THE BEST OF MASS 2019-2020

MASS

MONTHLY APPLICATIONS IN STRENGTH SPORT

ERIC HELMS | GREG NUCKOLS | MICHAEL ZOURDOS | ERIC TREXLER

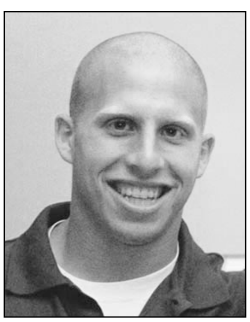
The Reviewers

Eric Helms

Eric Helms is a coach, athlete, author, and educator. He is a coach for drug-free strength and physique competitors at all levels as a part of team 3D Muscle Journey. Eric regularly publishes peer-reviewed articles in exercise science and nutrition journals on physique and strength sport, in addition to writing for commercial fitness publications. He's taught undergraduate- and graduate-level nutrition and exercise science and speaks internationally at academic and commercial conferences. He has a B.S. in fitness and wellness, an M.S. in exercise science, a second Master's in sports nutrition, a Ph.D. in strength and conditioning, and is a research fellow for the Sports Performance Research Institute New Zealand at Auckland University of Technology. Eric earned pro status as a natural bodybuilder with the PNBA in 2011 and competes in the IPF at international-level events as an unequipped powerlifter.

Greg Nuckols

Greg Nuckols has over a decade of experience under the bar and a B.S. in exercise and sports science. Greg earned his M.A. in exercise and sport science from the University of North Carolina at Chapel Hill. He's held three all-time world records in powerlifting in the 220lb and 242lb classes. He's trained hundreds of athletes and regular folks, both online and in-person. He's written for many of the major magazines and websites in the fitness industry, including Men's Health, Men's Fitness, Muscle & Fitness, Bodybuilding.com, T-Nation, and Schwarzenegger.com. Furthermore, he's had the opportunity to work with and learn from numerous record holders, champion athletes, and collegiate and professional strength and conditioning coaches through his previous job as Chief Content Director for Juggernaut Training Systems and current full-time work on StrongerByScience.com.



Michael C. Zourdos

Michael (Mike) C. Zourdos, Ph.D., CSCS, has specializations in strength and conditioning and skeletal muscle physiology. He earned his Ph.D. in exercise physiology from The Florida State University (FSU) in 2012 under the guidance of Dr. Jeong-Su Kim. Prior to attending FSU, Mike received his B.S. in exercise science from Marietta College and M.S. in applied health physiology from Salisbury University. Mike served as the head powerlifting coach of FSU's 2011 and 2012 state championship teams. He also competes as a powerlifter in the USAPL, and among his best competition lifts is a 230kg (507lbs) raw squat at a body weight of 76kg. Mike owns the company Training Revolution, LLC., where he has coached more than 100 lifters, including a USAPL open division national champion.

Eric Trexler

Eric Trexler is a pro natural bodybuilder and a sports nutrition researcher. Eric has a PhD in Human Movement Science from UNC Chapel Hill, and has published dozens of peer-reviewed research papers on various exercise and nutrition strategies for getting bigger, stronger, and leaner. In addition, Eric has several years of University-level teaching experience, and has been involved in coaching since 2009. Eric is the Director of Education at Stronger By Science.

Table of Contents

Letter from the Reviewers

Welcome to the 2019-2020 “Best Of” issue of MASS! Whether this is the first time you’re getting a peek inside our research review or you’ve been subscribed since day 1, we think you’ll love what you find in this special edition of MASS.

Since we launched MASS in April 2017, we’ve published 36 issues – that’s about 325 articles and videos, 3,000 pages of content, 200 audio roundtable episodes, 700 illustrative graphics, and 70 hours of video. We offer CEUs for NSCA and NASM and CECs for ACSM and ACE. As of April 2020, we have more than 3,500 active subscribers. (Not a subscriber yet? Join here.)

What you’ll find in these pages is a glimpse at some of our favorite content from the third year of MASS, but you can be confident that every issue is packed with rigorously examined and visually stunning reviews of the research that’s most relevant to strength and physique athletes, coaches, and enthusiasts.

If you (or your clients) want to build muscle, get stronger, and/or drop fat as efficiently and effectively as possible, MASS is for you. We know you want to stay on top of the research, but doing so can be time-consuming, expensive, and confusing. That’s why we do all the heavy lifting for you and distill the most important findings into an easy-to-read monthly digest.

This free issue should give you an idea of what you can expect from MASS. In our written pieces, we cover using velocity to autoregulate, the principle of specificity, the link between processed food and overeating, using fat-free mass index, the placebo effect, RPE and RIR, Vitamin D supplementation, hypertrophic supercompensation, and more.

In our unique video content, Mike tells you everything you need to know about plus sets, and Eric Helms examines sticking points.

Each issue will tackle new topics like these, keeping you up to date with the current research and giving you a thorough understanding of the best science-based practices. We hope you enjoy it, and we hope you’ll subscribe so you can stay on the cutting edge of our field to get the best results possible for yourself or your clients.

Thanks so much for reading.

The MASS Team

Eric Helms, Greg Nuckols, Mike Zourdos, and Eric Trexler

Study Reviewed: Comparison of Velocity-Based and Traditional Percentage-Based Loading Methods on Maximal Strength and Power Adaptations. Dorrell et al. (2019)

Using Velocity to Autoregulate May Increase Strength Gains

BY GREG NUCKOLS

We've talked about using velocity to autoregulate training before in MASS, but when the rubber meets the road, does autoregulating training using velocity targets and velocity stops ultimately lead to larger strength gains than percentage-based training? This study says "yes."

KEY POINTS

1. Over six weeks, velocity-based training led to significantly larger gains in bench press strength and jump height than traditional percentage-based training in trained lifters.
2. Across four lifts – squat, bench press, overhead press, and deadlift – strength gains were almost 50% larger with velocity-based training, in spite of the fact that training volume was slightly lower.

Some days, you hit the gym feeling great, and your prescribed workout barely challenges you. Other times, you're tired and fatigued, and your performance in the gym is well below your usual level. Autoregulation strategies, which we've talked about in MASS many times before (one, two, three, four, five, six), help you take advantage of the good days and pull back on the bad days in a logical, controlled manner.

One method of autoregulation is via the use of velocity. As loads increase, mean concentric velocity decreases in an almost perfectly linear fashion. Because of this, you can use velocity as a stand-in for traditional percentages of 1RM for prescribing intensity. However, percentages of 1RM don't change until the next time you max, whereas velocity is responsive to day-to-day fluctuations in strength, making velocity a prime candidate for autoregulation strategies.

However, until now, we didn't have firm evidence that autoregulating training using velocity actually led to larger strength gains than training with a traditional percentage-based approach. A recent study (1) found that, in trained subjects, velocity-based training led to significantly larger increases in jump height and bench press strength than traditional percentage-based training over six weeks. This finding puts autoregulatory strategies using velocity on a much firmer footing.

Purpose and Research Questions

Purpose

The purpose of this study was to compare the effects of velocity-based training and percentage-based training on strength and power adaptations after a six-week block of training.

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

Table 1 Subject characteristics

Age (years)	Body mass (kg)	Height (m)	Squat (kg)	Bench press (kg)	Overhead press (kg)	Deadlift (kg)
22.8 ± 4.5	89.3 ± 13.3	180.2 ± 6.4	140.2 ± 26	107.7 ± 18.2	61.3 ± 8.7	176.6 ± 27.2

Data are mean ± SD

Research Questions

1. Would velocity-based or percentage-based training lead to larger strength gains in the squat, bench press, overhead press, and deadlift after a six-week block of training?
2. Would velocity-based or percentage-based training lead to larger increases in counter-movement jump height after a six-week block of training?

Hypotheses

No hypotheses were directly stated, but the wording of the introduction implies that the authors expected that velocity-based training would lead to larger gains in strength and counter-movement jump height.

Subjects and Methods

Subjects

Of the 30 men that initially volunteered for this study, 3 got injured and 11 failed to meet all inclusion criteria, leaving a final sample of 16 subjects. Subjects were required to have at least two years of resistance training experience. They turned out to be a pretty well-trained sample by the standards of most research in the area; the average 1RM squat was a little over 1.5x bodyweight, and the average deadlift was nearly double bodyweight.

Design

This study took place over approximately seven weeks, with a day of testing pre- and post-training, and six weeks of training. The pre-testing day took place at least 96 hours before the first training session, and the post-testing day took place at least 96 hours after the last training session. Testing consisted of counter-movement jump height and 1RMs for back squat, bench press, overhead press, and deadlift.

Training took place twice per week. Both days included back squat, bench press, and squat jump. Day 1 also included overhead press, seated rows, and walking lunges, while day 2 also included deadlifts, plyo push-ups, and barbell hip thrusts. The program itself included two three-week waves, with the first wave increasing in intensity from 70% 1RM to 85-88%, and the second wave increasing from 80-82% to 95%. More details about the training program can be seen in Table 2.

Table 2 Descriptive characteristics of the base training program*†

	Week 1		Week 2		Week 3		Week 4		Week 5		Week 6	
Exercise	Reps	%1RM	Reps	%1RM	Reps	%1RM	Reps	%1RM	Reps	%1RM	Reps	%1RM
Session 1												
Back squat	8, 8, 8	70, 70, 70	8, 6, 5	70, 75, 80	6, 5, 3	75, 80, 85	8, 6, 5	70, 75, 80	6, 5, 3	78, 85, 90	5, 3, 2+	85, 90, 95
Bench press	8, 8, 8	70, 70, 70	8, 6, 5	70, 75, 80	6, 5, 3	75, 80, 85	8, 6, 5	70, 75, 80	6, 5, 3	78, 85, 90	5, 3, 2+	85, 90, 95
BB squat jump	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM		
Strict OHP	8, 8, 8	70, 70, 70	8, 6, 5	70, 75, 80	6, 5, 3	75, 80, 85	8, 6, 5	70, 75, 80	6, 5, 3	78, 85, 90	5, 3, 2+	85, 90, 95
Deadlift											5, 3, 2+	85, 90, 95
Seated row	6, 6, 6	2 RIR	6, 6, 6	2 RIR	6, 6, 6	2 RIR	6, 6, 6	2 RIR	6, 6, 6	2 RIR		
Walking Lunge	10, 10, 10		10, 10, 10		10, 10, 10		10, 10, 10		10, 10, 10			
Session 2												
Back squat	8, 8, 8	70, 70, 70	8, 6, 5	70, 75, 80	6, 5, 3+	75, 83, 88	8, 6, 5	70, 75, 82	6, 4, 2	78, 88, 92	4, 4, 4	70, 70, 70
Bench press	8, 8, 8	70, 70, 70	8, 6, 5	70, 75, 80	6, 5, 3+	75, 83, 88	8, 6, 5	70, 75, 82	6, 4, 2	78, 88, 92	4, 4, 4	70, 70, 70
BB squat jump	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM		
Strict OHP											4, 4, 4	70, 70, 70
Deadlift	8, 8, 8	70, 70, 70	8, 6, 5	70, 75, 80	6, 5, 3	75, 80, 85	8, 6, 5	70, 75, 80	6, 5, 3	78, 85, 90	4, 4, 4	70, 70, 70
Plyo push-up	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM	2 (3), 2 (3)	BM		
BB hip thrust	8, 8, 8	+BM	8, 8, 8	+BM	8, 8, 8	+BM	8, 8, 8	+BM	8, 8, 8	+BM		

**RM = repetition maximum; BB = barbell; 2 (3) = cluster set, 2x3 repetitions; BM = body mass; OHP = overhead press; RIR = repetitions in reserve; Plyo = plyometric; +BM = completed with body mass on the barbell*
† = walking lunge load calculated (Ebben et al., 2008): 0.6 (6RM squat [kg; 0.52] + 14.82kg)

One group used a percentage-based program, and one group used a velocity-based program. The percentage-based program is the one in Table 2. In order to equate the two programs, the velocity-based group used velocity zones and velocity stops, rather than percentages and prescribed numbers of reps. Loads were dictated by the subject's performance on each training day with the velocity-based program, so that when their velocities were higher or lower than normal, they could train with heavier or lighter loads to stay in the correct velocity range.

It's not entirely clear how the velocity zones were defined in this study; the authors note that "group zones for each movement were created using a combination of previously published data and data collected within the pretesting 1RM assessments," but no additional information is provided about how those two data sources were integrated or how they determined the size of each range. The velocity stops are a bit ambiguous as well. The authors state "velocity stops were integrated into each set at 20% below the target velocity of each specific zone." They cite this paper (which was previously reviewed in MASS) as a reference (2), and in that study, they terminated each set when velocity dropped by more than 20% from the first rep in the set. I *think* that's what they did in this study. However, that statement could also be interpreted to mean that each set was terminated when velocity fell 20% below the bottom end of the target velocity range.

That's a non-negligible distinction, because their velocity targets seem to be fairly wide. For example, the velocity target for the squat to correspond to 70% 1RM was 0.74-0.88 m/s. If the velocity stop kicked in when rep speed dropped by 20% within a set, then someone whose first rep was 0.88m/s would terminate a set when their velocity dropped to 0.70m/s, and someone whose first rep was 0.74m/s would terminate a set when their velocity dropped to 0.59m/s. If the velocity stop kicked in when rep speed dropped to 20% below the bottom of the target range, then each squat set in this intensity range would terminate at a velocity of 0.59m/s, regardless of where the first rep fell in the 0.74-0.88m/s range.

I'll admit that I may just be being a bit too pedantic, since I'm sure I could do a bang-up job of approximating their target velocity ranges for each intensity, and since both potential interpretations of their velocity stop method would probably be fine in practice. However, for a study that's this novel in the literature, I'd really like to know *exactly* how the velocity-based program was executed, but the methods section doesn't provide me with enough information to know exactly how they prescribed loads and how they decided when to terminate each set.

Findings

Both groups got significantly stronger in the squat, bench press, and overhead press. Only the velocity-based group got significantly stronger in the deadlift. Additionally, only the velocity-based group had a significant increase in counter-movement jump height. There were only significant between-group differences for the bench press and counter-movement jump. Overall, the velocity-based group added an average of 37.3kg to their four main lifts, while the percentage-based group added 25.1kg.

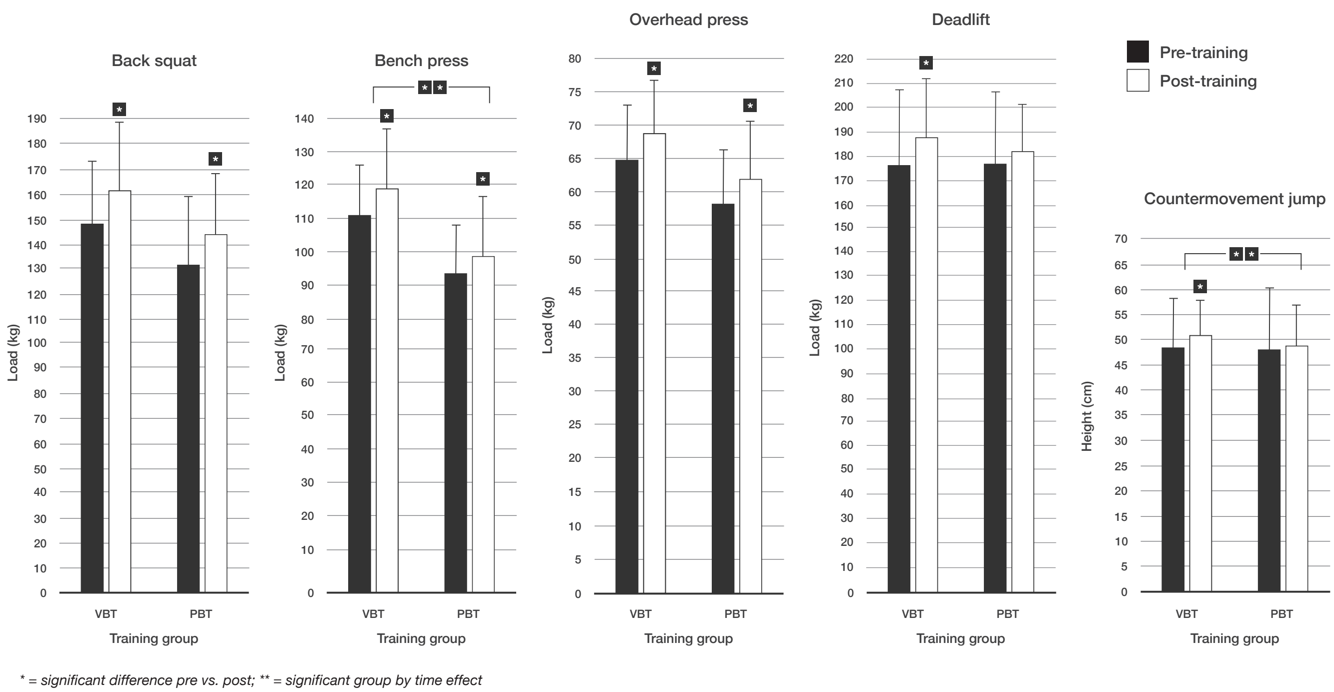
NOW THAT WE CAN SEE THAT USING VELOCITY TO ASSIGN TRAINING LOADS ACTUALLY LEADS TO FASTER STRENGTH GAINS THAN USING PERCENTAGES, THAT LETS US KNOW THAT ALL OF THAT WORK FLESHING OUT THE LOAD-VELOCITY LITERATURE WASN'T IN VAIN.

