn that the data on this topic is inconsistent and lacking in rigor.

The authors conducted a systematic review and meta-analysis of the existing literature, using a very explicit and well-described approach that yielded 92 studies of the adult male athletic heart, conducted with either echocardiography or MRI. Pooling this data and subjecting it to analysis, they found that both endurance and resistance athletes demonstrate similar cardiac adaptations compared to sedentary controls. Left ventricular mass, interventricular septal wall thickness, posterior wall thickness and left ventricular end-diastolic diameter were all increased in both types of athletes but not in controls. Only LV end diastolic volume and LV stroke volume (functional, not anatomic parameters) were different between endurance (higher) and resistance (lower) athletes. A training-specific pattern of classic concentric hypertrophy was not observed in resistance-trained athletes. There was no difference in ejection fraction – perhaps the most important hemodynamic variable of all – between any of the groups. This paper is hampered by the high heterogeneity of its source data, a relative paucity of data on resistance-trained athletes compared with endurance athletes, and its (necessarily) narrow selection criteria. It is nevertheless a serious challenge to the conventional wisdom of differential cardiac adaptation. The take-home message is that both RT and ET result in increased cardiac mass and wall thickness, while ET has slightly greater implications for diastolic (as opposed to systolic) function...which makes sense if you think about it. So think about it.

Three papers addressing the Valsalva maneuver deserve your attention. Adams et al [28] were primarily interested in standard recommendations for post-sternotomy patients, so-called “sternal precautions,” which are meant to prevent dehiscence and other complications. These recommendations are quite conservative (“don’t lift more than five pounds”) but are based on little data.

The authors recruited some sixty bros. They excluded those who couldn’t tolerate having an esophageal pressure transducer installed. This procedure involves going Up Your Nose With a Rubber Hose, and, yes, it’s noxious. In med school, we were required to have a nasogastric tube inserted to “see what it’s like.” I did not complete this requirement, almost got arrested for assault, and went on to a career in which I ordered far fewer nasogastric tubes than my colleagues. Eleven of the bros recruited for this study couldn’t take it: they rang the bell, and washed out.

The Elite Nose Bros (n=49) then performed bench presses with and without Valsalva at various percentages of 1RM while their thoracic pressures were measured, and this was compared to thoracic pressures obtained when sneezing was induced (perhaps by yanking on the tubes hanging out of their nostrils). The authors found that bench presses performed at low or moderate intensity with breathing generated pressures less than or roughly equal to those generated by a sneeze. This has implications for post-sternotomy recommendations, of course, but you don’t care about that. What’s interesting to us in the strength community is that the pressures generated by a *moderate intensity bench press with Valsalva was essentially no different from the thoracic pressure generated by a sneeze*. The highest bench press-Valsalva recorded (153.9 kg) was roughly twice that of the highest sneeze pressure recorded (83 kg). The thoracic pressures generated by lifting under Valsalva just aren’t that much higher, if at all, than those generated by a sneeze, a cough, *le petit mort*, or straining against a stubborn turd.

Hackett and Chow present us with a systematic review of the literature on the Valsalva maneuver [29]. Actually, this is two systematic reviews: one on the effect of Valsalva on intra-abdominal pressure (IAP), which the authors implicitly take to be a surrogate marker for spinal stability; and one on the safety of Valsalva in resistance training. I won’t belabor the analysis of this paper, because I dealt with this topic exhaustively in a 2013 article [30], and the timely appearance of Hackett’s paper was an important source of information for my own (non-systematic) rantings on the topic. The

authors find that the literature does indeed support the position that Valsalva increases IAP in a way which could be expected to support spinal stability, and also that the health risks of the Valsalva remain undemonstrated. For me, however, one of the most important findings of this paper is that the literature strongly indicates that the Valsalva is a reflexive and virtually unavoidable response to lifting loads at high intensity. And this underscores a point I made in my own review: *prohibition of the Valsalva is tantamount to a prohibition of heavy lifting itself.*

IID. Aging and atrophy

Luo et al [31] present data in the rat supporting pro-autophagic and anti-apoptotic effects of chronic resistance training on aging muscle. **Autophagy** is a process in which dysfunctional or damaged proteins are tagged, bagged and disposed of by the cell. This process is an important first step in hypertrophy and cellular remodeling (the “demo,” as it were), and is known to be suppressed in aging and atrophic tissues. The investigators report increased levels of pro-autophagic signaling molecules in the muscle of aging resistance-trained rats, although their raw Western blot data looks a bit underwhelming to me. **Apoptosis**, as I’ve [discussed elsewhere](#), is a regulated form of “cell suicide.” The intrinsic form of apoptosis involves the leak of cytochrome *c* from mitochondria and the activation of **caspase** executioner molecules. The investigators report decreased cytochrome *c* leak and caspase activation in their resistance-trained geezer rats. Much as I would like to believe it, this data looks only slightly more convincing to me than the autophagy data. The data on myocyte apoptosis is cleaner. The authors used a standard TUNEL assay of muscle tissue and found far fewer apoptotic myocytes in resistance trained rats. They showed *increased* IGF-1 signaling, but *decreased* Akt and mTOR signaling – a bit of a conundrum. They try to make sense of this latter finding with some hand-waving, but I think it simply comes down to timing: muscle tissue was harvested 2 days after the last training bout, at a time when Akt/mTor-driven muscle protein accretion might be leveling off and autophagy might be expected to be ramping up again. Such a model would correlate with the general adaptation syndrome in a way I leave my more geekish and nerdy readers to work out on their own as a salutary exercise. This paper, with all its limitations, is a data point in favor of the view that *resistance training down-regulates muscle apoptosis.*

Two related papers by Nilwik and Verdijk, describing the responsiveness of aging muscle to training interventions, are discussed above in the section on fiber-type shifting.

III. BIG MEDICINE

In this section, we will examine papers published in 2013 addressing the impact of resistance training on health and various disease states.

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 and heart failure selected for review. A minor but

amusing academic brouhaha over the use of the Borg scale in patients with coronary artery disease is discussed later, in section VA (**Metrics**).

IIIA(1). Hypertension (“high blood pressure”) is a major public health problem, with untoward implications for every major organ system. The literature on the use of resistance training for hypertension continues to grow...and continues to be of low to middling quality.

This becomes especially apparent when one conducts systematic reviews or meta-analyses of this literature, as was done by Rossi et al in the *Canadian Journal of Cardiology* [32]. This study was conducted as part of the ongoing evolution of the Canadian Hypertension Education Program, which had previously included recommendations only for endurance exercise as an intervention for high blood pressure. The authors used strict inclusion criteria for their meta-analysis: only randomized controlled trials (RCTs) investigating blood pressure as a primary outcome, using RT-only intervention arms, were accepted, and subjected to data extraction and risk of bias assessment.

Only nine studies met these inclusion criteria, encompassing a grand total of 452 subjects – smaller than most RCTs for pharmacological interventions. Analysis of the pooled data indicated that RT lowered diastolic, but not systolic blood pressure, and not by much. There was no evidence that RT had any adverse effect, including raising blood pressure. On balance, the authors state there is insufficient evidence to recommend RT as a specific therapy for hypertension. But they are also at pains to point out the profound limitations of this literature. The small number of total subjects for pooled data analysis strongly suggests that it lacks the power to make robust conclusions. More importantly, all of the studies incorporated in the meta-analysis were – you guessed it – small and short. The median intervention duration was 16 weeks, and no study lasted longer than 24 weeks. Training approaches were all over the map, and poorly described. No long-term outcomes. And so on. A single, large, properly powered, long-term RCT using high-dose resistance training would answer this question. Why the hell doesn't somebody just *do* it?

IIIA(2). Congestive heart failure is one of the many unfortunate end-points of uncontrolled hypertension. As we've noted previously, it is a sonafabitch, which hasn't prevented it from becoming an increasingly popular way to go to a miserable death in Western societies. We've discussed papers on the safety and utility of physical training in CHF patients, and the literature emerging on this topic suggests that, while it may not reverse cardiac dysfunction, it is relatively safe and can improve exercise tolerance in this population.

Piepoli and Crisafulli [33] present a review article that gives us a tantalizing glimpse of how resistance training *might* slow the progression of heart failure, and also underscores a much larger and even more important physiological concept. The article addresses the so-called ***muscle metaboreflex*** model of hemodynamic modulation in healthy exercise, and how its derangement in the heart failure patient may help drive the progression of the disease.

In this model, muscle contraction during exercise results in the accumulation of metabolites such as lactate, adenosine, cations, inorganic phosphate, and a number of myogenic signaling peptides (myokines again!). These substances are proposed to interact with ***muscle metaboreceptors***, where are specialized nerve endings that in turn contribute to regulation of hemodynamic parameters. In healthy subjects, this metaboreflex optimizes hemodynamic function for exercise, leading to appropriate increases in heart rate, venous return, contractility and stroke volume (increasing cardiac output) and systemic vascular resistance. Increased systemic vascular resistance and increased cardiac output both contribute to an increase in blood pressure. This is particularly important in the face of extensive

exercise-induced vasodilation, which flushes muscle with blood but tends to decrease overall vascular resistance.

On this view, the increase in blood pressure during exercise is due primarily to an increase in cardiac output. In heart failure patients, however, the ability of the heart to respond to the metaboreflex by squeezing harder and faster is fundamentally impaired. So while both healthy and heart failure subjects increase blood pressure in response to exercise, in heart failure patients the primary mechanism – the only one really available to them – is to clamp down their vascular tree and increase systemic vascular resistance. This vasoconstriction, unlike the situation in healthy subjects, extends to the muscle tissue beds, exacerbating muscle ischemia and leading to profound exercise intolerance. (When I say “exercise intolerance,” I mean getting winded walking out to the mailbox and back.) And this increase in systemic vascular resistance increases the cardiac afterload and the overall sympathetic tone – both of which are problematic for the failing heart, and can be expected to accelerate the progression of the disease.

This model invokes a vicious cycle of decreased exercise tolerance, further derangement of the muscle metaboreflex, more sympathetic activation, more vasoconstriction, more strain on the failing heart, and more badness in general. Next thing you know, your legs are the size of Long Island and you're drowning in your own fluids.