Interestingly, volume load (sets x reps x weight) was slightly – though significantly – lower in the velocity-based group for the squat, bench press, and overhead press. The overall difference in volume load was small (5.9%), but the velocity-based group was a little stronger at baseline (~8.4% stronger), so relative volume load (sets x reps x %1RM) was closer to 19% lower in the velocity-based group.

Interpretation

This was a really cool study that was much-needed. In the past decade, there's

Figure 1 Mean changes in back squat, bench press, strict overhead press, and deadlift 1RM and counter-movement jump after 6 weeks of training



been a lot of work digging into load-velocity profiles. We've reviewed several load-velocity papers for MASS already (one, two, three, four, five, six). However, with any new form of monitoring, or new way to assign training loads, the most important question is, "does this actually matter?" If it doesn't ultimately help people reach their goals more effectively and efficiently, it's ultimately just mental masturbation and overcomplication for the sake of feeling more in control. Now that we can see that using velocity to assign training loads actually leads to faster strength gains than using percentages, that lets us know that all of that work fleshing out the load-velocity literature wasn't in vain (assuming these results replicate).

With that being said, I do have a few reservations about these results. First, this study was just six weeks long. Yes, that's a cheap critique, and I don't hold that against the authors (that's still a TON of work), but it's at least worth considering the possibility that results would have been different if the study ran longer. More substantially, I think there was an important confounding variable in this study: The subjects in the velocity-based group were told their velocity for each rep. Some research suggests that intentionally moving the bar as fast as possible leads to larger strength gains, and velocity feedback improves acute performance (3, 4). An assumption with velocity-based training is that you move each rep as fast as you can. If

Table 3

	Volume load (kg)			Relative volume load		
	Velocity-based	Percentage-based	Percent difference	Velocity-based	Percentage-based	Percent difference
Back squat	114896	125010	8.8%	777.4	947.8	21.9%
Bench press	117457	123982	5.6%	1060.1	1319.0	24.4%
Overhead press	65742	69593	5.9%	1017.7	1197.8	17.7%
Deadlift	66827	67735	1.4%	378.8	382.9	1.1%
Total	364922	386320	5.9%	3234.0	3847.4	19.0%

Relative volume load = volume load / pre-training 1RM

you don't, your velocity data is essentially worthless, since all of the ways you can prescribe training using velocity is predicated on the linear relationship between load and velocity, and between proximity to failure and velocity *when maximum effort is exerted*. Thus, the velocity-based group a) knew (or at least should have known) that they *really* needed to put forth their full effort on each rep to make the velocity-based load and volume prescriptions work in the first place, and b) the velocity feedback on each rep essentially functions as external cuing (reminding you to move the bar fast). As MASS readers should know by now, external cueing improves performance (5). Thus, the superior strength gains in the velocity-based group may have been due to the velocity-based training, but they may have been at least partially due to the constant velocity feedback. However, that *may* be a distinction without a difference, as velocity-based training *does* force you to stay intimately aware of your velocity on each rep and *does* force you to move each rep as fast as possible, neither of which are typical (and certainly not required) for percentage-based programs.

With that being said, I'm less skeptical of these results than I would be if velocity-based training didn't have strong theoretical underpinnings. The idea just makes sense: On days you're strong, a velocity-based approach will allow you to train with heavier loads or do more volume, and on days you're weak and under-recovered, a velocity-based approach will have you pull back on your training loads and/or volume to allow you to recuperate. Over time, those small marginal advantages in each session, resulting from improved matching of training stress and readiness, *should* lead to better results. I do think the ~50% faster average strength gains with velocity-based training in this study is pretty unrealistic (I think the effect they found is correct, but the relative magnitude of the effect is larger than the "true" magnitude), especially since the study ran just six weeks. I do think the theory is sound, though, and I feel even better about it now that it's been directly tested.

ON DAYS YOU'RE STRONG, A VELOCITY-BASED APPROACH WILL ALLOW YOU TO TRAIN WITH HEAVIER LOADS OR DO MORE VOLUME, AND ON DAYS YOU'RE WEAK AND UNDER-RECOVERED, A VELOCITY-BASED APPROACH WILL HAVE YOU PULL BACK ON YOUR TRAINING LOADS AND/OR VOLUME TO ALLOW YOU TO RECUPERATE.

One thing to note is that the load prescription in this study could have been even more individualized. The authors used *group* velocity targets for each lift and intensity, whereas individualized targets would be easy to figure out, and would do an even better job of personalizing load prescriptions. I understand the decision completely: it would be a HUGE pain in the ass to come up with individualized velocity targets for each lift, each intensity, *and* each subject (4 exercises x 9 different intensity targets x 16 subjects = 576 velocity targets you need to calculate *and* keep track of, without making mistakes during data collection), but it's entirely realistic for two individuals to move the bar at speeds that differ by 0.1-0.2m, even when performing the same exercise at the same intensity. Basically, if you put all training programs on a continuum from maximally rigid to maximally autoregulated, the method of assigning loads to the velocity-based group in this study would certainly be much closer to the maximal autoregulation pole than the maximal rigidity pole, but it could get even more personalized and autoregulation-y.

If you saw this study in a vacuum, it may surprise you. After all, the traditional group trained with higher absolute volume loads (and even higher relative volume loads) but still managed to gain less strength. However, results like this should be familiar to MASS readers. Mike covered a study a while back showing that terminating each set after a 20% velocity loss led to larger gains in jump height and possibly larger strength gains than terminating each set after a 40% velocity loss, even though volume load was way lower in the 20% velocity loss group (2). For that study, I suggested that perhaps the 40% velocity loss group was just more fatigued at post-testing. However, that explanation doesn't fly in this study. The second workout of week 6 is intentionally easy (2 sets of 3 with 70% 1RM), and post-testing didn't take place until at least 96 hours after the last training session, so both groups rolled into post-testing after about a week of deloading. So, how can you equate for

APPLICATION AND TAKEAWAYS

If you have a device for measuring bar velocity, you may be able to use velocity targets and velocity stops to create a training program that is more responsive to you and that will ultimately lead to faster strength gains. If you don't, RPE stops and RPE load targets may work just as well, given the emerging work on RPE programs, which Mike reviewed this month.

intensity, have a lower volume load, and still make larger strength gains? Intensity is the primary driver of strength gains (6), and I think that staying further from failure during training helps ensure that subsequent workouts are also high quality.

If you're interested in making your own load-velocity profile and having your own personalized velocity targets, you can make a copy or download this spreadsheet (do not request editing access) which will do most of the heavy lifting for you, as long as you have a device you can use to measure velocity in the first place.

Next Steps

As I mentioned, I think the velocity feedback in one group and not the other could have biased the results of this study a bit. To remedy that, future studies should either a) provide velocity feedback to both groups or b) simply have the researchers encourage both groups to move every rep as fast as possible, without providing velocity feedback to either group (i.e. the researchers would be watching the velocities to know when an appropriate load has been reached, and would tell their velocity-based subjects when to cut a set and rack the bar based on velocity loss criteria).

References

1. Dorrell HF, Smith MF, Gee TI. Comparison of Velocity-Based and Traditional Percentage-Based Loading Methods on Maximal Strength and Power Adaptations. J Strength Cond Res. 2019 Feb 18.
2. Pareja-Blanco F, Rodríguez-Rosell D, Sánchez-Medina L, Sanchis-Moysi J, Dorado C, Mora-Custodio R, Yáñez-García JM, Morales-Alamo D, Pérez-Suárez I, Calbet JAL, González-Badillo JJ. Effects of velocity loss during resistance training on athletic performance, strength gains and muscle adaptations. Scand J Med Sci Sports. 2017 Jul;27(7):724-735.
3. González-Badillo JJ, Rodríguez-Rosell D, Sánchez-Medina L, Gorostiaga EM, Pareja-Blanco F. Maximal intended velocity training induces greater gains in bench press performance than deliberately slower half-velocity training. Eur J Sport Sci. 2014;14(8):772-81.
4. Nagata A, Doma K, Yamashita D, Hasegawa H, Mori S. The Effect of Augmented Feedback Type and Frequency on Velocity-Based Training-Induced Adaptation and Retention. J Strength Cond Res. 2018 Feb 14.
5. Wulf G. Attentional focus and motor learning: a review of 15 years. International Review of Sport and Exercise Psychology 2013 6:1, 77-104
6. Schoenfeld BJ, Grgic J, Ogborn D, Krieger JW. Strength and Hypertrophy Adaptations Between Low- vs. High-Load Resistance Training: A Systematic Review and Meta-analysis. J Strength Cond Res. 2017 Dec;31(12):3508-3523.

Study Reviewed: Phase-Specific Changes in Rate of Force Development and Muscle Morphology Throughout a Block Periodized Training Cycle in Weightlifters. Suarez et al. (2019)

The Principle of Specificity Holds True, But Is It All That Matters?

BY MICHAEL C. ZOURDOS

This study observed competitive weightlifters undergoing block periodization for seven months. The adaptations tended to be block-specific. So, how can you periodize training but still prioritize specificity to peak over the long term? This article breaks it down.

KEY POINTS

1. This study observed changes in rate of force development and muscle size in nine competitive weightlifters over the course of a block periodized macrocycle.
2. The principle of specificity ruled the day, as lifters got bigger during the highest volume block but tended to lose size during lower volume blocks. Lifters also increased rate of force development during strength- and power-focused blocks, but tended to see declines in rate of force development during the high-volume training block.
3. Ultimately, specificity is one of the most important training principles that should be adhered to; therefore, this article discusses how to maintain some semblance of specificity, even during volume phases of a macrocycle when your ultimate goal is strength.

Two things that are hard to come by in the realm of training studies are: 1) Truly long-term studies, and 2) the use of well-trained and competitive lifters. The presently reviewed study (1) has both of those items, as it examined muscle hypertrophy and changes in rate of force development over a macrocycle of about seven months in nine competitive male and female weightlifters. Specifically, this study observed weightlifters who completed a block periodized program with three different training blocks during the macrocycle: a strength endurance block, a strength/power block, and a peaking block (i.e. one-week overreach followed by a taper). Rate of force development and muscle size were assessed before and after each specific training block. The results of this study weren't groundbreaking, as adaptations occurred in concert with the long-standing principle of specificity. In other words, hypertrophy tended to occur after the strength endurance block (i.e. the highest volume phase), but muscle size tended to decrease following the low-volume strength power block. On the other hand, rate of force development tended to decrease following the high-volume strength endurance block and increase following the strength/power and peaking blocks. Just because these findings aren't groundbreaking doesn't mean they aren't interesting. The question now becomes: If adaptations are specific to the training block, is it necessary to run training blocks

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

Table 1 Subject characteristics

Sex	Age (years)	Height (cm)	Body mass (kg)	Boday fat (%)	RT age (years)	WL age (years)	Snatch 1RM (kg)	C&J 1RM (kg)
Males (n=5)	22.4 ± 1.6	169.9 ± 3.8	83.7 ± 7.0	11.7 ± 3.0	5.4 ± 1.0	3.8 ± 0.4	117.6 ± 8.2	147.8 ± 13.6
Females (n=4)	20.5 ± 2.6	157.3 ± 4.0	57.6 ± 7.2	16.8 ± 1.9	7.0 ± 3.1	6.5 ± 3.2	69.3 ± 8.0	90.8 ± 10.1

RT Age = Years engaged in resistance training, WL Age = Years engaged in Weightlifting training specifically, C&J = Clean and Jerk, 1RM = One-Repetition Maximum

that are wholly unspecific to the main goal (i.e. maximal strength and power) for either mechanistic or practical reasons? To answer this question, we must speculate, as is often the case with long-term program design. This article will examine these results and discuss the broader scope of periodization for strength development, along with the relationship between hypertrophy and strength in an attempt to answer this question.

Purpose and Hypotheses

Purpose

The purpose of this study was to assess muscle hypertrophy and changes in rate of force development following each training block with a specific focus over a full macrocycle in competitive weightlifters.

Hypotheses

A formal hypothesis was not given. However, from reading the introduction of the paper, it can reasonably be assumed that the authors expected adaptations to be specific to the block of training. For example, greater hypertrophy was expected after the training block with the greatest volume.

Subjects and Methods

Subjects

Nine competitive collegiate weightlifters participated. The lifters had been engaged in general resistance training for about six years, and had been training for weightlifting specifically for about five years. The lifters had all previously competed at very high levels in the sport, ranging from the university national level to the international level as a junior in weightlifting. The remainder of available subject details are in Table 1.

Study Design

This study was observational in nature. This means that subjects were just observed over the course of the training program, but weren't allocated to different groups for a monitored intervention. There were three different training blocks, each with a different focus, and outcome measures were tested before and after each training block. All athletes performed a strength endurance block, a strength and power block, and a peaking block, which consisted of a one-week overreach followed by a three-week taper prior to competition. However, each sub-

Table 2 Exercises performed in each training session

Day	Strength-endurance	Strength-power	Peak/Taper
Monday/Thursday	**AM** Back squat **PM** Push press Press from split DB press	**AM** Back squat **PM** Push press Jerk lockout BTN press DB press	**AM** Back squat* **PM** Jerk Dead stop parallel Squat** BTN press DB press*
Wednesday	**AM** Snatch tech CGSS CG pull-floor **PM** Snatch tech CGSS CG pull-pp CG SLDL DB row	**AM** Snatch tech CGSS CG pull-floor **PM** Snatch tech CGSS CG pull-knee CG SLDL CG bent over row	**AM** Snatch tech CGSS CG pull-pp **PM** Snatch tech SGSS SG pull-floor CG SLDL* DB row*
Saturday	Snatch tech SGSS Snatch C&J SG SLDL DB row	Snatch tech SGSS Snatch C&J SG SLDL SG Bent over row	Snatch tech SGSS Snatch C&J SG SLDL DB row

AM/PM = Morning/Afternoon, DB = Dumbbell, BTN = Behind the Neck Press, CGSS = Clean Grip Shoulder Shrug, SLDL = Stiff-Legged Deadlift, SG = Snatch Grip, SGSS = Snatch Grip Shoulder Shrug, C&J = Clean and Jerk
** = This exercise was not performed during the last week of the taper*
*** = This exercise was only used on that day and time during the one-week overreach*

ject's total macrocycle (i.e. entire duration of training for the study) length was not the same, as training was planned differently depending on when the lifter was competing and the lifter's training age.