Although there is a growing body of literature supporting the use of resistance training in CHF patients, there is precious little data on how such training impacts *this mechanism*, which remains putative and a little controversial. But it's not a stretch to postulate that an increase in exercise tolerance would moderate muscle metaboreflex changes in CHF, cooling down sympathetic overactivation and reducing afterload. What are the *actual* effects on heart function? We don't have enough data to tell. From a research perspective, this is potentially a mother lode waiting to be mined.

Perhaps more importantly, it underscores an important emerging concept I alluded to earlier: the idea that *muscle tissue is an endocrine organ*. It is, in fact, a **gland**, an organ that participates in physiological regulation by releasing signaling molecules with profound tissue-level and systemic effects. And like any gland, it can get sick. Sick, weak, atrophic muscle is like a sick, weak, atrophic thyroid, pituitary or adrenal. The consequences are potentially devastating.

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. In future reviews, strength-related studies of thyroid, adrenal, pituitary, pancreatic, and some hepatic and renal diseases will fall into this category.

The Exercise and Sports Science Australia (ESSA) position statement on exercise and chronic kidney disease [34] highlights findings we've seen in previous reviews: patients with CKD appear to benefit from resistance training. The authors are nevertheless at pains to point out, as we have, that this literature is sketchy. They also recommend that patients with CKD who are given exercise prescriptions should be trained by “qualified personnel.” This actually seems prudent, but one wonders just what qualifications those personnel should have, since to my knowledge *that* specific question has never been addressed in the literature with patient-centered outcomes.

2013 saw more papers on the role of exercise medicine in the treatment or prevention of insulin resistance, metabolic syndrome and diabetes. Unless and until a very large, well-done longitudinal study with definitive clinical outcome measures comes along to tell us that training doesn't help, I tend

to consider that old news, so I'll just point out two representative papers for interested readers. The first is an original investigation by Croymans et al [35], who observed an increase in insulin sensitivity in resistance-trained obese young men. The second is a non-systematic review of the evidence by Mann et al [36]. Their survey of the literature found that the preponderance of the evidence shows any form of exercise improves metabolic control and insulin sensitivity, but that a combination of resistance training and conditioning seems to have the most powerful effect.

This literature on resistance training and metabolic regulation is dominated by small, short studies and low-dose exercise medicine, and *still* manages to show a profound effect on what is probably the single most important health derangement in industrialized countries: the *metabolic syndrome*. It is now abundantly clear that this constellation of visceral fat accretion, insulin resistance, hyperlipidemia, and hypertension is a central player in the development of the pathologic aging phenotype that is taking over the modern world. That phenotype comes to full bloom with coronary artery disease, heart failure, diabetes, morbid obesity, sarcopenia, dynapenia, stroke, dementia, depression and frailty. *One simple, powerful medicine fights all this misery.*

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.

This year my nets picked up more relevant papers on this topic than previously, and the news is all good. Two papers confirm that muscle tissue is responsive to training in the setting of COPD. Theriault et al [37] presented an abstract with data indicating that COPD patients have similar numbers of satellite cells (muscle “stem cells”) as healthy controls, and that a bout of resistance training induces a similar increase myonuclei in both groups. Constantin et al [38] showed that both COPD patients and healthy controls mounted adaptive responses, although anabolic and transcription factor responses were blunted in the COPD group (possibly an effect of the significantly lower dose of exercise medicine administered to this group). The authors make the astonishing claim that increased protein and carbohydrate intake is not a prerequisite for normal training response in COPD, a conclusion at odds with what everybody in the observable universe knows about training, and which cannot possibly be supported by their data.

Benton and Wagner [39] provide us with convincing evidence that single-set resistance training improved upper body strength and function in COPD patients also undergoing pulmonary rehabilitation...but not quality of life. To my mind, this casts considerable doubt on assays of quality of life.

Finally, Strasser et al [40] examined the effect of resistance training on pulmonary function with a systematic review and meta-analysis of fourteen randomized controlled trials. Pooled data from these studies indicates that resistance training improves forced vital capacity and minute ventilation, but not forced expiratory volume. Given the profound structural changes that produce pulmonary

dysfunction in COPD, these results are more positive than I would have anticipated. Confirmatory studies are clearly indicated.

IIID. Neurologic, Neuromuscular and Myopathic Disorders.

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. An updated review by Voet et al [41] concludes that tailored, careful resistance training is safe in most such patients, but many clinicians and some researchers are still concerned about the consequences of “overuse.” In that light, the 24-week randomized controlled study of 30 boys with Duchenne muscular dystrophy, subtitled “No Use is Disuse” would seem to be of interest. The authors used bicycle training to increase strength and endurance in the extremities, and found no evidence of deterioration with exercise therapy. Indeed, boys treated with exercise therapy demonstrated stabilization of the Motor Function Movement assay, whereas boys in the control group manifested minor declines, consistent with the progressive nature of this horrific disease. The authors conclude that this kind of therapy is safe, although eventual decline is inevitable given the currently available medical interventions. This seems encouraging from a palliative perspective, but as another systematic review by Gianola et al [42] observes, the existing literature on this topic is dominated by small studies, uncertain results, and a lack of large, meaningful RCTs with clinically relevant end points. A large randomized trial controlling for extent of disability, total work, intensity-volume products and other confounders, with rigorous outcome metrics, and probably involving concentric-only exercise, is needed before doctors can make informed recommendations about resistance training in this population.

IIIE. Psychosocial Health and Disease.

In a study that is bound to get some attention, Keating et al [43] found an *association* between strength training and *self-reported* academic performance in University students. This would be something to crow about if the data weren't so limited and if we knew the causal direction, if any, of this association. Were these students smarter because they trained? Did they train because they're smarter? Or was there no relationship, other than a statistical one, between the two variables? We don't know. This is a hypothesis generator, nothing more.

IIIF. Obstetrics and Gynecology

Hamad et al published a methodological paper addressing the measurement of intra-abdominal pressure (IAP) [44]. They used a wireless intravaginal transducer in 13 women, and then tied themselves in knots trying to find the best approach to getting meaningful data from...this approach. (The first part of the paper's title is “More Complicated Than It Looks...”). The take-home point for those of us who aren't keen to start measuring our client's intravaginal pressures on our iPhone is that the measurement of this clinically relevant parameter is still in its infancy...which hasn't stopped any number of authorities, on the basis of virtually no evidence, from proscribing certain activities (such as lifting) in women with pelvic floor disease. Since doing your squats is likely to improve rather than worsen pelvic floor strength, we have to wish Dr. Hamad and her team the best of luck.

IIIG. Geriatrics

In a well-intentioned but ultimately useless Norwegian study, Lohne-Seiler et al compared traditional training with machines to “functional training,” which involved lifting boxes, running up stairs with backpacks, standing up from chairs, obstacle courses and the like [45]. They report no difference in any outcome of interest between the two training regimens, and then deliver a protracted confession about how their study could have been better. It completely misses the point. The real problems with the study are that it was small and short, and it compared resistance exercises that can be programmed and progressively overloaded to functional exercises that cannot be so programmed. The only important thing about this study is buried in the discussion, where the authors (while remaining oblivious to the real implications) remind us of literature suggesting that power development, not just raw strength, is important in older adults. *Athletes need to do their power cleans or their dynamic effort sets.* There is no athlete for whom the stakes are higher than the aging athlete, and no “sport” more demanding than aging well. It’s not for sissies.

A study by Farinatti et al, looking at the role of training frequency on 10RM and functional strength in older women, is briefly discussed in the Programming section.

McCrory et al [46] published a comparison of bone mineral density (BMD) in senior competitive athletes and healthy controls in which they report *no difference* in BMD between the two groups. This result isn’t as interesting or as contrary as it might seem at first blush. This was a cross-sectional, observational study, not an intervention trial – which means that it did not and could not evaluate the effect of exercise on the BMD of any individual. The athletes chosen for study competed in running, swimming and cycling, not strength sports. The controls against which they were matched were healthy and active, and the authors did not control for the activity level of this group. In fact, the authors concede that the lifetime physical activity of the study participants was unknown, and the questionnaires used for collection of data on physical activity appear to have been improperly administered. The regression modeling used by the authors gave primacy to lifestyle factors other than exercise. And this passage from the Discussion speaks for itself:

After the completion of the study, we realized that our activity surveys were incomplete, particularly in regard to resistance training. Therefore, we cannot truly quantify the amount of resistance training done by the subjects. We accept that this is a major limitation of the study as resistance training can have a large influence on BMD.

Whoops. To atone for their oversight, the authors performed a *post-hoc* analysis of variance of BMD comparing athletes who self-reported use of RT and those who didn’t. They found no difference. This sort of “data dredging” after the fact cannot be taken seriously. A robust finding that active senior populations do not have higher BMD would be important and interesting, and would be at odds with a great deal of published data – including data previously published by these authors. The current paper cannot, however, be categorized as “robust.”

IIII. Other

Pederson et al [47] showed that a progressive RT program decreased musculoskeletal pain in female office workers in a dose-dependent manner. They used silly little dumbbell exercises and the changes in pain index they report aren’t exactly mind-blowing, but the paper does remind us of an increasing body of literature revealing that exercise medicine demonstrates a dose-response relationship.

Murlasita and Mohammad [48] present a nice overview of a problem that should be on the minds of all serious strength coaches and older athletes: the potential for adverse interaction between statin medications and training. Statins are among the most widely prescribed drugs in the world. They work by inhibiting an enzyme called HMG-CoA reductase, which catalyzes the rate-limiting step in the synthesis of cholesterol. By lowering serum cholesterol levels, statins are supposed to lower the risk of cardiovascular disease. Careful evaluation of the available evidence casts [serious doubt on the effectiveness](#) of widespread statin use. Setting that aside, the present fact is that many of our middle-aged and older clients are on statins. And that's a problem, because there's mounting evidence that statin-induced myopathy is more prevalent than previously thought, that it is more common in the aged and individuals engaged in strenuous exercise, and that it can on occasion lead to serious consequences, including full-blown rhabdomyolysis, acute tubular necrosis and renal failure. It is therefore imperative for coaches (and their clients) to understand what is known about statin myopathy and be able to recognize it. There is some suggestion that initiating statin therapy after exercise has begun may be protective, and that supplementation with CoQ10 and vitamin D may also be protective. But they are just that – suggestions, hardly proven in human populations. This is an arena where the impulse of the trainer and the client to increase strength and function may be at odds with the physician's desire to optimize a laboratory value. Which is more important for the patient's long-term health?