Training Protocol

Importantly, the researchers did not write or adjust the training programs. Rather the weightlifters had a "nationally certified coach" write their training. Although the paper wasn't too specific, it appears that a training template was created by the nationally certified coach for all athletes and then adjusted for them individually by this coach. The lifters trained four days per week (Monday, Wednesday, Thursday, Saturday), but performed seven total sessions per week as Mondays, Wednesdays, and Thursdays were two-a-days. The exercises performed can be seen in Table 2, and the sets and reps for each exercise are in Table 3.

Table 3 Sets, reps, and intensity during each specific block

Phase	Week	Sets x reps	Daily intensities (M, W, Th, S)
SE	1	3 x 10	M, M, VL, VL
SE	2	3 x 10	MH, MH, L, L
SE	3	3 x 10	L, L, VL, VL
SP	1	3 x 5 (1 x 5)	M, M, L, VL
SP	2	3 x 5 (1 x 5)	MH, MH, L, VL
SP	3	3 x 3 (1 x 5)	H, H, L, VL
SP	4	3 x 2 (1 x 5)	MH, L, VL, VL
PT	1	5 x 5 (1 x 5)	MH, M, L, VL
PT	2	3 x 3 (1 x 5)	M, MH, VL, VL
PT	3	3 x 3 (1 x 5)	MH, M, VL, VL
PT	4	3 x 2 (1 x 5)	ML, L, VL, Meet

SE = Strength Endurance Block, SP = Strength Power Block, PT = Peak Taper Block
The 1X5 in parentheses indicates that a drop set was performed for 1 set of 5 at 60% of one-repetition maximum (1RM) after the other working sets.
VL (Very Light) = 65-70% of 1RM, L (Light) = 70-75% of 1RM, ML (Medium Light) = (75-80%),
M (Medium) = 80-85% of 1RM, MH (Medium Heavy) = 85-90% of 1RM, H (Heavy) = 90-95% of 1RM

Outcome Measures

Rate of force development was measured by having the subjects perform the isometric mid-thigh pull on force plates. Rate of force development was measured during the pull in milliseconds in the following time bands: 0-50, 0-100, 0-150, 0-200, and 0-250 ms. Peak force was also assessed on the isometric mid-thigh pull. Ultrasound was used to measure both cross-sectional area and muscle thickness for hypertrophy.

Findings

Rate of Force Development and Peak Force

According to the main statistical analysis (analysis of variance – ANOVA), there was no statistically significant

Table 4 Effect sizes for changes in variables from pre- to post-each block and for the entire macrocycle

Measure	Strength endurance	Strength power	Peaking	Total macrocycle
Rate of force development 0-50ms	-0.04 Trivial decrease	0.32 Small increase	0.44 Small increase	0.47 Small increase
Rate of force development 0-100ms	-0.11 Trivial decrease	0.41 Small increase	0.39 Small increase	0.39 Small increase
Rate of force development 0-150ms	-0.14 Trivial decrease	0.33 Small increase	0.22 Small increase	0.23 Small increase
Rate of force development 0-200ms	-0.14 Trivial decrease	0.33 Small increase	0.02 Trivial increase	0.04 Trivial increase
Rate of force development 0-250ms	-0.12 Trivial decrease	0.27 Small increase	-0.19 Trivial decrease	-0.13 Trivial decrease
Cross-sectional area	0.31* Small increase	-0.13 Trivial decrease	0.00 No change	0.09 Trivial increase
Muscle thickness	0.37 Small increase	-0.24 Small decrease	-0.02 Trivial decrease	0.14 Trivial increase

Effect size data shows that changes tended to be specific to the block. In other words, during the strength endurance block (highest volume) there tended to be favorable hypertrophy and decreased force development, whereas the opposite was true in the strength and power focused block.
**Significant increase (in terms of ANOVA, $p<0.05$) during that training block.*
#Significant decrease (in terms of ANOVA, $p<0.05$) during that training block.
Please note, that the effect sizes reported in the actual paper are different than the ones reported here. The actual study used standard deviation of the change scores to calculate effect size, whereas we in MASS believe you should use pre-testing standard deviations, thus I recalculted (thanks to Greg realizing this) the effect sizes.

change in any rate of force development time band during any training block. This isn't surprising since the lifters were pretty well-trained. However, small, but meaningful changes existed in terms of effect sizes. In brief, the specific adaptations to imposed demands (SAID) principle seemed to hold true. Rate of force development tended to see small increases following the strength and power and peaking blocks, but no change (or trivial decreases) following the highest volume block of strength endurance. Effect sizes did not show any meaningful change in peak force of the isometric mid-thigh pull during any training block.

Hypertrophy

Just as with rate of force development, hypertrophy results fell in line with the SAID principle. Cross-sectional area and muscle thickness tended to increase following the strength endurance block and tended to decrease following the strength/power and peaking blocks.

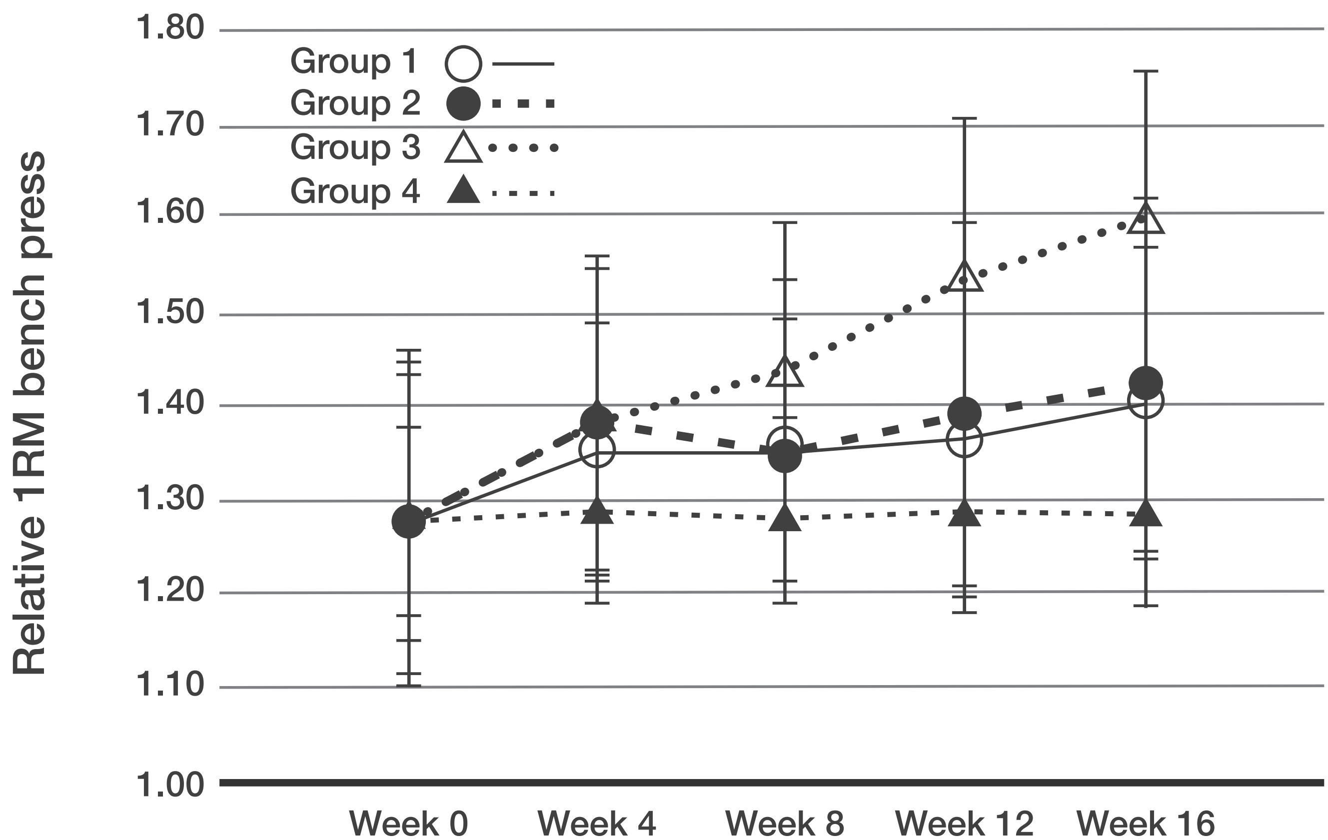
Effect sizes and their interpretations for changes in the rate of force development and hypertrophy following each block are in Table 4.

Interpretation

Perhaps the most important principle within strength sport or even sport in general is the principle of specificity. In our context, this means that if you want to get better at something, you need to train in a specific manner. Thus, if you are looking to increase squat strength, you squat, and you squat heavy. I'm not saying this is the only thing you do, but for most, it is the number one thing that you do. In its simplest form, this study shows the effects of specificity. Subjects increased rate of force development during strength blocks and incurred hypertrophy during the volume (strength endurance) block – not shocking or new. Further, the results of this study were highly individual, with some subjects showing small increases in hypertrophy and rate of force development throughout the entire macrocycle, while a few subjects saw small decreases. The individual responses could be due to a myriad of factors. First, the amount of training volume a lifter performs should be individualized (and is hard to get exactly correct), thus the magnitude of training volume may not have been optimal for some. Additionally, molecular factors such as satellite cell number and myonuclei per myofiber may be predictive of hypertrophy (2); therefore, these factors could have made some lifters less susceptible to positive changes in muscle morphology. Although as all lifters were reasonably competitive, I don't imagine the molecular factors to be the most likely rationale for the individual response. Lastly, as we have reviewed previously, the rate of force development is enhanced in some, with traditional strength-type training, while in others it is only enhanced with power-type training or speed work (3). So, it is possible that some subjects could have benefited from more speed work. Of course, I don't know that for sure, but all of the above are possibilities that could explain the individual responses. We should also be mindful of individual responses, as they occur in every training study. When we look at mean data, we are looking at what works better for most people, but this is not necessarily what works best for everyone. I have collected data for training studies and watched some lifters add 20kg to a squat or bench press during an eight-week study, while others have seen a 5kg decrease. This kind of result is not really picked up by the reader when looking at mean data.

Other than the individual responses, these findings aren't really that interesting in and of themselves. However, the reviewed study presents us with a good opportunity to discuss programming and periodization as a whole from a specificity standpoint. For starters, the adaptations in the study were specific to the block, yet we still recommend periodization. Let's use this article as an opportunity to discuss why we use periodization as opposed to just ultra-specific training year-round. However, at the same time, we'll discuss how to improve upon the strict block periodized model used in the presently reviewed study so that we can maintain some semblance of specificity to avoid the regressions seen in muscle size and rate of force development during nonspecific blocks. We'll focus on strength in the following discussion rather than rate of force development, since that is what's most

Figure 1 Periodized vs. non-periodized training from Willougby 1993



From Willougby 1993 (4)
** = Significantly greater than baseline; # = Significantly greater than both groups 1 and 2*
Group 1 performed 10 reps the entire training cycle. Group 2 performed 8 reps the entire training cycle. Group 3 periodized training. Group 4 was a control group who did not train and only did the testing sessions.

relevant to the MASS reader. As you continue, please note that this section ended up much longer than I intended, but I wanted to take advantage of the opportunity to expand upon periodization since we have not discussed periodization in great depth in MASS for about two years. If you would like to see the now two-year-old video series on the topic, here are the three parts: one, two, three.

Periodization

So, with the principle of specificity playing out as it did in this study, let's open up a broader discussion centered on the following questions in the context of training for strength: Why periodize training? If strength is the main goal, why not just train with low reps and high loads all the time?

First, the rationale for periodization is based on the evidence that periodized

Table 5 Example of adding volume to twice per week daily max program

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Squat Single @9-9.5 RPE 3x8 @70% of 1RM	Off	Bench Single @9-9.5 RPE 3x8 @70% of 1RM Deadlift Single @9-10 RPE 4x5 @70% of 1RM	Off	Squat Single @9-9.5 RPE 3x5 @77.5% of 1RM	Bench Single @9-9.5 RPE 3x5 @77.5% of 1RM Deadlift Single @9-10 RPE 4x2 @80% of 1RM

Note: This is just an example. There are many ways to configure this including experimenting with different frequencies, different volume configurations, and using RPE or velocity to prescribe volume work instead of percentages.
1RM = One-Repetition Maximum; RPE = Rating of Perceived Exertion

training tends to outperform nonperiodized training, and the lack of evidence supporting highly specific training year-around (though, to be clear, absence of evidence is not evidence of absence). To kick us off, a meta-analysis from Williams et al 2017 (reviewed by Greg) shows a benefit for periodized training versus non-periodized training (4), as does Greg's in-house Stronger by Science meta-analysis (5). This data in favor of periodization means that manipulating training variables in some capacity is a good idea to maximize strength versus simply keeping training variables static, even if that "static" training is highly specific. A classic example of this is a 16-week study from Willougby in 1993 (6). In that 16-week training cycle, individuals who decreased their reps and increased intensity on the squat and bench every four weeks (sets of 10 at 79% 1RM for the first four weeks, followed by sets of 8 at 85%, sets of 6 at 88%, and sets of 4 at 92%) gained more strength than subjects who performed either only 10 reps or only 8 reps per set for the entire 16 weeks. This study is just one example of the studies included in the meta-analysis, and this type of study is not immune to limitations and valid criticism. One criticism of this type of study is that it was only 16 weeks long, which is not truly long term. I agree with that criticism, and this is typical of almost all periodization studies. Another criticism is that the design is biased in favor of periodization, as the post-test came just after the periodized group was performing sets of 4 reps at ~92%, while the 8-rep, non-periodized group trained at ~83% for the final four weeks; thus, specificity to the test was greater in the periodized group versus the non-periodized group just before the test. However, to counter this, at week 8 of the study, bench press strength was already significantly greater in the periodized group versus the 8-rep-only group (Figure 1), even though the periodized group had trained at a lower average intensity than the 8 rep group to this point. I would also add that while the criticism of greater specificity to the test at the end of the study is not invalid in and of itself, the decrease in volume and increase in intensity at the end of a training cycle is simply an inherent benefit of periodization. So rather than criticizing the study design for that, I would say it's just a point in the column of periodization.

Nonetheless, if greater specificity is better toward the end of the training cycle, then why not simply train with extreme specificity all the time? In other words, how about maxing or near-maxing all the time? We know that daily 1RM squat training is effective to increase squat 1RM in competitive lifters over the course of 37 days (7), and that working toward a near max just twice a week is just as good for strength as typical volume training in novices over eight weeks on the leg extension and chest press (8). So, why not just use these specific strategies all the time? The short answer is that we don't have experimental evidence to show that these strategies are better than some type of periodization (linear, daily undulating, or block) over the long term. Sure, daily 1RM training increased squat 1RM by 10.8%, 9.5%, and 5.8% in three different well-trained lifters over 37 days, which translated to a 220kg squat increasing to 241kg for one lifter. While that type of increase would make most of us ecstatic in a little over a month (or even over the course of a year or two), we don't yet know how this plays out over the longer term from either a mechanistic or practical perspective. Importantly, MASS has previously discussed in detail that long-term daily 1RM training may be difficult from a practical perspective. However, performing a near max a couple times a week, as mentioned previously, seems likely to be more sustainable than every day, and is still much more specific to 1RM strength than a normal periodized plan. However, there is no long-term evidence showing the efficacy of this, and we do have longitudinal studies showing the benefits of periodization. So, I'm not sure if the specificity of maxing a couple times per week is enough to maximize long-term strength by itself. That's the most interesting question to me: not just what's going to work (because almost

IF DECIDING BETWEEN JUST MAXING A FEW TIMES PER WEEK AND USING A PERIODIZED PROGRAM WITH VOLUME BLOCKS, I'D CHOOSE THE PERIODIZED PROGRAM (WHEN THINKING LONG TERM) BECAUSE DATA EXISTS SUGGESTING THAT 70% OF THE VARIANCE IN STRENGTH CAN BE EXPLAINED BY MUSCLE SIZE.

anything works), but what maximizes strength potential over the long-term?