IV. PROGRAMMING

This category brings in papers that address issues of programming, periodization, exercise selection and set design.

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.

James Steele (not that one) penned an impassioned editorial on the use of the word *intensity* in the literature on resistance training [49], arguing that the word should be abandoned or at least defined in any study in which it is used. A pivotal point in his argument is the Oxford definition of the term, which I find curious. Many scientific disciplines use terms in a way that does not comport with their use in everyday language games [50], or even other scientific disciplines [51]. While I agree that it is important for us to use language carefully in science, it seems to me Steele is harping on an issue that is the *least* of the problems confronted by exercise science. I am not convinced that the term *intensity* is the subject or source of widespread confusion. Reading this paper put me in mind of the American Heart Association's decision, some years back, to change the name of a lethal arrhythmia from *electromechanical dissociation* to *pulseless electrical activity* (please observe that both of these terms mean pretty much exactly the same thing). This presumably gave some committee a sense of accomplishment, but did absolutely nothing to improve our understanding of the arrhythmia or our ability to treat it. Keep your eye out for more papers like this: confronted by the overwhelming challenge of actually finding things out that help people train more productively, some exercise physiologists will focus instead on *talking about how we should talk*.

A study by Farinatti et al [52] purports to demonstrate that active women older than 60 years old make bigger gains on a 3-day/week RT program than on a 2-day or 1-day program. However, since

the observed differences were of highly questionable practical significance, since the primary strength outcome measure was a 10RM, and since the program studied was dominated by silly exercises like dumbbell curls, calf raises and knee extensions, its relevance to coaches trying to assemble a serious training program for geriatric patients is open to debate.

IVB. Set Design and Interset Recovery.

Oliver et al [53] compared two different approaches to set design for hypertrophic training. Half the bros performed a “traditional” (TRD) protocol of 4 sets of 10 reps with 120 seconds of rest in between. The other half performed an “intra-set rest” (ISR) protocol of 8 sets of 5 reps with 60 seconds of rest. Training was conducted for 12 weeks. Not surprisingly, the ISR produced greater gains in squat and bench strength. The authors also report, using a bit of statistical hand-waving, that ISR produced greater bench, jump and squat power, but these differences did not reach statistical significance. Hypertrophic adaptations, evaluated as lean mass gains, were not different between the two groups, and fiber type shifting from Type IIx to Type IIa was also no different. These results are at odds with those of other investigators, but the study is well-conducted and well-controlled, although as usual it is small and short. The authors took care to control for total training time, total volume-intensity products, and other variables. One variable they were not able to control was time-under-tension, important for hypertrophic adaptation. I think what the authors are trying say is that a training period focused on hypertrophic gains can incorporate rest intervals that allow for minor acquisitions in strength and power without sacrificing mass accretion or training time. Additional study will be required to reconcile the differences in outcome between this paper and other investigations, like those of Izquierdo et al [54]. Still, the findings will be of interest to collegiate and professional S&C coaches designing periodized programs for their athletes, especially with regard to hypertrophy phases.

IVC. Time of day.

Ekstrand et al [55] studied the effect of a resistance training bout in the morning on afternoon power performance. This was a crossover study with fourteen bros and babes, each of whom participated in three sessions: a familiarization session and two testing sessions. In the control condition, the subjects were assessed for power output in the vertical jump and throwing power using the backward overhead shot throw. In the experimental condition, the subjects performed a morning resistance training bout consisting of cleans and squats, 4-6 hours before testing. No sample size analysis is provided, and the authors, acknowledging the small size of their sample, rely heavily on effect size calculations. The authors report no difference in vertical jump power between the conditions. However, they are pleased to report that a morning resistance training session increased backward shot throw “significantly:” From 11.46 ± 1.28 m to 11.76 ± 1.37 m. They assure us that, while this difference may seem small, 31 cm can be the difference between winning and losing. This would be rather more convincing if their sample wasn’t so painfully small, inviting speculation that a study of 20 bros instead of 14 would have yielded a significant difference in the other direction, or (b) the backward overhead shot throw were an actual competition event, instead of just a weird assay of throwing power. I’m really at a loss to understand why they didn’t just have these guys do a regular competition shot put. After all, they already had a measure of power production with the vertical jump – with no difference. So why not measure a truly competition-specific metric? You can’t get much more competition-specific than the event itself.

If I were to take one thing from this paper, it would not be that resistance training in the morning can improve afternoon performance. Rather, I would take this paper as very weak evidence that resistance training in the morning won't significantly degrade your performance in afternoon practice. *That* deserves more investigation.

IVD. Conditioning.

Perhaps the most important paper in this subcategory for 2013 comes from Lundberg et al [56], who challenge the classic *interference effect* of concurrent aerobic and resistance training. The paper has received a great deal of attention, not necessarily corresponding with the strength of its conclusions or the quality of its methods.

A quick review: in 1980, Hickson [57] demonstrated the classic interference effect between endurance and resistance training when practiced concurrently. Since then, the literature has offered competing data on this point, but it appears the prevailing view among exercise physiologists and coaches alike is that such an interference effect exists to some degree or another [58]. This model has recently found a molecular mechanism in the *AMPK-Akt switch* we discussed in the last Review. However, Lundberg's group recently published biochemical findings at odds with the AMPK-Akt switch model, and now follow up with a study of strength-hypertrophy in the setting of concurrent training.

For the study, ten bros were recruited and subjected to *both* a concurrent resistance exercise-aerobic exercise (RE-AE) *and* resistance-exercise-only training program (RE). How, you may ask, can this be? Simple: bros got on fancy bikes and exercised one leg with AE only, then exercised both legs with RE. This program went on for five weeks, after which bros were subjected to final strength and endurance testing, the inevitable muscle biopsy for a snapshot of some proteins, and MRI of the quads. The authors found that quads in both legs underwent hypertrophy, but quads in the RE-AE leg got more swole. You would think that bigger muscles would be stronger...but this was not the case. The increase in torque (specific strength) was greater after RE than RE-AE, suggesting that "muscle hypertrophy...dissociated from the increase in muscle strength or power."

Dissociated. I hate it when that happens.

If the basic design of the study seems screwy to you, then you're not alone. Although I'm sure the investigators thought they were being clever by forcing bros to serve as their own controls, their model completely ignores the potential confounding effect of contralateral adaptation [59]. But the problems go much deeper. The authors claim to show a superior effect of concurrent training on *hypertrophy*, not force development. Hickson's interference phenomenon was concerned with strength, not hugeness. Be that as it may, I think the authors have failed to convincingly demonstrate even a hypertrophic effect: they offer no measure of anabolic signaling or muscle protein accretion, and their MRI assays of muscle cross-sectional area demonstrate high signal intensities in the RE-AE legs, but not the RE legs. This strongly suggests that the enhanced size of the RE-AE group is due primarily to water accumulation, not protein accretion. In other words, they found that legs that did more overall work got more pump. Stop the presses. The authors wave their hands wildly about why a mere pump doesn't account for their findings, but in the end they must concede that "it cannot be precluded that the robust muscle hypertrophy in part could have been due to expanded sarcoplasmic or interstitial fluids."

There are other problems here, such as a 15-workout program for RE-AE compared to a 12-workout program for RE. But to my mind the biggest issue is the study duration. Hickson's paper

showed that strength and endurance both increased at the onset of concurrent training, and that endurance could continue to improve for as long as 10 weeks (the duration of the original study). Strength only began to fall off at about five weeks in Hickson, and had taken a nose-dive by 10 weeks. But this paper only observed bros for five weeks. In other words, *this study terminated at exactly the time point we would expect to see the very interference effect the authors are challenging.*

Hickson's paper was published a long time ago, and it's not perfect by any means. It has drawn contrary data and criticism [60] and it deserves to be challenged, notwithstanding the mechanistic underpinnings of the AMPK-Akt switch. The thing about switches is that they can be manipulated, and nothing in the literature rules out the prospect of programming or nutritional manipulations that can exploit, bypass or temporize the switch to minimize interference. But this paper is so problematic that it cannot contribute substantively to such a project, nor to our understanding.

And now for one of the most talked-about, most controversial and, from a scientific standpoint, most *unimportant* papers of the year: the "Crossfit Paper" by Smith et al [61]. The authors of this study classified Crossfit as **HIPT**, or **high intensity power training**, as a variant of high-intensity interval training, or HIIT. This is reasonable, and it has become abundantly clear that, whatever its specific bioenergetic impact (see the papers by Abboud and Shing in section IIB above), HIIT results in positive conditioning and body composition adaptations. The authors wondered whether a HIPT program, being a variant of HIIT, will have similar effects. Okay, sure, that encompasses a reasonable hypothesis. It seemed to me a bit like a foregone conclusion, but I suppose it *could* have turned out that a Crossfit-based HIPT program would fail to improve VO_{2max} and body composition.

Well, it didn't. In one of the most unsurprising findings of the year, the authors demonstrate that thrashing around with barbells and kettlebells, Crossfit-style, burns fat and improves your conditioning. The methods are fine, the hypothesis is clearly stated, the primary outcomes are clear and well-measured, and the study's findings are *completely uninteresting*, in my opinion. What would have been *far* more interesting would have been to compare HIPT to a volume-intensity-product-matched program of standard strength training and bicycle-ergometer based HIIT that didn't involve crazy shit like doing snatches for time, and compared the results, adding strength to the outcome measures of VO_{2max} and body composition. A well-powered finding in support of the null hypothesis would have been monumentally more interesting, controversial and explosive than the tempest-in-a-teapot that erupted from this paper.

About that tempest: the authors report that 11 of the original 54 participants dropped out. This was not a primary outcome, and the study was not designed to assess injury rate. Two of the dropouts cited time constraints, and the authors claim that they were given report that 9 subjects (16% of the recruited subjects) cited overuse or injury.

Now, like the primary findings of the study, this should surprise exactly nobody. We're talking about a fitness brand that sports a clown in the throes of rhabdomyolysis attached to a dialysis machine as a mascot [62], and whose founder has declared, publicly, that this exercise modality can kill you [63]. And we're talking about one of a number of fitness programs singled out for concern by the Consortium for Health and Military Performance and the American College of Sports Medicine for their potential to create injury and for their violation of accepted training doctrine [64].

That didn't insulate the authors of this paper from being sued by an Ohio Crossfit affiliate for reporting that 9 people had dropped out because of overtraining and injury. (Presumably, suing a few exercise physiologists looked more like low-hanging fruit than suing the US military and the American College of Sports Medicine.) All sorts of shenanigans, accusations, hijinks, gnashing of teeth and pulling of hair have gone on, including alleged phone calls to the dropouts by a senior Crossfit

official, some or all of whom may have refuted the study's assertions. (Oh, how I would *love* to have eavesdropped on those calls.)

How the authors collected the dropout data is not explicitly described in the paper, (there's a lesson here), and how the information was *actually* gathered appears to be a major issue of contention. From my reading of the complaint filed in the Franklin County Ohio Court [65], it looks like it may boil down to *he-said-then* vs. *she-said-now*. Really, I don't know what happened, and I don't care. The study just isn't that important. But a lawsuit? *Really?* It's hard to see how this does anybody any good.

Except, of course, for the lawyers. Shakespeare was right.

V. Training, Coaching, and PERFORMANCE

In this section, we consider papers relevant to coaching and training and the enhancement of performance on the platform and on the field. I want to begin by referring readers to what is in my biased opinion an extremely important document published by the Starting Strength Coaches' Association on the SS website in October of 2013, drafted by Rippetoe, the senior coaching staff, and Yours Truly. This document lays out the requirements for [Maintenance of Certification for the Starting Strength Coach](#) [66]. I believe it sets forth a uniquely rigorous benchmark for strength and conditioning professionals and establishes a new standard for the industry – which, indeed, was our intention.

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.

Which brings us to a trio of papers focused on little old ladies and middle-aged diabetics. Nascimento et al [67] show that measurement of 1RM in geezettes gets more and more reliable with an increased number of workouts – as few as three for the bench press, curlz and leg press. I'm willing to bet that measurement of 1RM for deads, presses and squats would take just a few more, but let's set that aside, along with any methodological critique (which, as we'll see, is totally beside the point). Let's just take these results, and those of the other two papers, at face value. We shouldn't be at all surprised that as familiarity with an exercise increases (which happens rapidly with exercises like those used here), neuromuscular integration is improved and there's less variance in performance. Those of us who actually spend time training people, especially older people, will not exactly be stunned by such a finding, or by similar results in middle aged type-2 diabetic patients reported by Abdul-Hameed et al [68]. For the same reasons, the finding by Benton et al [69] that trained middle aged women can be more efficiently and accurately assessed for 1RM (of chest and leg press) than untrained women is not much of a revelation.

So let's just say that it's possible to get a consistent 1RM measurement for relatively simple exercises like leg presses, curlz and benches in our more mature clients. Fine. *But none of these papers tell us why in the world we would want to do that.* They all assume we just...*have* to. Nascimento says

it's "considerably important." Benton says it's of "critical importance." *Why?* My coaching practice is focused on older clients, and I've never found a 1RM assessment to be necessary or even useful. None of the coaches I know who work with this demographic use the 1RM assessment for their novice lifters. Nascimento asserts that 1RM testing is safe. He does not cite a peer-reviewed study but rather the ACSM Guidelines. To be useful, one-repetition maximum testing in *any* novice, not to mention an older lifter, must present a probability of benefit that far, *far* outweighs any risk of injury. But the probability of benefit is virtually nil. Coaches get people stronger without this measurement every day. Putting an inexperienced lifter under a maximum load *to get a number you don't need to make that lifter stronger* is irresponsible and stupid. This is a great example of where exercise science is so far removed from common coaching experience that it just gets surreal.

Scherr et al [70] reported on a very large study (n=2560) of the correlation between the Borg Rating of Perceived Exertion (RPE) scale and heart rate. They also looked at the relationship between RPE and lactate production. Their intention was to determine whether the RPE could be used as part of an exercise prescription (a "dose," as it were) for patients with coronary artery disease (CAD). They found that RPE correlated strongly with heart rate and fixed lactate threshold, and concluded that the RPE could be used to prescribe exercise intensity for patients with CAD. Shepard, in a letter to the journal [71] in which Scherr's article was published, argued that the standard deviations in Scherr's data are too wide to allow the RPE to be used safely for exercise prescription in the setting of CAD. Scherr wrote back [72] and agreed that a blanket prescription based on average values was indeed too inaccurate – and this indicated a need for exercise testing of individuals prior to the commencement of exercise. In essence, I think Scherr was saying that we need to generate an RPE-HR curve for every patient with CAD beginning an exercise program, which is, of course, deeply silly. It's a fascinating exchange, but I think both parties ultimately miss the point, because they're thinking in terms of *exercise* instead of *training*. Scherr's paper is nevertheless interesting from an exercise physiology perspective. It is the largest study to date of the correlation between a subjective metric (RPE) and objective physiologic metrics, and I find the relationships between RPE, lactate threshold and anaerobic threshold particularly intriguing. But application to actual coaching practice, particularly for special populations, is uncertain at best.

Tian et al [73] studied heart rate variability as a diagnostic for nonfunctional overreaching in elite female wrestlers, using the *relatively* inexpensive Omegawave monitor. Nonfunctional overreaching is part of a spectrum that extends from functional overreaching (which we in the SS community call "training"), to nonfunctional overreaching (which we call "early overtraining"), to overtraining ("overtraining"), to collapse. The study is worth a read primarily for its introduction and discussion, both of which contain some interesting material on what is known about overreaching and overtraining at the metabolic, neurobiologic and hormonal levels. Moreover, their findings might be of interest to coaches working with elite athletes. In such athletes, diagnosis of overtraining *must* occur before the syndrome is established, and it can be difficult to convince such athletes to reduce their training load based solely on Coach's say-so. But this paper provides us with no demonstration that using a fancy heart rate monitor in conjunction with an even fancier array of frequency domain parameters is any better at diagnosing the early onset of early overtraining than the clinical judgment of an experienced coach. This underscores an important point: as far as I know, *there is no single generally accepted gold standard biomarker for the diagnosis of nonfunctional overreaching*. Although many different biomarkers have been studied, including heart rate variability and hormonal responses, none have ever been shown to be superior to the assessment of a vigilant coach on the lookout for clinical signs and symptoms: a performance detriment that fails to improve within one reduced-load training cycle, chronic pain,

emotional lability, relative resting tachycardia, decreased motivation to train, decreased appetite, and depressed affect. It would be nice to have a biomarker for overtraining that was practical, inexpensive, sensitive, specific and superior to clinical judgment – something like a home pregnancy test. When your overtraining test strip turns blue, it's time to back off. A major advantage would be that you could wave a positive strip in an athlete's face and end the argument right then and there. As of now, no such biomarker exists. Overtraining remains a clinical diagnosis.

I've discussed a few of Brad Schoenfeld's papers now, and in particular I find his reviews of the literature to be clear-headed, thoughtful, penetrating, skeptical and useful. Case in point is his implicit literature review with Bret Contreras [74], which asks an important question: is DOMS a useful metric for strength training? The first part of the review is a concise, clear and very readable overview of the mechanisms producing exercise-induced muscle damage (EIMD), almost universally considered the underlying cause of DOMS, and how these same mechanisms are proposed to enhance muscular adaptation to training. This material, as elaborated by the authors, shows that the question they address has biological plausibility. But as they go on to explain, the actual clinical evidence and coaching experience falls far short of any demonstration that DOMS can be used as a reliable gauge for determining the adequacy and appropriateness of training stimuli. Although routines that produce no soreness may indicate a lack of adequate hypertrophic stimulus (bodybuilders beware!) no such conclusion can be drawn for general strength or power training. DOMS is too subjective, its effects too variable, and its potential for abuse too high, to serve as an appropriate metric for training. Here is another paper that belongs in the literature file of every S&C coach.

VB. General coaching practice and concepts.

Jackson et al [75] took pictures of a guy doing various exercises with a barbell and dumbbells, then showed them to “fitness professionals” self-identifying as academics, personal trainers, coaches or clinicians. They found – surprise! – that the nomenclature applied by these professionals to the various exercises was inconsistent. In other words, half of strength coaches called it a “bench press” while the other half called it a “chest press.”

The authors seem to think this is some kind of catastrophe. They call for more standardization of the nomenclature. There's nothing wrong with that, but the real problem isn't the lack of consistency in everyday use among these professionals. It's that so much exercise science literature fails to adequately document how exercises are actually performed, so that we know what kind of movement is being studied. Like the Steele paper discussed in Programming, this kind of work, while benign, tries to make a contribution by focusing on the language. The real problem with exercise science isn't terminology. It's *methodology*.

The practical application of the phenomenon of post-activation potentiation (see also the paper by Esformes and Bampouras under Squatology) is the subject of a paper by West et al [76]. We now have abundant literature showing that a preloading stimulus, such as a heavy resistance movement, can improve subsequent peak power output. The authors sought to compare the power enhancement induced by a high-intensity resistance upper body movement and a low intensity upper-body ballistic movement, using a counterbalanced, randomized crossover design. Twenty professional rugby players were assayed for their maximum bench press. They then underwent baseline upper body power testing via the ballistic bench press throw (performed on a Smith machine with spotters) at 30% of 1RM. After baseline testing, the subjects were randomized to one of two stimulus conditions: heavy bench presses (3x3 at 87% 1RM) or ballistic bench throws at 30% 1RM. After 8 minutes of rest, they were

again tested for upper body power by ballistic bench throw. The investigators found no difference between the two conditions. I'm bothered that one of the stimulus conditions (the ballistic bench throw) was exactly the same as the assay...in other words, *the dependent variable was identical to one of the independent variables*. And, again, the finding of no difference between the two conditions is deeply undercut by the complete lack of any documentation that the study was adequately powered to detect a difference [1]. At best, the paper generates an interesting hypothesis requiring further study, and is a reminder of the importance and power of post-activation potentiation, a phenomenon that I suspect has not yet been exploited fully.