Perhaps we can get the best of both worlds. For example, if someone maxed just a couple times per week, it would be pretty easy to perform some normal volume training along with it. We know there is a relationship between volume and strength adaptation from a meta-analysis (9), so it seems a reasonably good idea to add some volume after the max work if choosing to just max a couple times per week as your training program. Granted, the studies in the Ralston et al meta-analysis didn't examine if adding volume to a max-type program was beneficial; rather, it just showed that when a "normal" strength training program is done, there is a dose-response relationship to a point, but this does show that max strength can be impacted by both highly specific training (i.e. 1RM training) and by submaximal training (i.e. 60-90% of 1RM). Therefore, if maxing two or three times per week, it makes sense to try to take advantage of both methods of adding strength, whereas this might be more difficult in a daily 1RM training program. An example of setting this up can be seen in Table 5.

Additionally, it makes sense to perform added volume when we consider the relationship between muscle hypertrophy and strength. While this relationship has been questioned by some (10) – and has been the basis for some to also question the necessity of periodization for strength (11) – we do have data showing strong relationships between hypertrophy and strength adaptation (12). The basis of the argument that hypertrophy is not a causative factor in strength adaptation is that there is not long-term experimental evidence showing a causative relationship. While that is a fair and healthy skepticism, it is also fair to say that absence of evidence does not mean evidence of absence. So, perhaps from a purely scientific perspective, it's an okay position to state that we don't know if increasing muscle size causes an increase in muscle strength, but from a practical perspective, we need to hedge our bets one way or the other because we must decide how to train. So, if deciding between just maxing a few times per week and using a periodized program with volume blocks, I'd choose the periodized program (when thinking long term) because data exists suggesting that 70% of the variance in strength can be

PERIODIZATION REPRESENTS LONG-TERM TRENDS IN TRAINING VARIABLES, WHILE PROGRAMMING IS THE SETS AND REPS THAT YOU ARE DOING TODAY.

Table 6 Sample volume blocks transitioning into intensity integrating DUP into a block fashion with a linear framework

Volume block 1 (5-8 weeks)					
Monday	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**
Squat 3x10 @65% or 5-7 RPE	Bench 3X10 @65% or 5-7RPE	Squat 4X8 @70% or 5-7RPE	Bench 4X8 @70% or 5-7RPE	Squat 4x6 @75% or 5-7 RPE	Bench 4X6 @75% or 5-7RPE
Deadlift 8x1 @75%				Deadlift 4x1 @85%	
Back and Biceps Assistance 1-2 exercises of each 3-5 sets of 15-20 reps @5-8RPE	Chest, Shoulders, and Triceps Assistance 1-2 exercises of each 3-5 sets of 15-20 reps @5-8RPE	Back Assistance 1-2 exercises for 3-5 sets of 10-15 reps @5-8RPE	Chest, Shoulders, and Triceps Assistance 1-2 exercises of each 3-5 sets of 10-15 reps @5-8RPE	Back and Biceps Assistance 1-2 exercises for 3-5 sets of 6-10 reps @5-8RPE	Chest, Shoulders, and Triceps Assistance 1-2 exercises of each 3-5 sets of 6-10 reps @5-8RPE
Volume block 2 (5-8 weeks)					
Monday	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**
Squat 3x8 @72.5% or 6-8 RPE	Bench 3X8 @72.5% or 6-8RPE	Squat 3x6 @77.5% or 6-8 RPE	Bench 3X6 @77.5% or 6-8RPE	Squat 4x4 @82.5% or 6-8 RPE	Bench 4X4 @82.5% or 6-8RPE
Deadlift 7x1 @80%				Deadlift 3x1 @87.5%	
Back and Biceps Assistance 1-2 exercises of each 3-5 sets of 12-15 reps @5-8RPE	Chest, Shoulders, and Triceps Assistance 1-2 exercises of each 3-5 sets of 12-15 reps @5-8RPE	Back Assistance 1-2 exercises for 3-5 sets of 8-12 reps @5-8RPE	Chest, Shoulders, and Triceps Assistance 1-2 exercises of each 3-5 sets of 8-12 reps @5-8RPE	Back Assistance 1-2 exercises for 3-5 sets of 6-8 reps @5-8RPE	Chest, Shoulders, and Triceps Assistance 1-2 exercises of each 3-5 sets of 6-8 reps @5-8RPE
Intensity block 1 (3-5 weeks)					
Monday	**Tuesday**	**Wednesday**	**Thursday**	**Friday**	**Saturday**
Squat 3X5 @80% or 7-9RPE	Bench 3X5 @80% or 7-9RPE	Squat 3X3 @85-87.5% or 7-9RPE	Bench 3X3 @85-87.5% or 7-9RPE	Squat Double @9RPE	Bench Double @9RPE
Deadlift 3x1 @85%		Deadlift 1x1 @87.5%		Deadlift 3x1 @90%	
Back and Biceps Assistance 1 exercise of each 3 sets of 10-12 reps @7-9RPE	Chest, Shoulders, and Triceps Assistance 1 exercise of each 3 sets of 8-10 reps @7-9RPE	Back and Biceps Assistance 1 exercise of each 3 sets of 8-10 reps @7-9RPE	Chest, Shoulders, and Triceps Assistance 1 exercise of each 3 sets of 8-10 reps @7-9RPE	Back and Biceps Assistance 1 exercise of each 3 sets of 6-8 reps @7-9RPE	Chest, Shoulders, and Triceps Assistance 1 exercise of each 3 sets of 6-8 reps @7-9RPE

Note: Please be aware that this is just an example and of course other sets and reps should be performed. Additionally, as we have covered in MASS, there are many ways to program load (percentage, RPE, and velocity) all of which are valid and many ways to progress load, all of which are valid. The above is just a template. This template can be used, but other articles and videos should be used to fill in those gaps. Further, please be aware that the magnitude of volume and frequency needed is individual, thus the above may be too much for some and not enough for others. The above is just a guide to show how DUP can be implemented within a specific block, yet still follows a linear pattern over the long-term.

explained by muscle size (12). Further, it seems logical that adding more sarcomeres (i.e. more contractile units), which contributes to hypertrophy, is a benefit for long-term strength. It is possible that a multi-year study will show my above argument to be wrong, but since we have to choose how to train (i.e. periodization or solely specificity), then I would hedge my bets in favor of the arguments above and would choose periodization from both mechanistic (hypertrophy contributes to strength) and practical (it's more sustainable) perspectives for long-term strength.

Despite the available evidence pointing toward periodization and not solely specificity, it does seem logical that above all, specificity is still the most important factor. Therefore, we should determine how to make our periodized programs as specific as possible, if strength is our goal. This allows us to address what I believe to be a limitation of the purely block periodized design implemented in the presently reviewed study (1). When aiming to make your periodized program as specific as possible, it's important to look at the various "types" of periodization. The main types analyzed in the strength literature are linear and daily undulating periodization (DUP). Linear periodization is as described above in the Willoughby study (6), and DUP simply involves multiple training sessions per week (typically two or three, but this can really be any frequency) and altering the reps between those days (13, 14). This might mean training a muscle group three times per week with 10 reps on Monday, 8 reps on Wednesday, and 6 reps on Friday, as just one example. In the Willoughby study of linear periodization, it took 8 weeks before subjects were performing 6 reps, which meant 8 weeks of unspecific training (i.e. lower intensities and higher reps). In a DUP model, you can ensure that heavy-ish training occurs about once per week, even if volume is the main focus of the block. Thus, a DUP program has some element of specificity at all times. Of course, the above are just examples and you could use a wider undulation pattern of 10, 6, 2 and then you would be even more specific to maximal strength every week. Importantly, the available experimental evidence supports DUP versus LP when presented in a binary fashion for maximal strength (5). I say "when presented in a binary fashion," because as I've discussed before, these really aren't mutually exclusive concepts.

In reality, linear periodization, DUP, and block periodization can and probably should all be integrated. First, periodization represents long-term trends in training variables, while programming is the sets and reps that you are doing today. So, if DUP allows you to alter the reps and intensities you are using within a week, that is really just a programming strategy. In the textbook definition of DUP, volume still decreases over time and intensity still increases similar to linear periodization; therefore, it's probably more appropriate to look at DUP as a programming strategy that fits into the broader scope of linear and block periodization. In the presently reviewed study, the athletes performed blocks, which had distinct phases. You can still do this while using DUP within weeks (i.e. a programming strategy) and while organizing your training into blocks that fit a linear or traditional periodization trend. Table 6 provides a practical example of this integrated strategy. In the table, please note how the average RPE increases from block to block. As intensity increases and volume decreases, proximity to failure also increases.

As you can see in Table 6, just because reps are "undulated" within a week, this doesn't mean that a specific focus of the entire training block doesn't still exist. Clearly, in this example of training with no less than six reps, volume is still the priority, so the training block is focused on hypertrophy. However, in the present study and in the aforementioned Willoughby study, the subjects went full mesocycles without training any lower than 10 reps, which for most of us means training around 70% of 1RM, which is quite unspecific. When we started this discussion, we said that specificity was still of paramount importance, even if it wasn't the only factor of importance. Indeed, a within-week DUP design allows for more specificity of strength training year-round while still organizing training into blocks. Ultimately, you of course don't have to always use this model. For example, once you are a month or so out from competition, you can move to a frequent maxing strategy, as we discussed earlier.

Another way to incorporate specificity throughout a macrocycle when training for strength is to simply add a heavy-ish single prior to the volume work (examined previously in MASS). Therefore, if you are in a volume training block similar to the examples above, you could simply work up to a single at an 8RPE (about 88-92% of 1RM) prior to your working sets. If you did this once a week for most of the year, you could still venture through the entire integrated macrocycle as sketched out conceptually above, but this would allow you to keep some semblance of true specificity year-round. Although the reviewed study uses strict block periodization, it seems there are other strategies for hypertrophy and strength, which can be used to maintain specificity for longer rather than for just one specific block. If hypertrophy is your main goal, we have laid out strategies before in MASS that use wider undulation patterns to maintain volume even when you are training for strength.

As a side note, the literature does not reveal that periodization is important for hypertrophy versus non-periodized programs (15), but the periodized studies included in this meta-analysis had the programs written aimed at maximizing strength and not hypertrophy. Some type of periodization may be a good idea for hypertrophy, but perhaps volume should increase over a macrocycle instead of decrease. Wouldn't that be more specific to maximizing muscle growth? I'm not saying that is definitely the case; I'm just saying we don't truly have evidence to answer that question.

It must be stated that the above is not comprehensive; that is not really possible in one MASS article. However, it should drive home the point that we need to continually evolve our understanding of periodization. Further, while specificity is not the only factor, it seems foolish to ignore it for full training blocks at a time. The strategies presented above try to balance specificity while still providing distinct training phases, which seems to be a step for-

APPLICATION AND TAKEAWAYS

1. Throughout a macrocycle, a group of competitive weightlifters, comprised of both men and women, tended to only increase specific qualities (rate of force development and muscle size) in training blocks specifically designed for each specific adaptation. This illustrates the importance of the principle of specificity.
2. It is important to ask the question "why periodize?" if specificity is perhaps the most important principle. Data does indeed support periodization, but that doesn't mean we should neglect specific training year-round.
3. In the context of strength, it probably makes sense to never veer completely away from your main goal. This means that heavy singles can be incorporated into volume blocks and periodization strategies can be integrated to allow for within-week fluctuations of volume and intensity in an effort to maintain some semblance of specificity even when in a volume block.

ward from traditional block periodization when programming for strength adaptation.

Next Steps

First, to tie our discussion back into the reviewed study from Suarez et al (1), we should use this as a lesson to understand how to conceptually extrapolate from studies. In reality, we know very little for sure. However, all the above interpretation does is highlight that the present study reaffirms the principle of specificity and then extrapolates how to apply that for strength adaptation over the long-term. Going forward, we simply need truly long-term data in well-trained lifters (isn't that the case with everything?), which compares periodized programming versus ultra-specific training such as frequent maxing. For now though, I'd be interested to see a mesocycle-length study comparing the same two periodized programs but have one group perform a heavy-ish single a couple times per week before the volume. In that study, researchers could not only see if strength was greater in the group that included the heavy singles, but also if the heavy singles affected the volume work (they shouldn't). The researchers could administer questionnaires regarding enjoyment and sustainability of the practice. That could be a feasible step in the right direction.

References

1. Suarez DG, Mizuguchi S, Hornsby WG, Cunanan AJ, Marsh DJ, Stone MH. Phase-Specific Changes in Rate of Force Development and Muscle Morphology Throughout a Block Periodized Training Cycle in Weightlifters. Sports. 2019 Jun;7(6):129.
2. Petrella JK, Kim JS, Mayhew DL, Cross JM, Bamman MM. Potent myofiber hypertrophy during resistance training in humans is associated with satellite cell-mediated myonuclear addition: a cluster analysis. Journal of applied physiology. 2008 Jun;104(6):1736-42.
3. Peltonen H, Walker S, Hackney AC, Avela J, Häkkinen K. Increased rate of force development during periodized maximum strength and power training is highly individual. European journal of applied physiology. 2018 May 1;118(5):1033-42.
4. Williams TD, Tolusso DV, Fedewa MV, Esco MR. Comparison of periodized and non-periodized resistance training on maximal strength: a meta-analysis. Sports medicine. 2017 Oct 1;47(10):2083-100.
5. Nuckols G. Periodization: What the Data Say. Stronger by Science, 2018 Jan.
6. Willoughby DS. The effects of mesocycle-length weight training programs involving periodization and partially equated volumes on upper and lower body strength. The Journal of Strength & Conditioning Research. 1993 Feb 1;7(1):2-8.
7. Zourdos MC, Dolan C, Quiles JM, Klemp A, Jo E, Loenneke JP, Blanco R, Whitehurst M. Efficacy of daily one-repetition maximum training in well-trained powerlifters and weightlifters: a case series. Nutrición Hospitalaria. 2016;33(2):437-43.
8. Mattocks KT, Buckner SL, Jessee MB, Dankel SJ, Mouser JG, Loenneke JP. Practicing the Test Produces Strength Equivalent to Higher Volume Training. Medicine and science in sports and exercise. 2017 Sep;49(9):1945-54.
9. Ralston GW, Kilgore L, Wyatt FB, Baker JS. The effect of weekly set volume on strength gain: a meta-analysis. Sports Medicine. 2017 Dec 1;47(12):2585-601.
10. Loenneke JP, Dankel SJ, Bell ZW, Buckner SL, Mattocks KT, Jessee MB, Abe T. Is muscle growth a mechanism for increasing strength? Medical hypotheses. 2019 Apr 1;125:51-6.
11. Mattocks KT, Dankel SJ, Buckner SL, Jessee MB, Counts BR, Mouser JG, Laurentino GC, Loenneke JP. Periodization: what is it good for? Journal of Trainology. 2016 Apr 13;5(1):6-12.
12. Trezise J, Collier N, Blazevich AJ. Anatomical and neuromuscular variables strongly predict maximum knee extension torque in healthy men. European journal of applied physiology. 2016 Jun 1;116(6):1159-77.
13. Miranda F, Simão R, Rhea M, Bunker D, Prestes J, Leite RD, Miranda H, de Salles BF, Novaes J. Effects of linear vs. daily undulatory periodized resistance training on maximal and submaximal strength gains. The Journal of strength & conditioning research. 2011 Jul 1;25(7):1824-30.
14. Zourdos MC, Jo E, Khamoui AV, Lee SR, Park BS, Ormsbee MJ, Panton LB, Contreras RJ, Kim JS. Modified daily undulating periodization model produces greater performance than a traditional configuration in powerlifters. The Journal of Strength & Conditioning Research. 2016 Mar 1;30(3):784-91.

15. Grgic J, Mikulic P, Podnar H, Pedisic Z. Effects of linear and daily undulating periodized resistance training programs on measures of muscle hypertrophy: a systematic review and meta-analysis. PeerJ. 2017 Aug 22;5:e3695.

Study Reviewed: Ultra-Processed Diets Cause Excess Calorie Intake and Weight Gain: An Inpatient Randomized Controlled Trial of Ad Libitum Food Intake. Hall et al. (2019)

The Poptart Problem: Processed Foods and Overeating

BY ERIC HELMS

It's oft-repeated in our community that so long as energy, macros (especially protein), and fiber are matched, the amount of processed food in your diet is inconsequential for body composition. But is this true when it comes to how such a diet impacts your habitual energy intake and ad libitum consumption of food?

KEY POINTS

1. In a metabolic ward, 20 adults were *presented* diets consisting of unprocessed or "ultra-processed" food matched for energy, macronutrients, sugar, fat, and fiber for two weeks each. However, while presented with diets matched for these variables, during each diet, participants could consume as much as they wanted.
2. During the processed diet, participants ate ~500kcal per day more than during the unprocessed diet, increasing body mass and fat mass from baseline, while during the unprocessed diet, they lost body mass and fat mass. More carbohydrate and fat were consumed at lunch and breakfast, absolute protein intake remained stable, and both diets had similar energy densities. However, specific foods had higher energy density, which the researchers controlled (perhaps unsuccessfully due to using liquid) by dissolving fiber in the processed diet beverages.
3. Participants ate processed foods faster and the satiety hormone PYY increased and the hunger hormone ghrelin decreased compared to baseline during the unprocessed diet. Further, PYY was significantly higher during the unprocessed compared to the processed diet. Thus, processed foods are consumed in excess for multiple reasons: they're eaten faster, suppress appetite less, and require more energy be consumed to achieve a similar protein and food mass – both shown to regulate energy intake – as compared to unprocessed foods.

The diet wars have intensified. Blame for obesity shifts across a gamut from fat, carbs, sugar, processed foods, animal products, and back again. As discussed (article here), the battleground outcome of carbs versus fat is the least promising at the population level in terms of providing a solution. Differences in low fat versus low carb interventions are clinically insignificant at the group level (though they often matter for individuals). This is just one example of the failed attempts to find a "smoking gun" in the obesity epidemic. As you'll see in my video this month, weight management is better accomplished with multi- versus single-variable interventions. Rather than a smoking gun, there is a firing squad of mechanisms driving weight gain. In this first causal analysis of processed foods as a contributor to weight gain (1), researchers examined a multitude of mechanisms related to energy consumption and body composition

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

change. Researchers used a tightly controlled metabolic ward trial that mimicked real-life using a free-eating model. Specifically, 20 adults were *presented* diets dominated by processed or unprocessed foods for two weeks apiece, and the diets as presented were matched for energy, macronutrients, sugar, fat, and fiber. However, the subjects were able to consume as much as they desired of the food presented. During the processed diet, participants ate faster and consumed more energy, consuming greater amounts of carbs and fat but similar protein compared to the unprocessed diet; therefore, they gained weight and fat mass when consuming the processed diet, but lost weight and fat mass while consuming the unprocessed diet. Additionally, hormone markers of hunger were lower compared to baseline during the unprocessed diet, and satiety was higher during the unprocessed compared to the processed diet. In this article, we'll discuss which aspects of the diets could be controlled by the researchers, and which they could only control to some degree. This discussion hints at the inherent issues of processed diets, and why they may be among the deadliest of the firing squad.

Purpose and Hypotheses

Purpose

The purpose of this study was to determine if processed foods play a causal role in ad libitum (at will or, as desired, "free eating") energy intake, and subsequent changes in body mass.

Hypotheses

The authors did not present hypotheses, but stated there is compelling indirect evidence, albeit no direct causal evidence, that processed diets might lead to energy overconsumption and weight gain.

Subjects and Methods

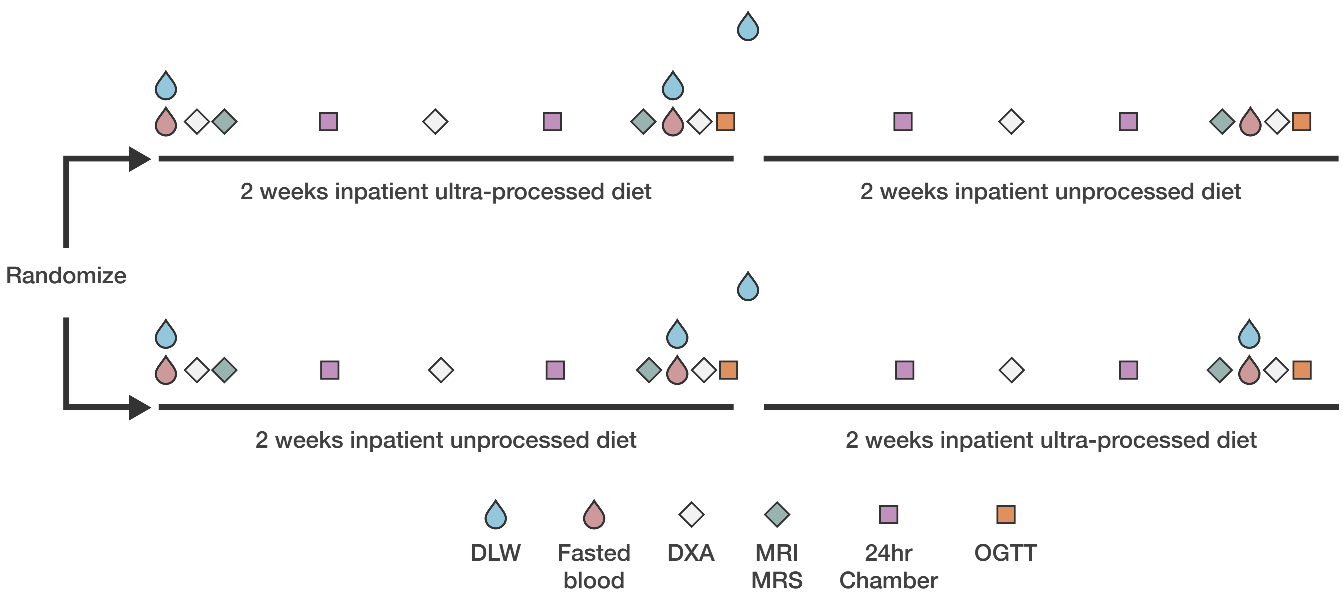
Subjects

20 (10 male, 10 female) weight-stable adults (31.2 ± 1.6 years; BMI = 27 ± 1.5 kg/m^2) participated in this randomized, controlled, two-week crossover trial.

Methods

Participants lived in a metabolic ward research center continuously for 28 days, during which time they were randomly allocated to either the processed or unprocessed diet condition for two weeks, immediately followed by the opposite condition. Hormone and metabolic data from blood samples, body composition measurements via DXA, 24-hour energy expenditure changes in a metabolic chamber, average energy expenditure from doubly labeled water, and visceral fat changes from MRI were all collected. Figure 1 provides an overview of

Figure 1 Overview of study design



Twenty adults were confined to the metabolic ward at the NIH Clinical Center, where they were randomized to consumed either an ultra-processed or unprocessed diet for 2 consecutive weeks followed immediately by the alternate diet. Every week, subjects spent 1 day residing in a respiratory chamber to measure energy expenditure, respiratory quotient, and sleeping energy expenditure. Average energy expenditure during each diet period was measured by the doubly laced water (DLW) method. Body composition was measured by dual-energy X-ray absorptiometry (DXA) and liver fat was measured by magnetic resonance imaging / spectroscopy (MRI/MRS). Glucose concentrations were measured following a 75 g oral glucose tolerance test (OGTT).

the study design and the timing of these measurements.

Additionally, participants assessed aspects of their eating and food experience via visual analog scales. They rated hunger, fullness, satisfaction, eating capacity, familiarity, and pleasantness, and the researchers tracked how much total food was consumed, which foods were consumed at which meals, and the rate that the participants consumed their food.

Participants were given three meals daily and told to eat as much as they wanted, with each meal period lasting up to 60 minutes. Subjects also had access to snacks. The subjects were presented food for each meal; if the subjects ate all of the food they were presented, they would have consumed approximately 3900kcal/day, and the diets would have been matched for energy, macronutrients, fiber, total sugar, sodium, and energy density, differing only by the percentage of energy from processed or unprocessed foods as defined by the NOVA classification (2). The subjects were free to eat as much or as little of each presented food as they pleased, meaning the actual diets consumed were free to differ substantially. Briefly, the NOVA system is a peer-reviewed published system using a checklist to group foods according to the extent and purpose of industrial processing. This includes processes and ingredients used to manufacture ultra-processed foods which are designed to create low-cost, long shelf-life, ready-to-consume, hyper-palatable products likely to displace unprocessed or minimally processed foods. How much of the presented diets were consumed, and whether additional food was consumed via snacks, was ad libitum. Diet specifics are shown in Table 1.

Findings

Energy, energy macronutrient composition, energy distribution across meals, hunger and satiety scores, and eating speed are shown in Figure 2. In short, during the processed diet, participants ate ~500kcal more on average per day (also shown in Table 2) and ate at a faster rate, while rating hunger and satiety similarly on both diets. Additional calories were consumed during the unprocessed diet via carbohydrate and fat, while reaching the same protein intake. The subjects primarily consumed more at breakfast and lunch during the unprocessed diet, while energy intake at dinner was similar in both diets.

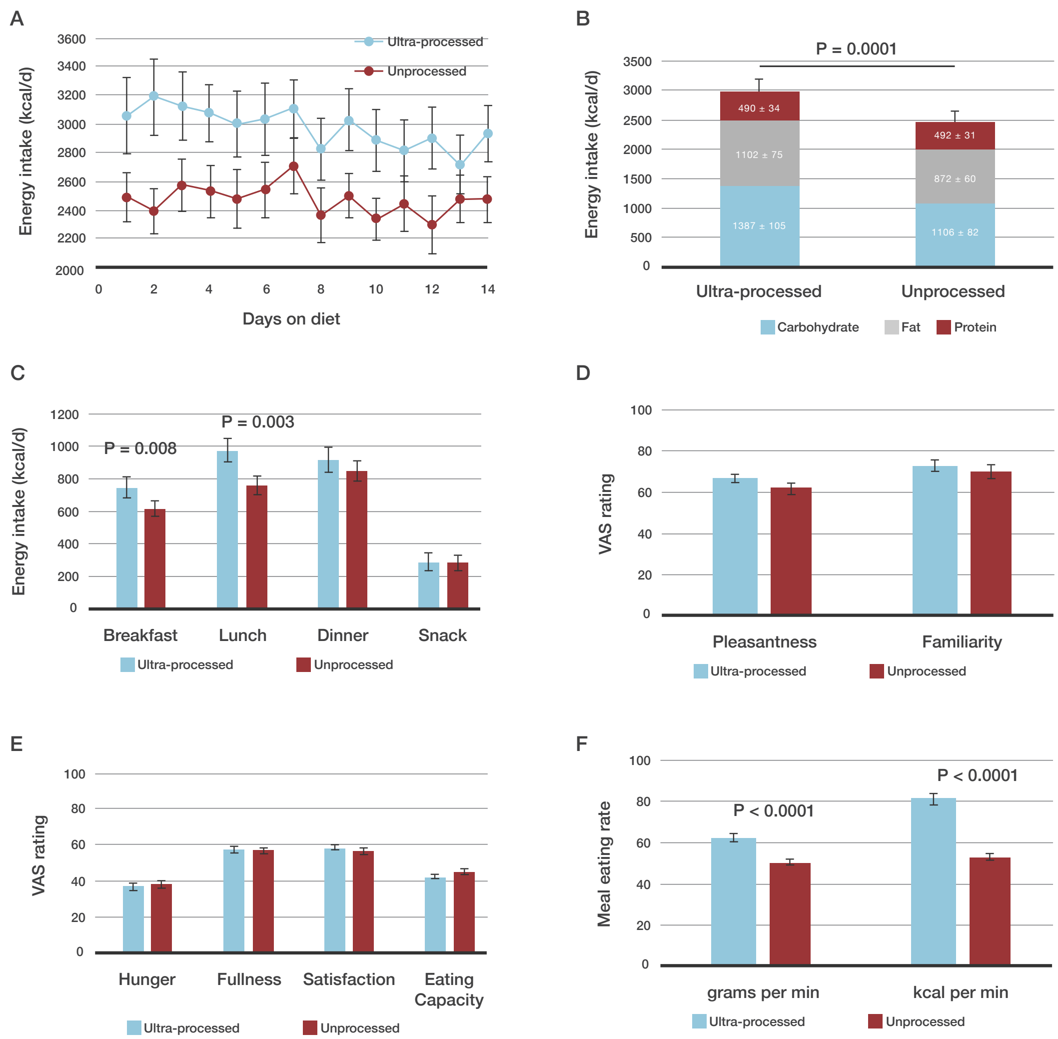
Body weight increased during the processed diet but decreased during the unprocessed diet (Figure 3). There was a strong, significant association between energy intake relative to baseline and increases in body weight. Body fat was the only significant body composition change, and changes in lean mass approached significance. However, as I'll

Table 1 Diet composition of the average 7-day rotating menu presented to the subjects during the ultra-processed and unprocessed diet periods

	Ultra-processed diet	Unprocessed diet
Three daily meals		
Energy (kcal/day)	3905	3871
Carbohydrate (%)	49.2	46.3
Fat (%)	34.7	35.0
Protein (%)	16.1	18.7
Energy density (kcal/g)	1.024	1.028
Non-beverage energy density (kcal/g)	1.957	1.057
Sodium (mg/1000 kcal)	1997	1981
Fiber (g/1000 kcal)	21.3	20.7
Sugars (g/1000 kcal)	34.6	32.7
Saturated fat (g/1000 kcal)	13.1	7.6
Omega-3 fatty acids (g/1000 kcal)	0.7	1.4
Omega-6 fatty acids (g/1000 kcal)	7.6	7.2
Energy from unprocessed (%)*	6.4	83.3
Energy from ulta-processed (%)*	83.5	0
Snacks (available all day)		
Energy (kcal/day)	1530	1565
Carbohydrate (%)	47.0	50.3
Fat (%)	44.1	41.9
Protein (%)	8.9	7.8
Energy density (kcal/g)	2.80	1.49
Sodium (mg/1000 kcal)	1454	78
Fiber (g/1000 kcal)	12.1	23.3
Sugars (g/1000 kcal)	24.8	95.9
Saturated fat (g/1000 kcal)	7.7	4.4
Omega-3 fatty acids (g/1000 kcal)	0.3	4.0
Omega-6 fatty acids (g/1000 kcal)	9.6	21.9
Energy from unprocessed (%)*	0	100
Energy from ulta-processed (%)*	75.9	0
Daily meals + snacks		
Energy (kcal/day)	5435	5436
Carbohydrate (%)	48.6	47.4
Fat (%)	37.4	37.0
Protein (%)	14.0	15.6
Energy density (kcal/g)	1.247	1.126
Non-beverage energy density (kcal/g)	2.147	1.151
Sodium (mg/1000 kcal)	1,843	1,428
Fiber (g/1000 kcal)	18.7	21.4
Sugars (g/1000 kcal)	31.9	51.0
Saturated fat (g/1000 kcal)	11.5	6.7
Omega-3 fatty acids (g/1000 kcal)	0.6	2.2
Omega-6 fatty acids (g/1000 kcal)	8.1	11.5
Energy from unprocessed (%)*	4.6	88.1
Energy from ulta-processed (%)*	81.3	0

** = the calculated energy percentages refer to the fraction of diet calories contributed from groups 1 and 4 of the NOVA classification system: (1) unprocessed or minimally processed, (2) processed culinary ingredients, (3) processed foods, and (4) ultra-processed foods*

Figure 2 Ad libitum food intake, appetite scores, and eating rate



(A) Energy intake was consistently higher during the ultra-processed diet. Data are expressed as mean ± SE
(B) Average energy intake was increased during the ultra-processed diet because of increased intake of carbohydrate and fat, but not protein. Data are expressed as mean ± SE, and p values are from paired, two-sided t-tests
(C) Energy consumed at breakfast and lunch was significantly greater during the ultra-processed diet, but energy consumed at dinner and snacks was not significantly different between the diets. Data are expressed as mean ± SE, and p values are from paired, two-sided t-tests
(D) Both diets were rated similarly on visual analog scales (VASs) with respect to pleasantness and familiarity. Data are expressed as mean ± SE
(E) Appetitive measures were not significantly different between the diets. Data are expressed as mean ± SE
(F) Meal eating rate was significantly greater during the ultra-processed diet. Data are expressed as mean ± SE, and p values are from paired, two-sided t-tests

discuss in the interpretation, the non-significant "lean mass" gains were likely dominated by changes in body water and were pretty variable between individuals, while changes in body fat were significant and consistent across individuals.