Keese et al [77] conducted a study of the effect of Very Special Stretching on muscle fatigue. This Very Special Stretching is called ***proprioceptive neuromuscular facilitation*** (PNF). It's Very Special because it combines passive stretching and isometric contractions, a process that supposedly results in salutary neuromuscular properties and an increased range of motion. This is all very important, you see, because there's now a fair amount of evidence that stretching really doesn't do shit for you (see discussion of Sands et al, below). But, the authors point out, most of that data was obtained using *static* stretching protocols, and looked primarily at *strength*. What if we look at Special Stretching (PNF) and *fatigue*? So they did that. They recruited 19 bros and had them do leg curls with and without PNF, and counted their reps. The results? *No difference*. From this the authors conclude that "a moderate level of PNF could be used before resistance exercise with a minimal negative effect." *My conclusion is somewhat different. I conclude, based on this fairly weak data, that "a moderate level of PNF could profitably be skipped altogether with no negative effect and a highly beneficial increase in available training time."*

Sands et al seem to agree [78]. In an important non-systematic review published in *Strength and Conditioning Journal*, the authors have given stretching proponents what Hartmann has given the squats-are-bad-for-the-knees crowd: a thorough and ruthless deconstruction. They begin by making a critical distinction between ***range of motion exercises***, which strengthen movement patterns within current boundaries of tissue extensibility, and ***stretching***, which involves taking the joint range of motion to levels that increase tissue and joint forces beyond those normally available. This is critical: a bodyweight squat or a few reps under an empty bar is a range of motion exercise – not a stretch. Splits and sit-and-reach are another story. The authors then make a penetrating observation:

A serious problem permeates nearly all studies of stretching – how does one measure stretching intensity? ... There does not appear to be a single metric ever proposed to ascertain the level, intensity or magnitude of stretching, short of static measurements of maximum range of positions (e.g., sit-and-reach tests) that are too often completely lacking in a conceptual framework and sport specificity.

Ouch. And yet, despite the lack of consistent metrics in this field of study, on balance the available literature on stretching for warmup or recovery *does not indicate any benefit*. In fact, the best data we have strongly indicates that serious stretching to increase range of motion through acquisition of stretch tolerance *makes you weaker*. The authors try to soften their conclusions and recommendations a bit, more than they needed to, I think. But I have to give you one more paragraph from the end of this very important review:

Possibly the most heretical remark to make about stretching is to suggest that the dedicated use of stretching sessions may not even be necessary, especially since many athletes dispense entirely with special stretching or even warm up sessions before or after training without suffering injury in training or competition.

This is one of those must-have/must read papers. Get it.

In a brief editorial, Glasgow et al [79] present an intriguing model of athletic injury prevention, focusing not on the attainment of a particular ideal movement pattern or training status, but rather on training the ability to adapt to variable stimuli. This model has particular implications for those involved in coaching dynamic sports (soccer, football, combat sports, etc), but it also sheds light on the extremely low injury rates we see with resistance training. It's a short and thought-provoking read.

Staub et al [80] present a study of the effect of “augmented verbal feedback” on the ability to express power. I was disappointed to find that “augmented verbal feedback” didn't mean “yelling.” Rather, this was *knowledge of performance feedback*, in which the athlete was given specific information about his performance – in this case, his or her power output as recorded by a force plate for countermovement jump. The authors found that when athletes were given this information, they performed somewhat better than when they were not coached. The authors conclude that giving athletes feedback on their performance is, well, *good*. No kidding.

Cherie Pettit [81] presents an article on Wellness Coaching certifications. This is not something I recommend that you go out and do. The paper is primarily of interest because of the section on how new health care legislation has the potential to have a profound impact on our industry and elevate fitness professionals to a new tier of importance in the provision of health care.

VC. Sport-specific studies.

Tack [82] proposes a set of “evidence-based” guidelines for strength and conditioning in MMA. I'm disinclined to be too harsh on this paper, because my impression is that the role of strength training is underappreciated in the fighting arts, and because the general approach endorsed by the author (presented in the figure on page 85) is rational, and similar to periodized training approaches we have seen described for other sports (particularly rugby; see the 2011 Review). The author lays down a fair bit of gobbledegook and silliness in his approach to this result, but he still gets there: a program that progresses from general assessment and preparation, to raw strength (using compound barbell movements), to power (using Olympic lifts and complexes), to fight-specific power and conditioning, to strength-power maintenance and technical practice; all integrated into a program that manipulates volume and intensity in a rational way that could be expected to result in peaking just before a competition. Any implication that this approach is “evidence-based” stretches credulity, however. Although the author draws on a wealth of literature (the bibliography is a bonanza of seminal exercise science studies), there is of course no empirical evidence that the approach outlined *here* will yield superior or even adequate results. The paper is definitely worth a read, and the program outlined in the figure is a good starting point for designing your own sport-specific periodized program.

VD. Gear.

McBride's group presents us with a study of effect of compression suits on biomechanical parameters in the squat [83]. The study is well-done, and I won't belabor the details of the results, because most of us aren't using squat suits and I get the idea that equipped powerlifting is starting to lose its luster. Suffice to say that the investigators found that suits did not produce any significant difference in peak concentric forces, but they did have a favorable effect on peak eccentric force, and this seemed to translate into higher peak concentric velocity (and therefore power). The authors do a nice job of summing up what's going on here:

The primary findings in the investigation indicate that squats with (squat suits) elicited higher velocity and power during the concentric portion of the exercise. It is theorized that this is because of the suit's ability to store elastic energy during the eccentric phase of the squat and its release in the concentric phase. One can see the same effect with squats that use the stretch-shortening cycle. (Emphasis added.)

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.

MacDonald et al [84] present the first peer-reviewed data examining the impact of self-myofascial release via foam roller on muscle performance and joint mobility. Although foam rolling has become quite popular, there is virtually no hard data on how the practice actually impacts performance. The theory behind foam rolling and other forms of myofascial torture is that these maneuvers promote the transition of fascial tissue from a relatively solid, inelastic state to a “thixotropic” or gel phase, and that they break up adhesions between adjacent fascial tissues. The authors took 11 Canadian bros and measured their knee ROM, rate of force development (RFD), maximum voluntary contraction force, and evoked force (tetanic). They found that just 2 minutes of quadriceps rolling with a very firm roller (PVC covered by neoprene) resulted in no acute change in the neuromuscular dependent variables, but did produce an increase in knee ROM. They claim these findings support the use of rolling. I suppose that's reasonable; in any event their findings did not show any detriment of the practice and a possible benefit, and I'm predisposed to be sympathetic to such findings (my PVC roller has changed my life). The authors are to be commended for their data presentation: instead of just showing the usual bar graphs (which hide a multitude of sins) they also include plots showing data points for every subject. (See concluding remarks of the [2011 review](#) for my thoughts on this.) On the other hand, there is no power analysis, so the finding that rolling had no impact on the neuromuscular parameters should be taken with a grain of salt. On the whole, the paper is a weak data point in favor of the practice.

Cheng-Yu et al [85] report that icing delays recovery from eccentric exercise-induced muscle damage, with a study that is a perfect example of everything that is wrong with the sports science and exercise physiology literature. The authors took eleven bros and randomized them to ice or no ice. Do the math on how big these study groups were. No power analysis was performed to assure us that such a pathetically small study comes anywhere near having the power to demonstrate anything. Bros did eccentric exercise. Then they underwent five sessions of 15-minute cold pack application at 0, 3, 24, 48 and 72 hours. Control bros got a “sham” treatment which is not described. As one would expect, all the bros leaked creatine kinase and myoglobin. Iced bros seemed to leak more, particularly at 48 and 72 hours. Changes in inflammatory cytokines were all over the place. Changes in VAS-measured pain and fatigue showed statistically significant differences, but the clinical significance of these differences was nil, especially since neither group reported particularly acute pain or fatigue. Despite the report of statistical difference, strength recovery – the real outcome variable of interest – was practically *identical* at every studied time point. This is a poorly designed study with conclusions that cannot possibly be supported by its data. It's almost certain to be quoted widely.

An important review article by Trappe on the use of nonsteroidal anti-inflammatory drugs is discussed below in section **VIIC**.

VII. Nutrition, Supplements and Drugs

VIIA. Anabolics and hormones.

Tavares et al [86] looked at the effect of growth hormone (GH) administration on muscle strength in men over 50 years old. The authors randomized 14 healthy geezers who engaged in regular (and completely unspecified) physical exercise into a control group and a treatment group. All subjects received baseline evaluation of GH, testosterone, body composition and muscle strength. The GH group received an initial dose of 0.5 IU/day, with readjustments to 1 and 1.5 UI/day after 1 and 2 months of treatment respectively. The investigators found no significant effect on bench press strength after 6 months of therapy, and a clinically insignificant effect on leg press strength.

This would seem to be a black eye for GH, and it wouldn't be the first. But the study is small, with no sample size analysis to demonstrate that it's adequately powered, and all that jazz you've heard me rag on again and again. But that's not the biggest problem.

There was no training program here. No progressive overload, no documentation of exercises, no supervised resistance training at all. It's just flabbergasting – these authors actually expected a bunch of geezers to get stronger just because they got growth hormone, without any sort of programmed training intervention. And this paper didn't fall out of *Brocurl Bob's BroScience Quarterly* or any of the other Usual Suspects. This paper was published in the *International Journal of Endocrinology* – which means doctors will read this paper and come away with all sorts of ill-founded conclusions. We still don't really *know* whether healthy seniors in the midst of somatopause will benefit from GH supplementation (probably not). But this paper may actually set the field back. Astonishing.

A non-systematic review of comprehensive treatment strategies for sarcopenia by Wakabayashi and Sakuma [87] is a *little* more useful. The authors make note of the importance of sarcopenia for aging Western populations, and the usefulness of resistance training, but their primary focus is on pharmacological adjunctive therapies. The upshot of this review is that sarcopenia is a target-rich environment, therapeutically speaking, with most pharamacotherapies converging on the Akt/mTOR pathway for upregulation of protein synthesis and muscle protein accretion. Their review of the available evidence of pharmacotherapy is implicit, not a meta-analysis, and should be read with caution. That being said, they find there is some evidence supporting the use of testosterone in men, ghrelin, vitamin D for patients with demonstrated vitamin D deficiency, fish oil, and possibly angiotensin converting enzyme inhibitors. The authors do not find that the literature, on balance, supports the use of dehydroepiandrosterone, estrogen for women, or growth hormone. For each of these putative therapies, the authors correctly point out the available evidence is scant and/or unsatisfactory.

Hackett et al [88] conducted a survey of training practices and ergogenic aids used by male bodybuilders. You'll be surprised to learn, based on the inevitably limited data yielded by such a study, that bodybuilders (a) tend to train in the hypertrophic set-rep range, (b) take dietary supplements, and (c) use steroids. During the off season, the most commonly used anabolics were nandrolone, sustanon, boldenone and testosterone. In preseason training, stanazolol, boldenone, and oxandrolone were most commonly used, along with the nonsteroidal agents clenbuterol, Liothyroxine, and Clomifene. None of this strikes me as a particularly good idea...but then, neither does bodybuilding.