Blood measurements followed the changes in body mass (see Table 3), such that markers clearly indicated or trended toward showing a shift into a calorie deficit during the unprocessed diet. During the unprocessed diet, insulin and leptin were lower versus baseline and the processed diet (while not reaching the cut-off for significance), and T3 (thyroid hormone) was significantly lower, while free fatty acids were significantly higher (indicating body fat was metabolized).

Table 2 Energy expenditure and food intake during the respiratory chamber and doubly labled water periods

	Ultra-processed diet (Week 1)	Ultra-processed diet (Week 2)	Ultra-processed diet (2-week average)	Unprocessed diet (week 1)	Unprocessed diet (week 2)	Unprocessed diet (2-week average)	p Value[a]
Respiratory chamber days							
Energy intake (kcal/day)	2715 ± 86	2588 ± 66	2651 ± 53	2657 ± 86	2597 ± 66	2627 ± 53	0.75
Food quotient	0.850 ± 0.002	0.856 ± 0.003[c]	0.853 ± 0.002	0.846 ± 0.002	0.843 ± 0.003	0.845 ± 0.002	0.002
Energy expenditure (kcal/day)	2328 ± 28	2344 ± 29	2336 ± 19	2320 ± 28	2248 ± 29[c]	2284 ± 19	0.056
24h respiratory quotient	0.907 ± 0.005	0.899 ± 0.005	0.903 ± 0.003	0.875 ± 0.005	0.869 ± 0.005	0.872 ± 0.003	< 0.0001
Sleeping energy expenditure (kcal/day)	1515 ± 28	1550 ± 33	1532 ± 19	1516 ± 27	1535 ± 33	1525 ± 19	0.81
Sedentary energy expenditure (kcal/day)	1590 ± 21	1573 ± 30	1581 ± 17	1549 ± 21	1530 ± 30	1540 ± 17	0.084
Physical activity expenditure (kcal/day)	738 ± 29	771 ± 21	755 ± 18	771 ± 29	717 ± 21	744 ± 18	0.67
Doubly labled water period[b]							
Energy intake (kcal/day)	3099 ± 87	2865 ± 64[c]	2963 ± 74	2555 ± 82	2486 ± 64	2491 ± 74	0.0003
Food quotient	0.851 ± 0.002	0.854 ± 0.002[c]	0.854 ± 0.002	0.852 ± 0.002	0.856 ± 0.002[c]	0.855 ± 0.002	0.93
Adjusted respiratory quotient	0.903 ± 0.01	0.902 ± 0.009	0.901 ± 0.007	0.847 ± 0.01	0.836 ± 0.009	0.842 ± 0.007	< 0.0001
Daily CO2 production (L/day)	468 ± 13	505 ± 19	477 ± 6.9	444 ± 13	388 ± 19	420 ± 6.9	0.0001
Daily energy expenditure (kcal/day)	2496 ± 83	2693 ± 80	2546 ± 39	2497 ± 79	2309 ± 85	2375 ± 39	0.0064
Daily physical activity METs (via accelerometry)	1.502 ± 0.002	1.509 ± 0.003	1.5055 ± 0.002	1.507 ± 0.002	1.505 ± 0.003	1.5065 ± 0.002	0.71

a = p values refers to the comparison between the 2-week average values for ultra-processed versus unprocessed diets
b = n=19 because one subject's doubly labeled water failed quality control for the calculated deuterium dilution space
c = p < 0.05 comparing means for week 2 with week 1 within each diet period; mean ± SE

Interpretation

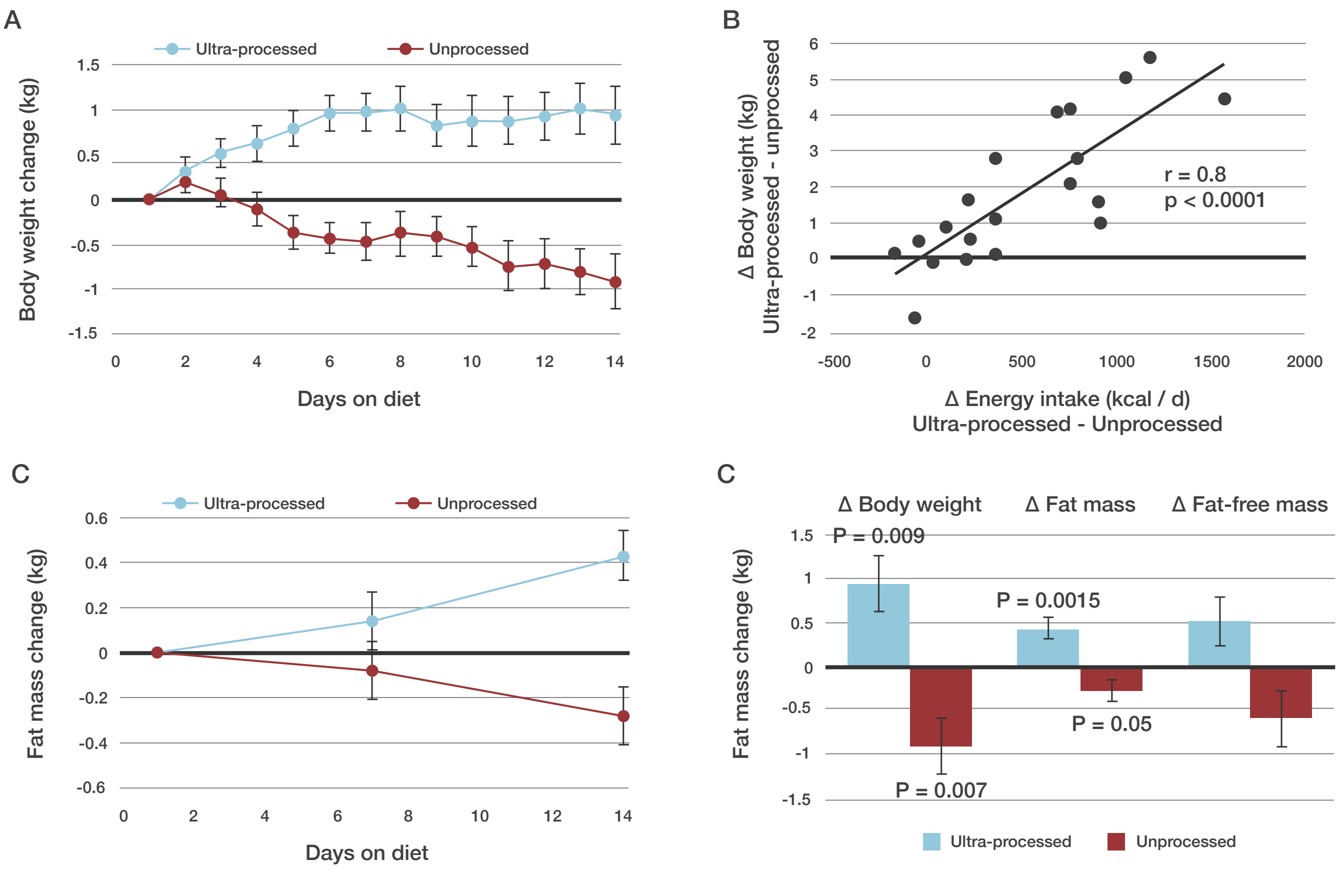
The broad interpretation of this study is pretty straightforward. A diet largely dominated by processed foods is less filling, easier to eat quickly, and more likely to result in greater consumption of total calories, largely driven by a lower protein content per calorie and higher energy density. However, to know what this means in terms of practice, and to understand the nuanced "whys" behind the outcomes, there is a lot more to examine. To unpack it all, I think it's helpful to first break it down into the topics I'll go over in this interpretation:

1. The difference between factually correct information and useful information.
2. Why processed diets result in "spontaneous" weight gain, and unprocessed diets result in "spontaneous" weight loss.
3. Interesting nuances of the findings.

The difference between factually correct information and useful information

While less common these days, there was a time in the "evidence-based II-

Figure 3 Body weight and composition changes



(A) The ulta-processed diet led to increased body weight over time whereas the unprocessed diet led to progressive weight loss. Data are expressed as mean ± SE
(B) Differences in body weight change between the ultra-processed and unprocessed diets were highly correlated with the corresponding energy intake differences. Data are expressed as mean ± SE
(C) Body fat mass increased over time with the ultra-processed diet and decreased with the unprocessed diet. Data are expressed as mean ± SE
(D) Body weight, body fat, and fat-free mass changes between the beginning and end of each diet period. Data are expressed as mean ± SE, and p values are from paired, two-sided t-tests

FYM (if it fits your macros) community" that the common response to any questioning of certain foods being "acceptable" for health, fitness, fat loss, or bodybuilding would be met with a statement along the lines of: "diet quality doesn't matter for body composition change if macros (mainly protein), calories, fiber, and (sometimes) sugar (depending on who was talking) are matched (with the occasional caveat of also taking a multivitamin)." Now, this is factually true, at least mostly, and at least in the short term. I can think of strange diet setups where certain essential fatty acids, amino acids, and micronutrients are lacking and eventually cause problems, but I digress. Despite being a factual statement, I don't think this is a very *useful* statement. This factually true statement only has applicability if all these variables are controlled, meaning you are tracking energy and/or macros.

However, most people don't, and at least in the long term, shouldn't be in a

Table 3 Fasting blood measurements at baseline and at the end of the ultra-processed and unprocessed diet periods

	Baseline	Ultra-processed diet	p Value, ultra-processed vs. baseline diet	Unprocessed diet	p Value, unprocessed vs. baseline diet	p Value, ultra-processed vs. unprocessed diet
Leptin (ng/mL)	44.3 ± 1.7	45.1 ± 1.7	0.75	40.4 ± 1.7	0.11	0.058
Active ghrelin (pg/mL)	61.4 ±3.5	54.1 ± 3.5	0.15	48.3 ± 3.5	0.01	0.24
PYY (pg/mL)	28.9 ± 1.9	25.1 ± 1.9	0.15	34.3 ±1.9	0.047	0.001
FGF-21 (pg/mL)	397 ± 59	289 ± 59	0.21	362 ± 59	0.67	0.39
Adiponectin (mg/L)	7.3 ± 0.7	8.0 ± 0.7	0.43	4.6 ±0.7	0.007	0.0007
Resistin (ng/mL)	13.5 ± 0.4	12.4 ± 0.4	0.05	12.1 ± 0.4	0.01	0.49
Active GLP-1 (pg/mL)	1.88 ± 0.19	1.25 ± 0.19	0.027	1.57 ±0.19	0.26	0.25
Total GIP (pg/mL)	79.7 ± 5.4	67.9 ± 5.4	0.13	64.3 ± 5.4	0.052	0.64
Active GIP (pg/mL)	27.4 ± 2.8	20.0 ± 2.8	0.07	18.2 ± 2.8	0.025	0.65
Glucagon (pmol/L)	12.0 ± 0.8	11.0 ± 0.8	0.42	9.8 ±0.8	0.07	0.29
Hgb A1C (%)	4.98 ± 0.03	5.02 ± 0.03	0.28	5.00 ± 0.03	0.55	0.64
Glucose (mg/dL)	90.5 ±0.9	88.6 ± 0.9	0.16	88.0 ± 0.9	0.06	0.62
Insulin (μU/mL)	11.9 ± 0.9	11.3 ±1.0	0.64	8.9 ± 1.0	0.03	0.09
C-peptide (ng/mL)	2.19 ± 0.06	2.14 ± 0.06	0.62	1.94 ± 0.06	0.01	0.032
HOMA-IR	2.8 ± 0.3	2.5 ± 0.3	0.50	1.9 ± 0.3	0.03	0.14
HOMA-beta	152 ± 10	159 ± 11	0.63	129 ± 10	0.13	0.053
Total cholesterol (mg/dL)	155 ± 3	152 ± 3	0.54	137 ± 3	0.0002	0.001
HDL cholesterol (mg/dL)	58.2 ± 0.8	55.0 ± 0.9	0.01	48.3 ± 0.8	< 0.0001	< 0.0001
LDL cholesterol (mg/dL)	82 ± 3	84 ± 3	0.61	77 ± 3	0.21	0.085
Triglycerides (mg/dL)	72 ± 3	62 ± 3	0.02	59 ± 3	0.003	0.45
Free fatty acids (μmol/L)	409 ± 40	384 ± 40	0.67	556 ± 40	0.013	0.004
Uric acid (mg/dL)	4.9 ± 0.8	4.5 ± 0.8	0.0007	4.9 ± 0.8	0.55	0.004
TSH (μIU/mL)	2.2 ± 0.1	2.6 ± 0.1	0.054	2.5 ± 0.1	0.24	0.42
Free T3 (pg/mL)	3.17 ± 0.06	3.20 ± 0.06	0.72	3.03 ± 0.06	0.11	0.051
Free T4 (ng/dL)	1.19 ± 0.02	1.22 ± 0.02	0.36	1.27 ± 0.02	0.019	0.13
T3 (ng/dL)	113 ± 2	112 ± 2	0.80	104 ± 2	0.011	0.019
T4 (μg/dL)	6.8 ± 0.1	6.9 ± 0.1	0.70	6.8 ± 0.1	0.91	0.79
PAI-1 (ng/mL)	4.0 ± 0.5	4.6 ± 0.5	0.42	4.7 ± 0.5	0.34	0.89
hsCRP (mg/L)	2.7 ± 0.3	2.4 ± 0.3	0.48	1.5 ± 0.3	0.014	0.072

All data are presented as mean ± SE

constant state of tracking macros or calories. As discussed in my video series on flexible dieting (video one, two, three), tracking can be used as an instructive tool, but it should be temporary, as there are potential pitfalls from primarily relying on external cues for regulating energy intake. In the real world, general population gym-goers largely find tracking tedious and unsustainable, in my experience. Thus, unless your goal is not intended to be sustained (for example, a contest preparation resulting in stage leanness), the behaviors utilized shouldn't be unsustainable either, and 99% of people aren't going to track their macros for the rest of their lives.

So again, is it useful to know that a processed diet can be just as effective as an unprocessed one so long as all nutritional variables like calories and macros are controlled? Not really, because in reality, those variables generally won't be controlled. The only utility of that statement I can see is if you're talking to someone who is actually afraid of specific processed foods and thinks for some insidious magical reason they are harmful at any frequency or dose. In that case, I could see the value in letting someone know that occasionally having a Snickers bar in the context of a healthy diet is absolutely fine. Moreso, even if someone does track habitually, the knowledge that a processed diet could work isn't really helpful. Indeed, it could cause problems, as following such a diet would make the qualitative difficulty of maintaining a deficit via a processed diet higher. If people find the same level of satiety and hunger occurs when eating an additional 500kcals (as was shown in this study), it's logical to assume they'd feel hungrier on a processed diet when eating the same amount of calories they'd otherwise consume on an unprocessed diet.

Why processed diets result in "spontaneous" weight gain, and conversely, why unprocessed diets result in "spontaneous" weight loss

In the real world, energy density is higher on processed diets. If you're thinking "what is energy density?", watch my video series on it that I just concluded in this issue (Part 1 here). While the researchers attempted to control for energy density, it probably couldn't be properly accounted for because processed foods are simply so much higher in energy density. Thus, an attempt was made by adding fiber to the beverages of the processed diet, but unfortunately, fluids aren't as well "recognized" by the body for inducing satiety (3).