VIIIB. Stimulants.

Ergogenic was the term I previously used, because the term *stimulant* made me feel a bit hinky. I'm over it now.

The Year in Strength Science 2013

In a study of the effect of sugar-free Red Bull on strength and muscular endurance, Eckerson et al [89] have added more mud to the muddy literature on caffeinated energy drinks. The study design was fairly straightforward and familiar. Seventeen bros were given either sugar-free Red Bull, a sugar-free caffeine and water solution, or a flavored placebo, and taken to the gym to measure 1RM bench press and the number of reps at 70% 1RM. They were brought back on two further occasions to repeat the experiment under all three conditions (crossover design). The authors found no difference in bench pressing strength or reps to failure in any testing condition. However (and I have to give the authors props for this), they present not just aggregate data but the results for each subject (Figure 1 of the paper), which is something I've talked about elsewhere. It is interesting to note from this figure that the greatest number of reps was put up by a lifter jacked up on Red Bull, and that the lowest number of reps got logged by a decaffeinated lifter.

But, really, this means nothing, because the study makes a finding that supports the null hypothesis. And, once again, *there is no sample size analysis to assure us that the study is properly powered to accept the null hypothesis*. The introduction and discussion provide a nice overview of the literature on this topic, which is of course all over the place. The authors speculate at length about the conflicting results of studies of caffeinated beverages in training, suggesting that the discrepancies may arise from different experimental conditions, dosing, training status of subjects, or a subpopulation of “non-responders” to caffeine. The one possibility they don't consider is that virtually the entire corpus of literature on this topic is just lousy science, so it's no wonder we don't have an answer yet. I haven't seen anything that would make me want to drink Red Bull, which tastes to me like the urine of an erythematous bovine diabetic; or to lift without coffee, which is of course the Nectar of the Gods.

Hurley et al [90] investigated the effect of caffeine on delayed onset muscle soreness (DOMS), using a crossover study design with an indeterminable number of “low-caffeine-consuming” bros doing preacher curls with and without caffeine on separate occasions. They report that bros had less DOMS, particularly on post-workout day 3, when they consumed caffeine. They also had less muscle tenderness to palpation and were able to do more reps. Creatine kinase levels (indicative of muscle damage) were not different between the two conditions. I'd like to believe this paper. But the differences reported are of highly questionable practical significance, the controls are soft (the authors' boasting to the contrary notwithstanding), the data presentation (in aggregate, instead of individual data points) is inappropriate, and the methods for the tenderness assay, which is especially subject to bias, are not well-described. Moreover, while the authors give us a cursory power analysis, they do not tell us whether it was performed before or after the study was completed, or for which outcome variable(s) the power was calculated. Worse, they report that their power analysis dictated a sample size of *ten* bros. The abstract reports that they recruited *nine* bros. The methods section states they recruited *twelve* bros. Which is it? This is the kind of thing that calls an entire paper into question. It doesn't speak particularly well of the journal editor or reviewers, either.

Petitt et al [91] studied the effect of noncaffeine ingredients in Red Bull, assessing VO_2 , respiratory exchange ratio, rating of perceived exertion, heart rate, and EPOC in 8 bros performing a graded exercise test, and found (surprise!) that they add *nothing*. The study is small and there are minor methodological issues, but these findings are in line with those of other investigators, and I'm predisposed to think they got the right answer.

VIIC. Analgesics and anti-inflammatories.

I never say I told you so, and I can't stand people who do. However, I am compelled to point out that in an [article published on the Starting Strength site in 2012](#), I inveighed at nerdish length against the

longstanding, non-evidence-based mythology that the use of anti-inflammatory therapies, particularly NSAIDs and cooling, would inhibit our adaptation to resistance exercise and stunt our gains. As I noted in that article, Trappe had made particularly important contributions to this field, in the form of (a) a flawed basic science study that seemed to support the anti-NSAID position and (b) a clinical study in older lifters with clinically relevant endpoints (strength gain, hypertrophy) that totally shot that position down. Strangely enough, exponents of the anti-NSAID view often quote (a), but never (b).

Now, Trappe and co-author Sophia Liue have produced an in-depth review of the literature on this topic [92], even nerdier than mine, but couched in more civilized language. It should be required reading for all strength and conditioning coaches. Trappe and Liu undertake an extensive review of the physiology of prostaglandin production by the various cyclooxygenase isoforms, the pharmacology of NSAIDs and acetaminophen, and the relevant research in animals and humans. The review is worth the read just for this stuff, but their conclusion, based on the data we have, is the money shot:

Collectively, these chronic studies, albeit of a limited number, highlight three main points: (1) Chronic consumption of commonly consumed COX inhibitors at over the counter doses during exercise training does not appear to interfere with the muscle mass and strength gains expected from typical resistance exercise training regimens, (2) There appears to be a threshold of the amount of drug that is needed to influence skeletal muscle metabolism and adaptation, (3) There may be differences between the acute and chronic COX inhibitor effects on muscle metabolism between younger and older individuals.

I told you so.

VIII. Nutrition.

Sawyer et al [93] conducted a study of short-term carbohydrate restriction on strength and power performance. They conclude that seven days of restricted carbohydrate intake (about 31 g per day, which is low indeed) resulted in decreased body mass, decreased body fat, and unchanged fat-free mass, with no decline in strength or power. Indeed, the authors report an *increase* in isometric handgrip strength and squatting strength, with a trend toward an increase in bench press strength. Careful examination of the data, however, reveals that changes in body mass and composition were of virtually no practical significance, nor were the changes in strength. Little matter: there is no documentation that the study was adequately powered to detect a difference, anyway. I hate to sound like a broken record, but there it is.

Summary

Thus concludes our survey of the Strength Science literature for 2013. As I said in the introduction, my other efforts for the nascent SSCA constrained my ability to include as many papers or as many categories as I would have liked, and I'm sure many readers are aware of papers they would have expected to see here. Some of those papers will make it into the SSCA library instead – a good incentive to get your coaching certificate. The SSCA Science Committee now has a process in place for the screening, triage, selection, abstraction and presentation of articles to our coaching corps, and we have

also developed processes for testing coaches on these readings and archiving the material. Much of this effort will not have to be repeated, and next year's review will be more comprehensive.

This year, instead of using this section to rag on the state of exercise science (which still deserves it, [as has been pointed out elsewhere](#)), I'd like to instead focus on a number of exciting developments in the field. Papers on effect size statistic and the central importance of sample size analysis are hopefully harbingers of a new standard of rigor in the field. Our understanding of the molecular mechanisms of muscle adaptation and detraining, including the transcriptional response, is growing explosively. A growing body of literature in support of using full range of motion in the barbell back squat should pose a radical challenge to traditional thinking on this subject. The literature on the use of unstable surfaces, which always seemed a goofy concept anyway, seems to be reaching a consensus that bosu balls and balance boards are a big waste of time for serious athletes. There seems to be a growing interest in the coach-trainee relationship, and people are beginning to study the dynamics of interactions between the trainer and the athlete. We can expect this to produce a lot of silliness, but there will be pearls buried in all that gunk, and I'll be on the lookout for them. The use of resistance training for health maintenance and disease management continues to grow, and I think we may see a trend to larger randomized trials. This trend is still inhibited by the very nature of resistance training – it's not the kind of thing you can patent and make gazillions, so industry isn't lining up to fund studies. Even so, the NIH and other agencies can't ignore the need for definitive studies of resistance exercise medicine at a time when American health care is under siege, both epidemiologically and financially. I have no proof of this, mind you – I'm just using the Force here. But that's my *sense* of it. Future reviews will tell the tale.

In my discussion of the Egan-Zierath paper in Section IIB and the Piepoli and Crisafulli paper in section IIA(2), I touched on the topic of *myokines*, signaling peptides released from muscle tissue. This is an avenue of investigation that Pederson broke wide open in 1993 [94], and it has been picking up steam ever since. Since then, it has become clear that *muscle tissue functions as a secretory organ*, releasing a wide range of factors in differential response to exercise or disuse [95]. Our understanding of how training and detraining regulate the release of cytokines, and their role in health and disease, is growing rapidly. For example, recent work, as reviewed by Egan and Zierath, has revealed how the process of excitation-contraction coupling, which links motor neuron firing with membrane depolarization, calcium release and actomyosin formation, also **regulates the release of cytokines from muscle tissue**.

When we couple this large and growing body of data on myokines with the similarly expanding literature on *adipokines*, or signaling molecules released from fat tissue, we quickly come to a realization of staggering importance that has yet to fully penetrate the public imagination or the workaday medical mind: ***Muscle and fat are the two largest glands in the body, and both are directly affected by training.*** I hope to expound on these matters further in future reviews, articles, and a book-length project that is underway. For now, though, I don't think I need to belabor what a profound impact this could have on our picture of human biology in health and disease, or how emphatically it underscores the importance of what we do.

Jonathon M. Sullivan MD, PhD, FACEP, SSC is an attending physician in the Department of Emergency Medicine at Wayne State University/Detroit Receiving Hospital. His research is focused on molecular mechanisms of neuronal salvage and repair after cardiac arrest and stroke. He is a Starting Strength Coach and martial artist, and is particularly interested in the use of barbell training in the geriatric population, because he is well into the process of joining that population. His coaching practice, [GreySteel](#)

MYOKINES AND ADIPOKINES			
NAME	TYPE	ACTIONS	EXERCISE
Myostatin	Myokine	Inhibits muscle differentiation and protein synthesis	Decreased by exercise.
Interleukin-6 (IL-6)	Myokine	Inflammatory when produced at low levels by macrophages; anti-inflammatory when released at high levels in a pulsatile fashion. Pulsatile release enhances glucose and fat oxidation.	Exercise increases plasma levels up to 100-fold in a pulsatile fashion.
Interleukin-15 (IL-15)	Myokine	“Muscle-fat cross talk.” Increases muscle anabolism and fat catabolism.	Increased by exercise.
Interleukin-8 (IL-8)	Myokine	Angiogenesis: increases capillarity/vascularity of skeletal muscle beds.	Increased by endurance, but not concentric exercise.
Insulin-derived growth factor 1 (IGF-1)	Myokine	Survival and growth signaling, protein synthesis, osteogenesis.	Increased by exercise with autocrine effects in muscle, ? endocrine effects systemically.
Brain-Derived Neurotrophic Factor (BDNF)	Myokine	Survival and growth signaling, neuronal development, synaptic plasticity, metabolic regulation and fat oxidation.	Increased in muscle tissue by exercise. Unclear if plasma levels increase.
Fibroblast Growth Factor (FGF)	Myokine	Unclear	Released in response to post-exercise insulin stimulation?
Adiponectin	Adipokine	Regulates substrate oxidation, suppresses insulin resistance and the development of metabolic syndrome, hypertension and atherosclerosis.	Plasma levels increased by exercise.
Leptin	Adipokine	Signals for satiety; wide-ranging effects on multiple organ systems.	Unclear.
TNF-alpha	Adipokine	Pro-inflammatory	Supressed by exercise.
Interleukin-6 (IL-6)	Adipokine	Inflammatory when produced at low levels by macrophages and fat tissue; anti-inflammatory when released at high levels in a pulsatile fashion. Pulsatile release enhances glucose and fat oxidation.	Exercise may suppress low-level IL-6 adipokine response while increasing pulsatile, anti-inflammatory IL-6 myokine response.

Table 1. Myokines and adipokines. A sample of some of the more notable signaling molecules released by muscle and adipose tissue. The differential activity of IL-6 is particularly curious. Table prepared by the author from various sources.

[Strength and Conditioning](#), is located in Farmington, Michigan, and specializes in training adults over 40. Dr. Sullivan is a *deeply* conflicted person, but has no conflict of interest to report with regard to any commercial products or services addressed by articles reviewed herein. People with medical questions or issues should see their doctor. This article is offered for educational purposes only, and does not represent the opinions of the Detroit Medical Center, Wayne State University, the American College of Emergency Physicians or any affiliates, subsidiaries or employees of those organizations. This article does not represent medical advice for any specific patient, disease, injury or condition. So don't get any big ideas.

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REFERENCES

- 1 Beck TW. The importance of a prior sample size estimation in strength and conditioning research. JSCR 2013; 27(8):2323-37.
- 2 Flanagan EP. The effect size statistic – applications for the strength and conditioning coach. Strength Cond J 2013; 35(5):37-40.
- 3 Arandjelovic O. Does cheating pay: the role of externally supplied momentum on muscular force in resistance exercise. Eur J Appl Physiol 2013; 113:135-145.
- 4 Rippetoe M. *Starting Strength: Basic Barbell Training*, 3d Ed. 2011 The Aasgaard Co., Wichita Falls, TX.
- 5 Luk HY, Winter C, O'Neill E, Thompson BA. Comparison of muscle strength imbalance in powerlifters and jumpers. JSCR 2014; 28(1):23-27.
- 6 Comfort P, McMahon JJ, Fletcher C. No kinetic differences during variations of the power clean in inexperienced female collegiate athletes. JSCR 2013; 27(2):363-368.
- 7 The Effect of Loading on Kinematic and Kinetic Variables During the Midthigh Clean Pull. Comfort P, Udall R, Jones Pa. JSCR 2012; 26(5):1208-1214
- 8 Comfort P, Allen M, Graham-Smith P. Comparisons of peak ground reaction force and rate of force development during variations of the power clean. JSCR 2011; 25:3269-3273.
- 9 Saeterbakken AH, Fimland MS. Electromyographi activity and 6RM strength in bench press on stable and unstable surfaces. JSCR 2013; 27(4):1101-1107.
- 10 Saeterbakken AH, Fimland MS. Muscle force output and electromyographic activity in squats with various unstable surfaces. JSCR 2013; 27(1):130-136.
- 11 Yongming L, Cao C, Chen X. Similar electromyographi activities of lower limbs between squatting on a Reebok Core Board and ground. JSCR 2013; 27(5):1349-1353.
- 12 Cotter JA, Chaudhari AM, Jamison ST, Devor ST. Knee joint kinetics in relation to commonly prescribed squat loads and depths. JSCR 2013; 27(7):1765-1774.
- 13 Pereira GR, Leporace G, Virgens Chagas DD, et al. Influence of hip external rotation on hip adductor and rectus femoris myoelectric activity during a dynamic parallel squat. JSCR 2013; 27(10):2749-2754.
- 14 Esformes JI, Bampouras TM. Effect of back squat depth on lower-body postactivation potentiation. JSCR 27(11):2997-3000.
- 15 List R, Gulay T, Stoop M, Lorenzetti S. Kinematics of the trunk and the lower extremities during restricted and unrestricted squats. JSCR 2013; 27(6):1529-1538.

The Year in Strength Science 2013

- 16 Hartmann H, Wirth K, Klusemann M, et al. Influence of squatting depth on jumping performance. *JSCR* 2012; 26(12):3243-3261.
- 17 Hartmann H, Wirth K, Klusemann M. Analysis of the load on the knee joint and vertebral column with changes in squatting depth and weight load. *Sports Med* 2013. Epub ahead of print.
- 18 Nilwik R, Snijders T, Leenders M, et al. The decline in skeletal muscle mass with aging is mainly attributed to a reduction in type II muscle fiber size. *Exp Gerontol* 2013; 49:2-8.
- 19 Verdijk LB, Snijders T, Drost M, et al. Satellite cells in human skeletal muscle; from birth to old age. *Age* 2013; DOI 10.1007/s11357-013-9583-2, epub ahead of print.
- 20 Egan B, Zierath JR. Exercise metabolism and the molecular regulation of skeletal muscle adaptation. *Cell Metab* 2013; 17(2):162-184.
- 21 Mitchell CJ, Churchward-Venne TA, Bellamy L, et al. Muscular and systemic correlates of resistance training-induced muscle hypertrophy. *PLOS-1* 2013; 8(10):e78636.
- 22 Agergaard J, Reitelseder S, Pedersen T, et al. Myogenic, matrix and growth factor mRNA expression in human skeletal muscle: effect of contraction intensity and feeding. *Muscle and Nerve* 2013; epub ahead of print.
- 23 Abboud GJ, Greer BK, Campbell SC, et al. Effects of load-volume on EPOC after acute bouts of resistance training in resistance-trained men. *JSCR* 2013; 27(7): 1936-1941.
- 24 Kelly B, King JA, Goerlach J, Nimmo MA. The impact of high-intensity intermittent exercise on resting metabolic rate in healthy males. *Eur J Appl Physiol* 2013; 113:3039-3047.
- 25 Koziris LP. Sprint interval exercise for fat loss: good return on investment. *Strength and Cond J* 2013; 35(5):41-42.
- 26 Shing CM, Webb JJ, Driller MW, et al. Circulating Adiponectin concentration and body composition are altered in response to high-intensity interval training. *JSCR* 2013; 27(8):2213-2218.
- 27 Utomi V, Oxborough D, Whyte GP, et al. Systematic review and meta-analysis of training mode, imaging modality and body size influences on the morphology and function of the male athlete's heart. *Heart* 2013(3); epub ahead of print.
- 28 Adams J, Schmid J, Parker RD, et al. Comparison of force exerted on the sternum during a sneeze versus during low-, moderate-, and high-intensity bench press resistance exercise with and without the Valsalva maneuver in healthy volunteers. *Am J Cardiol* 2014, epub ahead of print.
- 29 Hackett DA, Chow CM. The Valsalva maneuver: its effect on intra-abdominal pressure and safety issues during resistance exercise. *JSCR* 2013; 27(8):2338-2345.
- 30 Sullivan J. The Valsalva and Stroke: Time for everyone to take a deep breath. Published online at startingstrength.com, 2013, by The Aasgaard Company, Wichita Falls, TX.
- 31 Luo L, Lu AM, Wang Y, et al. Chronic resistance training activates autophagy and reduces apoptosis of muscle cells by modulating IGF-1 and its receptors, Akt/mTOR and Akt/FOXO3a signaling in aging rats. *Exp Gerontol* 2013; 48:427-436.
- 32 Rossi AM, Moullec G, Lavoie KL et al. The evolution of a Canadian hypertension education program recommendation: the impact of resistance training on resting blood pressure in adults as an example. *Canad J Cardiol* 2013:1-6.
- 33 Piepoli MF, Crisafulli A. Pathophysiology of human heart failure: importance of skeletal muscle myopathy and reflexes. *Exp Physiol* 2013: ePub ahead of print.
- 34 Smart NA, Williams AD, Levinger I, et al. Exercise and Sports Science Australia (ESSA) position statement on exercise and chronic kidney disease. *J. Sci Med Sport* 2013; <http://dx.doi.org/10.1015/j.jsams.2013.01.005>.
- 35 Croymans DM, Paparisto E, Lee MM, et al. Resistance training improves indices of muscle insulin sensitivity and beta cell function in overweight/obese, sedentary young men. *J Appl Physiol* 2013; 115:1245-1253.
- 36 Mann S, Beedie C, Balducci S, et al. Changes in insulin sensitivity in response to different modalities of exercise: a review of the evidence. *Diabetes Metab Res Rev* 2014; 30(4):257-68 (reviewed ahead of print).
- 37 Theriault ME, Lemire BB, Pare ME, et al. Regenerative activity of the limb muscle in COPD at rest and after an acute bout of resistance training. *Am J Respir Crit Care Med* 2013; 187:A3811 (Abstract only).