Another reason processed diets lead to food overconsumption in the real world is due to a phenomenon known as protein leverage theory. Simply put, satiety is lower and hunger is higher until a certain threshold of protein is consumed in an ad libitum setting. Thus, because processed foods are so densely packed with carbohydrates and fat, you end up consuming more total calories to reach the same level of protein. Indeed, some research indicates a part of the reason processed diets are fattening is because they have proportionally less protein per calorie (4). In this study, it seems the ad libitum intake of the participants during the processed diet was primarily via carbohydrate- and fat-dominant foods, but there was a remarkably tight ad libitum intake of protein during both diets; because the participants ate more carbs and fats while eating the processed diet to achieve this same protein intake, more total calories had to be eaten.

If anything, I think the energy density, fiber, and protein differences in the real world would make this effect more pronounced. Yes, I think the 500kcal difference observed in this study is probably a conservative estimation of the true difference in the real world, because in the real world, there aren't researchers trying to match the energy density, protein, and fiber intakes of what's presented to you.

On top of these mechanisms, while the processed diet wasn't rated as more palatable or tasty or satisfying, it was eaten more quickly and the hormonal responses indicated less satiation and more hunger during the processed diet. This could be due to mouthfeel and texture of processed foods making it easier to eat them more quickly, and thus, more calories can be consumed before satiety "sets in." It's also possible that the questions related to hunger and satiety weren't sensitive or targeted enough to represent the qualitative eating experience differences between diets. Another perspective to consider is that the goal of a food manufacturer is to encourage the consumption of the food they manufacture. This is actually not accomplished by making something super hedonically satisfying, but rather tasty, but not so tasty to cross the threshold into satisfaction, which discourages further consumption (an example would be high fructose corn syrup, which is only slightly sweeter than regular sugar; as most have a 55/45 breakdown of fructose to glucose versus the 50/50 split of standard sugar). Thus, the qualitative descriptors in this study of "pleasantness" and "familiarity" and "satisfaction" might not have picked this up. Nonetheless, based on hormonal data, the participants were more full and less hungry eating the unprocessed diet, and they also ate slower, which might have allowed more time for these hormonal signals to be received, resulting in earlier meal cessation and lower calorie consumption.

According to the authors, protein could only explain, at most, 50% of the difference in energy intake between groups. Thus, all in all, energy density, eating speed, and hormonal differences explain the remaining differences (and perhaps other unmeasured factors). The good news is that unprocessed diets rock. They result in effortless fat loss; eating a whole food diet for the average person will result in higher levels of satiety, less hunger, and subsequently a lower energy intake, thus leading to fat loss. This will of course only last so long, but your "settling point" of adiposity will almost certainly be lower when consuming an unprocessed versus a processed diet. If you aren't sure what that looks like, think lots of fruits, vegetables, lean proteins, and (in general) a diet dominated by single ingredient foods. I know, groundbreaking stuff.

Interesting nuances of the findings

There were some outcomes which, at first glance, seem like head scratchers. For example, in a 2010 study, energy expenditure was shown to be lower acutely after consuming a processed versus an unprocessed sandwich. Yep, another knock against processed diets is they might reduce the energy-out side of the

Table 4 NOVA food classification system

Group 1: Unprocessed or minimally processed	Group 2: Processed culinary ingredients	Group 3: Processed foods	Group 4: Ultra-processed foods
Examples: fresh, frozen, or dried fruits and vegetables; pasta made from flours and water; spices and herbs, fresh or dried; meat, poultry, fish and seafood, whole or in the form of steaks, fillets and other cuts, chilled or frozen; eggs; unsweetened milk and yogurt; coffee, tea, water	Examples: salt, sugar, honey, cooking oils, butter, corn, starch, vinegar, baking soda	Examples: canned fruits and vegetables; salted nuts; salted, cured, or smoked meats; cheeses; freshly made bread; beer	Examples: sodas, sweet and salty packaged snacks, chicken nuggets, fish sticks, hot dogs, pre-prepared frozen meals, breakfast cereals, energy bars, packaged cakes and sweets

Source: Monteiro et al, 2016; NOVA

equation, likely via a reduction in the thermic effect of food and the cost to get the metabolizable energy content of the food (5). Because they've undergone substantial industrial processing, our body doesn't need to expend as many calories to actually digest and metabolize highly processed food. However, in the present study, energy expenditure was higher during the processed diet, almost reaching the cut off for significance (p = 0.06). While this appears contradictory, I don't think it is. In the prior study, the response to *a calorie-matched meal* was assessed, but in the present study, the researchers measured the energy expenditure response to an entire diet for 14 days, compared to another diet that was 500kcals higher. Thus, in the present study, we are likely just observing the ramping up of energy expenditure in response to a calorie surplus, which has been shown as a normal response to overfeeding (6), and likely an attempt by the body to maintain metabolic homeostasis (something that is easily overcome at a population level by our obesogenic environment in the modern world). So unfortunately, processed diets may still very well reduce energy expenditure when calorie matched compared to unprocessed diets.

Another interesting finding was that while the only significant body composition changes were changes in body fat, changes in lean mass were almost significant. There was a nonsignificant, small drop in lean mass during the unprocessed diet and a small increase during the processed diet. However, for one, there is an obligatory loss or gain of lean mass when fat mass changes, as adipose tissue is not completely composed of fat mass and has some lean tissue as part of its structure (7). Further, these changes in lean mass were significantly associated with changes in sodium intake (r = 0.63; p = 0.004), likely indicating that changes in extracellular fluid shifts appeared as lean tissue changes, contributing to this outcome.

As a final, unfortunate, nuanced note on this study, the cost of the processed diet per kcal was ~50% less than the un-

APPLICATION AND TAKEAWAYS

1. Processed diets resulted in significant increases in ad libitum consumption of energy. In spite of eating ~500kcal more when consuming a processed diet, participants in this study felt the same level of hunger and satiation on both diets, and subsequently gained body fat on the processed diet.
2. This is primarily due to the following combined aspects of processed diets: they have a higher energy density, less protein per calorie, less fiber per calorie, they are more likely to be eaten quickly, and subsequently, they are less satiating. Also, it's cheaper to get the same amount of calories from processed foods versus unprocessed, though that didn't factor into the results of this study.
3. On the flip side, unprocessed, whole food diets result in significant decreases in energy consumption and subsequently, fat loss. This is due to lower energy density, more protein per calorie, more fiber per calorie, slower eating times, and subsequently, more satiation. The downside is that whole food is more expensive.
4. As a final note, to help with application, check out Table 4 with the NOVA classification system so you can get a better idea of how processed and unprocessed foods are categorized.

processed diet. Meaning, it's cheaper to get your energy from processed foods, which doesn't bode well for society-level behavior change.

Next Steps

I would love to see this field of research expanded into a more applicable phase. Much of Hall's research is groundbreaking, but stems from the research battleground between carbs versus fat and carbs versus calories, which are – in my opinion – largely unproductive. No, the insulin model of obesity isn't correct (8); yes, energy balance ultimately dictates changes in body mass (9). But, unfortunately, elucidating these points rarely provides anything actionable. Qualitative interviews around barriers to adopting unprocessed diets, assessments of how to intervene to change behavior, and developing diagnostic tools to assess which behaviors are the largest contributors to an individual's adiposity to then provide individualized counseling are the directions I'd like to see future research go. Essentially, if we know that processed diets – even in a controlled research setting that likely dampens their effect on overeating – result in substantially more ad libitum food intake, what can we do to make it more likely that people will eat unprocessed diets?

References

1. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, Chen KY, Chung ST, Costa E, Courville A, Darcey V, Fletcher LA. Ultra-Processed Diets Cause Excess Calorie Intake and Weight Gain: An Inpatient Randomized Controlled Trial of Ad Libitum Food Intake. Cell metabolism. 2019 May 16.
2. Monteiro CA, Cannon G, Levy RB, Moubarac JC, Louzada ML, Rauber F, Khandpur N, Cediel G, Neri D, Martinez-Steele E, Baraldi LG. Ultra-processed foods: what they are and how to identify them. Public health nutrition. 2019 Apr;22(5):936-41.
3. DellaValle DM, Roe LS, Rolls BJ. Does the consumption of caloric and non-caloric beverages with a meal affect energy intake? Appetite. 2005 Apr 1;44(2):187-93.
4. Steele EM, Raubenheimer D, Simpson SJ, Baraldi LG, Monteiro CA. Ultra-processed foods, protein leverage and energy intake in the USA. Public health nutrition. 2018 Jan;21(1):114-24.
5. Barr S, Wright J. Postprandial energy expenditure in whole-food and processed-food meals: implications for daily energy expenditure. Food & nutrition research. 2010 Jan 1;54(1):5144.
6. Levine JA, Eberhardt NL, Jensen MD. Role of nonexercise activity thermogenesis in resistance to fat gain in humans. Science. 1999 Jan 8;283(5399):212-4.
7. Abe T, Dankel SJ, Loenneke JP. Body Fat Loss Automatically Reduces Lean Mass by Changing the Fat-Free Component of Adipose Tissue. Obesity (Silver Spring, Md.). 2019 Mar;27(3):357.
8. Hall KD, Guyenet SJ, Leibel RL. The carbohydrate-insulin model of obesity is difficult to reconcile with current evidence. JAMA internal medicine. 2018 Aug 1;178(8):1103-5.
9. Hall KD, Guo J. Obesity energetics: body weight regulation and the effects of diet composition. Gastroenterology. 2017 May 1;152(7):1718-27.

Study Reviewed: Upper and Lower Thresholds of Fat-Free Mass Index in a Large Cohort of Female Collegiate Athletes. Harty et al (2019)

Using Fat-Free Mass Index to Forecast Long-Term Gains for Males and Females

BY ERIC TREXLER

At some point in their lifting career, just about every lifter wonders, "Exactly how big can I get?" While fat-free mass index (FFMI) isn't going to tell you the exact limit to your potential, or perfectly weed out steroid-users from drug-free lifters, it's still a helpful metric for approximating muscularity and understanding how big most people can get. Read this article to figure out how big male and female lifters tend to get, and what this means for your natural potential for muscle growth.

KEY POINTS

1. Fat-free mass index (FFMI) has been used as a proxy for muscularity and has previously been used to estimate upper limits for lean mass accretion in drug-free lifters.
2. The current study (1) measured FFMI in a large sample of 372 female athletes. The 97.5th percentile value was 23.9, but multiple athletes had values over 25, and one athlete had a value over 27.
3. Current evidence suggests that it isn't rare for males to have an FFMI as high as 28, or for females to have an FFMI as high as 24. Lifters can use FFMI to help plan out their weight gain phases, but shouldn't use it to arbitrarily limit themselves or make baseless steroid accusations about others.

A large percentage of lifters, at some point in their career, have an interest in getting really, really huge. Inevitably, these lifters will reach a point in their training career where they begin to wonder exactly how big they could get if they absolutely maximized their natural potential. People have proposed several ways to estimate a lifter's genetic limit for lean mass gains, using tools such as a variety of online calculators, multiple joint and bone measurements, the ratio of muscle to bone, and fat-free mass index (FFMI). The beauty of FFMI is that it is remarkably easy to calculate, using very common measurements that are hard to mess up.

In 1995, Kouri et al (2) published a study assessing FFMI in resistance-trained males, both with and without a history of steroid use. The authors noticed that their drug-free lifters all had values below 25 kg/m^2, while many of the steroid users had values well above 25. Based on this information, it was implied that values above 25 might be considered a "red flag" with regard to steroid use. In the online fitness world, the idea of a "natural limit" of 25 became law, despite the very, very notable limitations of the Kouri study (for example, aiming to determine the upper limits of human potential based on 74 commercial gym-goers).

Unfortunately, the FFMI literature has been male-dominated to date, and there hasn't been as much public discourse regarding a proposed upper limit for female

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

muscularity. The current study sought to characterize FFMI values in a wide range of sports, and to provide a preliminary estimate of the FFMI upper limit for female athletes. Results indicated that FFMI differed between sports and was positively associated with indices of bone health. Most surprisingly, at least three participants had values above 25, with a maximal recorded value of 27.2! This article will discuss what these numbers tell us about FFMI upper limits for both males and females, and how to use FFMI to guide your next bulking phase.

Purpose and Hypotheses

Purpose

The authors stated that they had three central purposes; to report sport-specific norms for FFMI in female athletes, to determine if these values differed between sports, and to estimate an "upper limit" for FFMI in female athletes.

Hypotheses

The authors hypothesized that FFMI values would significantly differ between sports, with particularly low values expected in sports that they considered to be "weight-sensitive." Such sports included cross country, gymnastics, dance, swimming and diving, synchronized swimming, wrestling, and weightlifting.

Table 1 Body composition variables of subjects in the current study (1)

	Mean ± SD
Height (cm)	167.55 ± 7.50
Weight (kg)	69.46 ± 13.04
BF %	24.18 ± 5.48
FM (kg)	17.50 ± 7.18
FFM (kg) (w/ BMC)	52.93 ± 7.38
BMC (g)	2543.79 ± 364.77
BMD (g/cm³)	1.204 ± 0.096

SD = standard deviation; cm = centimeters; kg = kilograms;
BF% = body fat percentage (using DEXA);
FM = fat mass (using DEXA);
FFM = fat-free mass (using DEXA);
BMC = bone mineral content; g = grams;
BMD = bone mineral density

Subjects and Methods

Subjects

This study sampled 372 female collegiate athletes, representing a wide range of sports. Table 1 shows the general descriptive characteristics of the sample.