The Year in Strength Science 2013

- 38 Constantin D, Menon MK, Wollof LH, et al. Skeletal muscle molecular responses to resistance training and dietary supplementation in COPD. *Thorax* 2013; 68:625-633.
- 39 Benton MJ, Wagner CL. Effect of single-set resistance training on quality of life in COPD patients enrolled in pulmonary rehabilitation. *Respiratory Care* 2013; 58(3):487-493.
- 40 Strasser B, Siebert U, Schobersberger W. Effects of resistance training on respiratory function in patients with chronic obstructive pulmonary disease: a systematic review and meta-analysis. *Sleep and Breathing* 2013; 17(1):217-226.
- 41 Voet NBM, van der Kooij EL, Riphagen I, et al. Strength training and aerobic exercise for muscle disease. *Cochrane Neuromuscular Disease Group*, 2013; DOI: 10.1002/14651858.CD003907.pub4.
- 42 Gianola S, Pecoraro V, Lambiase S, et al. Efficacy of muscle exercise in patients with muscular dystrophy: a systematic review showing a missed opportunity to improve outcomes. *PLOS One* 2013; doi:10.1371/journal.pone.0065414.
- 43 Keating XD, Castelli D, Ayers SF. Association of weekly strength exercise frequency and academic performance among students at a large university in the United States.
- 44 Hamad NM, Shaw JM, Nygaard IE, et al. More complicated than it looks: the vagaries of calculating intra-abdominal pressure. *JSCR* 2013; 27(11):3204-3215.
- 45 Lohne Seiler H, Torstveit MK, Andressen SA. Traditional versus functional strength training: effects on muscle strength and power in the elderly. *J Aging Phys Act* 2013; 21:51-70.
- 46 McCrory JL, Salacinski AJ, Sellhorst SEH, Greenspan SL. Competitive athletic participation, thigh muscle strength, and bone density in elite senior athletes and controls. *JSCR* 2013; 27(11):3132-3141.
- 47 Pedersen MT, Andersen LL, Jorgensen MB et al. Effect of specific resistance training on musculoskeletal pain symptoms: dose-response relationship. *JSCR* 2013; 27(1):229-235.
- 48 Murlasits Z, Kabir M. Interactions of statin medications and exercise training: considerations for skeletal muscle myopathy. *Strength Cond J* 2013; 35(5):75-78.
- 49 Steel J. Intensity; in-ten-si-ty; noun. 1. Often used ambiguously within resistance training. 2. Is it time to drop the term altogether? *Br J Sports Med* 2013; epub ahead of print.
- 50 Wittgenstein L. *Philosophical Investigations*. Anscombe GEM (translator), 1953.
- 51 Kuhn T. *The Structure of Scientific Revolutions*. 3rd ed. Chicago, IL: University of Chicago Press, 1996.
- 52 Farinatti PTV, Geraldes AAR, Bottaro MF et al. Effects of different resistance training frequencies on the muscle strength and functional performance of active women older than 60 years. *JSCR* 2013; 27(8):2225-2234.
- 53 Oliver JM, Jagim AR, Sanchez AC, et al. Greater gains in strength and power with intraset rest intervals in hypertrophic training. *JSCR* 2013; 27(11):3116-3131.
- 54 Izquierdo M, Gonzalez-Badillo JJ, Hakkinen K, et al. Effects of loading on unintentional lifting velocity declines during single sets of repetitions to failure during upper and lower extremity muscle actions. *Int J Sports Med* 2005; 27:718-724.
- 55 Ekstrand LG, Battaglini CL, McMurray RG, Shields EW. Assessing explosive power production using the backward overhead shot throw and the effects of morning resistance exercise on afternoon performance. *JSCR* 2013; 27(1):101-106.
- 56 Lundberg TR, Fernandez-Gonzalo R, Gustafson Tm, Tesch PA. Aerobic exercise does not compromise muscle hypertrophy response to short-term resistance training. *J Appl Physiol* 2013; 114; 81-89.
- 57 Hickson RC. Interference of strength development by simultaneously training for strength and endurance. *Eur J Appl Physiol* 1980; 45:255-264.
- 58 Fleck SJ, Kraemer WJ. *Designing Resistance Training Programs, 4th Ed*, Chap 4. 2014; Human Kinetics Champaign IL.
- 59 Carroll TJ, Herbert RD, Munn J, et al. Contralateral effects of unilateral strength training: evidence and possible mechanisms. *J Appl Physiol* 2006; 101(5):1514-1522.

The Year in Strength Science 2013

- 60 Gur E, Asghari A, Akbari M, Orhan S. A study of the interference effect of concurrent training (strength and endurance) on strength and VO₂max. *Annals Biol Res* 2013; 3(1):214-221.
- 61 Smith MM, Sommer AJ, Starkoff BE, Devor ST. Crossfit-based high-intensity power training improves maximal aerobic fitness and body composition. *JSCR* 2013; 27(11):3159-3172.
- 62 <http://www.examiner.com/article/crossfit-s-uncle-rhabdo-ignites-volcanic-debate-it-can-fry-your-kidneys>
- 63 <http://www.nytimes.com/2005/12/22/fashion/thursdaystyles/22Fitness.html?pagewanted=all&r=0>
- 64 Bergeron MF, Nindl BC, Deuster PA, et al. Consortium for Health and Military Performance and American College of Sports Medicine consensus paper on extreme conditioning programs in military personnel. *Curr Sports Med Rep* 2011; 10(6):383-389.
- 65 <http://www.scribd.com/doc/214839654/Filed-complaint-against-Steven-Devor-Michael-Smith-and-the-NSCA>
- 66 Rippetoe M, Sullivan JM, Wolf M, Bradford S, Campitelli T, Baker A, Feigenbaum J, Hill S, Horn P, Reynolds M, Ward C, Park S. Starting Strength Coach: From Certificate to Certification. Starting Strength website, Oct 2013, Aasgard Company. http://startingstrength.com/articles/starting_strength_coach_moc.pdf
- 67 do Nascimento, MA, Januario B, Gerage AM, et al. Familiarization and reliability of one repetition maximum strength testing in older women. *JSCR* 2013; 27(6):1636-1642.
- 68 Abdul-Hameed U, Rangra MPThP, Shareef MY, Hussain ME. Reliability of 1-repetition maximum estimation for upper lower body muscular strength measurement in untrained middle-aged type 2 diabetic patients. *Asian J Sports Med* 2012; 3(4):267-273.
- 69 Benton MJ, Raab S, Waggenger GT. Effect of training status on reliability of one repetition maximum testing in women. *JSCR* 2013; 27(7):1885-1890.
- 70 Scherr J, Wolfrath B, Christle JW, et al. Associations between Borg's rating of perceived exertion and physiological measures of exercise intensity. *Eur J Appl Physiol* 2013; 113:147-155.
- 71 Shepard RJ. A critique of RPE as a basis for exercise prescription. *Eur J Appl Physiol* 2013; 113:1369-1370.
- 72 Scherr J, Halle M. Reply to the comment of Dr. Roy J. Shepard "A critique of RPE as a basis for exercise prescription." *Eur J Appl Physiol* 2013; 113:1371-1372.
- 73 Tian Y, Zi-Hong H, Zhao JX, et al. Heart rate variability threshold values for early-warning nonfunctional overreaching in elite female wrestlers. *JSCR* 2013; 27(6):1511-1519.
- 74 Schoenfeld BJ, Contreras B. Is postexercise muscle soreness a valid indicator of muscular adaptations? *Strength and Cond J* 2013; 35(5):16-21.
- 75 Jackson MC, Brown LE, Coburn JW et al. Towards standardization of the nomenclature of resistance training exercises. *JSCR* 2013;27(5);1441-1449.
- 76 West DJ, Cunningham DJ, Crewther BT, et al. Influence of ballistic bench press on upper body power output in professional rugby players. *JSCR* 2013; 27(8):2282-2287.
- 77 Keese F, Farinatti P, Massafferri R, et al. Acute effect of proprioceptive neuromuscular facilitation stretching on the number of repetitions performed during a multiple set resistance exercise protocol. *JSCR* 2013; 27(11):3028-3032.
- 78 Sands WA, McNeal JR, Murray SR, et al. Stretching and its effects on recovery: a review. *Strength and Cond J* 2013; 35(5):30-36.
- 79 Glasgow P, Bleakley CM, Phillips N. Being able to adapt to variable stimuli: the key driver in injury and illness prevention? *Br J Sports Med* 2013; 47:64-65.
- 80 Staub JN, Kraemer WJ, Pandit AL, et al. Positive effects of augmented verbal feedback on power production in NCAA Division I collegiate athletes. *JSCR* 2013; 27(8):2067-2072.
- 81 Pettitt C. Wellness coaching certifications: a new career frontier for personal trainers in health care. *Strength and Cond J* 2013; 35(5):63-67.
- 82 Tack C. Evidence-based guidelines for strength and conditioning in martial arts. *Strength Cond J* 2013; 35(5):79-92.
- 83 Blatnik JA, Skinner JW, McBride JM. Effect of supportive equipment on force, velocity, and power in the squat. *JSCR* 2012; 26(12):3204-3208.

The Year in Strength Science 2013

- 84 MacDonald GZ, Penney MDH, Mullaley ME, et al. An acute bout of self-myofascial release increases range of motion without a subsequent decrease in muscle activation or force. *JSCR* 2013; 27(3): 812-21.
- 85 Tseng CY, Lee JP, Tsai YS, et al. Topical cooling (icing) delays recovery from eccentric exercise-induced muscle damage. *JSCR* 2013;27(5):1354-1361.
- 86 Tavares ABW, Micmacher E, Biesek S, et al. Effects of growth hormone administration on muscle strength in men over 50 years old. *Internat J Endocrin* 2013; doi: 10.1155/2013/942030.
- 87 Wakabayashi H, Sakuma K. Comprehensive approach to sarcopenia treatment. *Curr Clin Pharm* 2014; 9:000-000. Epub.
- 88 Hackett DA, Johnson NA, Chow CM. Training practices and ergogenic aids used by male bodybuilders. *JSCR* 2013; 27(6):1609-1617.
- 89 Eckerson JM, Bull AJ, Baechle TR, et al. Acute ingestion of sugar-free Red Bull energy drink has no effect on upper body strength and muscular endurance in resistance-trained men. *JSCR* 2013; 27(8):2248-2254.
- 90 Hurley CF, Hatfield DL, Reibe DA. The effect of caffeine ingestion on delayed onset muscle soreness. *JSCR* 2013; 27(11):3101-3109.
- 91 Pettitt RW, Niemeier JD, Serton PJ, et al. Do the noncaffeinated ingredients of energy drinks affect metabolic responses to heavy exercise? *JSCR* 2013; 27(7):1994-1999.
- 92 Trappe T, Liu Sophia. Effects of prostaglandins and COX inhibiting drugs on skeletal muscle adaptations to exercise. *J Appl Physiol* 2013; epub ahead of print.
- 93 Sawyer JC, Wood RJ, Davidson PW, et al. Effects of a short-term carbohydrate-restricted diet on strength and power performance. *JSCR* 2013; 27(8):2255-2262.
- 94 Pedersen, B. K. et al. Searching for the exercise factor: is IL6 a candidate? *J. Muscle Res. Cell Motil.* 24, 113–119 (2003)
- 95 Pedersen BK. Muscles and their myokines. *J Exp Biol* 2011; 214:337-346.

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