Design

This study featured a very simple design: recruit a big group of athletes, measure body composition using dual-energy x-ray absorptiometry (DEXA), then crunch some numbers. The researchers followed typical

Table 2 Fat-free mass index values for each sport (1)

Sport	N	FFMI	Range
Cross country	11	$16.56 \pm 1.14^{c,d}$	14.71 - 18.58
Gymnastics	35	18.62 ± 1.12^{d}	16.16 - 20.84
Dance	2	17.86 ± 1.17	17.03 - 18.68
Swim & dive	31	18.16 ± 1.67^{d}	15.86 - 22.77
Synchronized swimming	29	$17.27 \pm 1.47^{c,d}$	15.07 - 20.68
Wrestling	19	$19.15 \pm 2.47^{a,b}$	15.04 - 24.45
Olympic weightlifting	15	$19.69 \pm 1.98^{a,b}$	17.19 - 23.84
Track & field	22	18.98 ± 2.50^{a}	15.27 - 24.38
Basketball	20	18.64 ± 1.87	15.96 - 22.37
Ice hockey	16	17.96 ± 1.04^{d}	16.22 - 19.76
Lacrosse	40	18.58 ± 1.84^{d}	15.94 - 26.35
Rugby	99	$20.09 \pm 2.23^{a,b}$	15.73 - 27.20
Volleyball	20	18.04 ± 1.13^{d}	15.35 - 20.72
Water polo	13	18.35 ± 1.92	14.85 - 22.85
Total†	372	18.82 ± 2.08	14.71 - 27.20

All FFMIRaw data is presented as mean ± SD
a = significantly different from cross country (p < 0.05)
b = significantly different from synchronized swimming (p < 0.05)
c = significantly different from olympic weightlifting (p < 0.05)
d = significantly different from rugby (p < 0.05)
† = significant difference between sports (p < 0.001)

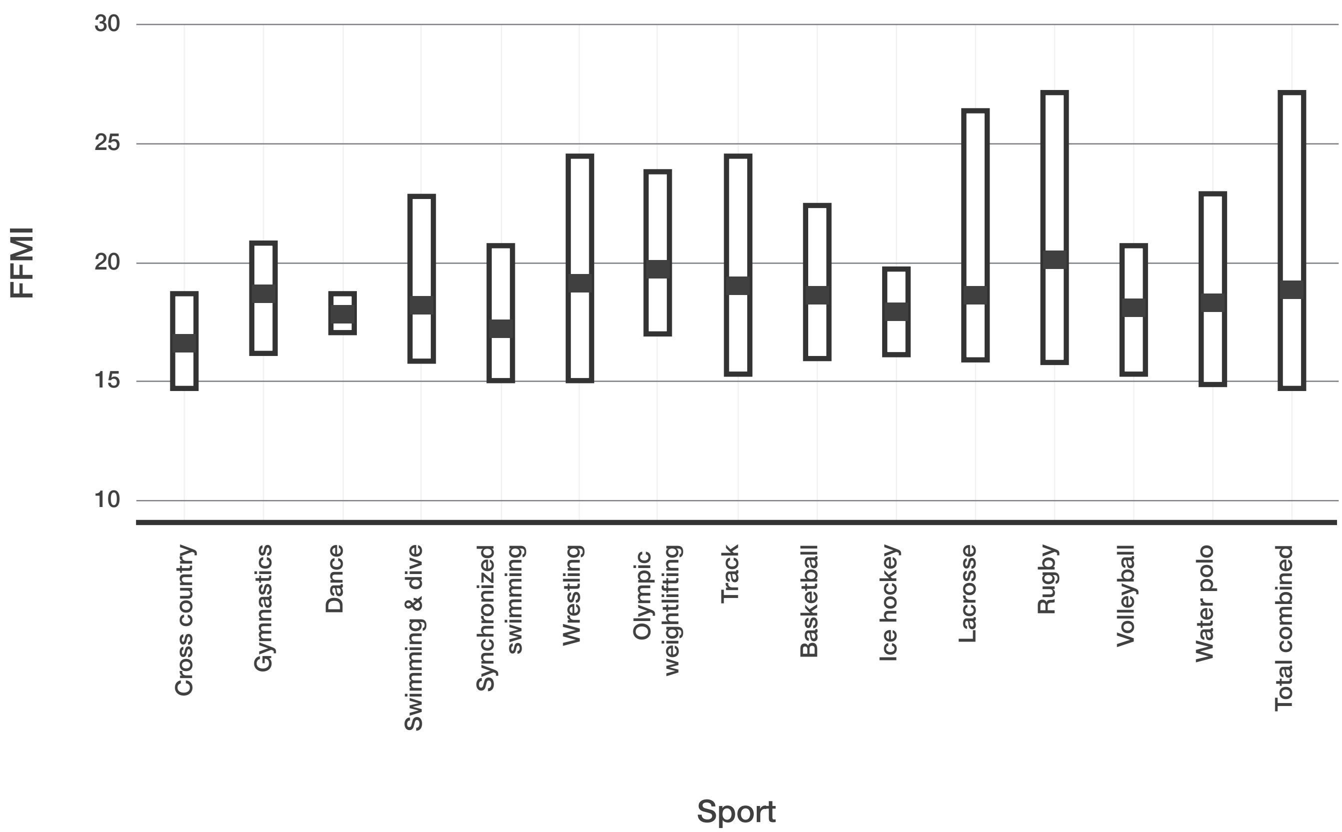
pre-visit guidelines for DEXA measurements, but weren't able to standardize menstrual cycle phase or competitive season (in-competition or off-season). These aren't particularly critical details, and they are extremely understandable, as studying collegiate athletes is extremely difficult from a logistical perspective.

After the scans were done, FFMI was calculated using the following equation:

$$\textbf{FFMI} = \frac{\text{Total lean mass (kg) + Total bone mineral content (kg)}}{(\text{Height [m]})^2}$$

For statistics, they were interested in reporting "typical" FFMI ranges for each sport, comparing between sports,

Figure 1 Fat-free mass index values for each sport



FFMI values for each sport. The top and bottom of each box represent the maximum and minimum, while the middle bar represents the mean value observed for that sport.

and assessing correlations between FFMI and markers of bone health.

A Note On Height Adjustments

If you check out the original paper, you'll see that they applied a couple of different height adjustments to their FFMI values. One adjustment equation was derived from a previous study by Kouri et al (2), and the other was derived from a study my colleagues and I published in 2017 (3). The general premise for height correction is that FFMI, if not adjusted, tends to be a bit biased, with higher values observed in taller individuals. Gains in body mass don't perfectly scale with height squared, and if we were to cube height instead of squaring it in the FFMI equation, that would lead to an over-correction. You could probably roughly account for this height bias by tweaking the exponent in the denominator, with the "ideal" value probably falling somewhere around the 2.4-2.6

Table 3 FFMI percentile cut-offs for weight-sensitive sports, other sports, and all sports combined

	All Sports	Weight-sensitive sports	Other sports
Percentile	FFMI (kg/m²)	FFMI (kg/m²)	FFMI (kg/m²)
5	15.96	15.71	16.21
10	16.38	16.11	16.95
15	16.92	16.35	17.24
20	17.20	16.78	17.51
30	17.63	17.19	17.83
40	18.10	17.63	18.41
50	18.52	18.14	18.72
60	18.93	18.52	19.23
70	19.55	18.94	19.91
80	20.26	19.65	20.71
90	21.52	20.65	22.16

range, but the most common approach is to use regression-based mathematical adjustments.

In this study, the authors found virtually no relationship between height and FFMI. This is probably related to the fact that not every athlete in the sample was necessarily trying to get as big as possible, as certain sports tend to self-select and reinforce specific height and body composition characteristics. So, for this study, the authors (wisely) ditched the height-adjusted values.

Findings

Table 2 shows the exact breakdown of FFMI values by sport. That's a large amount of numbers, so the same data are presented graphically in Figure 1, which is a little easier on the eyes. Finally, Table 3 presents some percentile ranges for all sports combined, in addition to separate values for weight-sensitive and non-weight-sensitive sports. The authors reported that the 97.5th percentile value was 23.9, which was operationally defined as the "upper threshold," or the upper limit that *most* female athletes can realistically shoot for. The lowest observed value was 14.7, and the highest was 27.2. In addition, FFMI was positively correlated with both bone mineral content ($r = 0.53$, $p < 0.001$) and bone mineral density ($r = 0.46$, $p < 0.001$).

Interpretation

I want to focus the majority of this discussion on the concept of using FFMI to establish upper limits for muscularity, but first I want to address the other results of this paper. The authors found, unsurprisingly, that FFMI differed among sports. This isn't shocking, but it's still helpful to actually quantify patterns that we know to be true. I used to do a bunch of research on college athletes that involved muscle ultrasound scans. One day, a fellow researcher walked by and whispered, jokingly, "Do you really need an ultrasound to know if you're looking at an athlete?" Point taken, but there's value in building a quantifiable profile for athletes on a sport-by-sport (or even position-by-position) basis, as it allows us to understand the body composition metrics that appear to be associated with high-level performance and injury reduction.

THESE RESULTS SUGGEST THAT IT'S NOT PARTICULARLY ATYPICAL FOR FEMALES TO ACHIEVE FFMI VALUES WELL INTO THE MID-20S, AND IF IT ONLY TOOK A FEW HUNDRED PEOPLE TO OBSERVE A VALUE OVER 27, YOU CAN BE CERTAIN THAT THERE ARE PLENTY OF DRUG-FREE FEMALES OUT THERE WITH VALUES COMFORTABLY HIGHER THAN THAT.

Along those lines, the authors found that FFMI was correlated with bone mineral density. This is pretty notable for female athletes in weight-sensitive sports, in which bone (and other) injuries are quite prevalent. We've known for a while that low energy availability (as discussed in three previous MASS articles by Mike, Eric Helms, and I) can lead to both performance and injury issues in athletes, and this seems to be particularly prevalent in female athletes (4).

FFMI CAN BE USEFUL FOR PLANNING PURPOSES. HOWEVER, IT'S REALLY IMPORTANT TO REMEMBER WHAT FFMI IS NOT GOOD FOR: IMPOSING LIMITS ON YOURSELF AND MAKING BASELESS STEROID ACCUSATIONS.

We also know that disordered eating is a common concern in female collegiate athletes, and researchers and practitioners use extremely careful language when discussing body composition with this population. This study is admittedly a bit dated, but researchers in the 1980s surveyed 42 female collegiate gymnasts, and found that 28 of them were told by a coach that they were too heavy, and 21 of these 28 were using at least one unhealthy weight-control behavior (5). So, while these initial FFMI findings might seem obvious, they give researchers and coaches a body composition metric, which likely relates to both performance and injury risk, that isn't fat-focused. As a result, they can more readily discuss objective, performance-oriented body composition goals with their athletes without dwelling on fat mass, which is pretty useful.

The "maximum limit" for fat-free mass index

As I mentioned in the introduction, a large segment of the online fitness world has long viewed 25 as the maximal upper limit for a natural, male lifter. So, if you were above 25, you weren't natural. In 2017, I published a paper showing that FFMI values exceeding 25 were not only possible, but pretty common, in high-level American football players. In fact, 31.3% of the Division I players exceeded 25, and we observed several individuals above 28. Some people were pretty bothered by the finding, and suggested that the study was no more than evidence of rampant steroid use. I disagreed then, and I still do. But now, as we'll discuss, there's even more evidence to support my contention.

As a reminder, the conclusions of the Kouri paper (2) rested on shaky ground all along. They sampled only 74 males from commercial gyms, and their inclusion criteria only required that subjects had been lifting for at least two years. If you train at a standard commercial gym, take a look around. Excluding the people who have ever used steroids, select 74 guys. Do you think you've isolated the upper and lower boundaries of human potential for *any* measurable characteristic?

The other aspect of the Kouri paper involved estimating FFMI of Mr. America winners from 1939-1959. They selected this time range because they felt it was still plausible to believe that the competitors had not yet started using anabolics. I don't feel comfortable using this data to support or challenge any hypothesis, because body composition was essentially determined by educated guesses. Nonetheless, there were several Mr. America winners that were comfortably above 25, and those physiques were built using the limited training- and nutrition-related knowledge and resources that were available over 60 years ago.

Overall, there's just never been strong evidence to suggest that a male cannot exceed 25, but there was still some pushback from reporting values above 25 in high-level male athletes. The present study has reported values above 25 in *females*, so I would imagine there are at least a few people losing their minds over it.

Upper thresholds for fat-free mass index in females

The current study recruited a sample that was likely to contain some pretty muscular females. They got a big group of people (n = 372), and they were sure to include sports that reward strength and power, such as wrestling, Olympic weightlifting, and rugby. In fact, they found 99 female collegiate rugby players, which is a surprisingly huge number based on the sample sizes for all other sports. The biggest rugby player clocked in with an FFMI of 27.2, but high values weren't restricted to the rugby players. A lacrosse player had a value of 26.35, and there were also individual values above 24 for the wrestling and track & field teams. These values might seem high, but my colleagues and I reported similar data from female collegiate athletes in a recent study (6). Our study featured a smaller sample size (266 versus 372), and a different selection of sports, so one wouldn't expect our values to line up perfectly. However, we also reported a female with a value over 25, and several individuals above 20. These results suggest that it's not particularly atypical for females to achieve FFMI values well into the mid-20s, and if it only took a few hundred people to observe a value over 27, you can be certain that there are plenty of drug-free females out there with values comfortably higher than that.

Upper thresholds for fat-free mass index in males

As I mentioned previously, our study in football players found a large number of males with an FFMI over 25 (3), and the 97.5th percentile cut-off was 28.1. The authors of the current study (1) recently published a very similar study in 209 male collegiate athletes (7), and their results line up quite nicely with our reported findings. Just by a visual estimate from their figures, it looks like they had three male athletes at or above ~29, and one of them was comfortably above 30. For their entire sample, which included several sports that place minimal value on attaining maximal levels of lean mass (such as cross country, golf, and swimming), the 97.5th percentile was 28.3. Finally, a 2018 study (8) sampled 95 large male athletes competing in American football, powerlifting, Sumo, or shot put, for the purpose of determining upper limits for muscularity. The results indicated that the *average* FFMI value was slightly over 25, there were numerous athletes with FFMIs well above 25, and a handful of athletes had values above 30. So, based on the three most recent studies in resistance-trained males, it seems like you don't have to look extremely hard to find drug-free males in the high 20s. Frankly, this shouldn't be shocking; a 1999 study on 36 Sumo wrestlers included two subjects with FFMIs above 36, and the authors found that an FFMI of >30 tended to separate the elite Sumo wrestlers from their sub-elite counterparts (9).

Using fat-free mass index

Fat-free mass index can be really useful, but there are a couple of things it should *not* be used for. If you read our study from back in 2017, you'll see that we aimed to report the 97.5th percentile observed, rather than identify a universal "maximum limit." The value was not intended to be the maximum value possible, but rather an upper threshold that *most* people could feasibly hope to aim for, because *most* people are, by definition, not outliers. Along these lines, you should not use FFMI to impose restrictive limits on your lifting goals, and you definitely shouldn't use FFMI to make baseless steroid accusations about others. We simply don't have enough data to know exactly how many people can achieve the remarkable FFMIs that have been reported in recent literature, but you could theoretically be one of them, and so could the random jacked person that everyone accuses of being on steroids.

What you *can* use FFMI for, is to help plan out your weight gain phases. You can estimate the FFMI of people who are excelling in whatever you want to excel in, whether that's bodybuilding, powerlifting, or some other athletic endeavor. You can also determine your "ideal" offseason body-fat percentage (BF%) or the highest body-fat percentage you'd be comfortable with at the peak of your weight gain phase. You can then plug them into the equation below, which gives you a weight to shoot for at the given body-fat percentage that you chose.

$$\textbf{Target Weight (kg)} = \frac{\text{FFMI} \times \text{Height(m)}^2}{1 - \frac{\text{BF\%}}{100}}$$

Based on the data currently available, it seems that values substantially over 28 are probably a stretch for a lot of males,

APPLICATION AND TAKEAWAYS

1. It doesn't seem particularly unusual for males to achieve FFMI values as high as 28, and for females to achieve FFMI values as high as 24. For college athletes, the highest observed values for males and females have been ~31 and ~27.
2. The body of FFMI literature is still small, so it would be premature to say we have a firm understanding of the natural limits of muscularity. We really need some huge studies that include genetically gifted, exceptionally well-trained individuals to enhance our understanding.
3. You shouldn't use FFMI to place restrictive, arbitrary limits on yourself or to make baseless steroid accusations about others, but you can use it as a tool to help plan your weight gain phases.

and values above 24 are likely a stretch for a lot of females. However, if you have good genetics and tend to respond well to training, you may be able to aim higher.

Limitations of FFMI studies

Whenever you read a paper that intends to establish normative values or upper limits for FFMI, there are a few key considerations to keep in mind. It's certainly important to consider whether or not steroid users were effectively excluded from participation. Many studies measure athletes that are subject to various drug-testing procedures, but it's always important to realistically consider how many drug users could have theoretically slipped through the cracks. The second (and most important) question is, "Did this sample include any freaks?" And I can assure you, I mean "freak" in the best possible way. If we want to make inferences about upper limits for muscularity, we have to make sure the sample is large enough and contains people who are genetically gifted enough and well-trained enough to at least be close to the top end of human potential. That's a lofty goal, so most samples fall short, with some falling way shorter than others.

There are also plenty of inherent limitations for the use of FFMI. As we've discussed, it's slightly biased toward higher values in taller people. It fails to directly account for differences in the density of fat-free mass, which varies on the basis of age, sex, race, and several other factors. It fails to directly account for the fact that, at least to some degree, lean mass and fat mass are gained and lost in unison; this is why you can find plenty of Sumo wrestlers with values well into the mid-30s (9), but you're unlikely to find much of that among a sample of contest-ready natural bodybuilders. Finally, as noted previously, there are several methods for trying to estimate an individual's genetic ceiling for muscle gain, and they each have pros and cons. Fat-free mass index is not a perfect proxy for muscularity, but it seems to generally get the job done.

Despite some limitations, FFMI can be useful for planning purposes. However, it's really important to remember what FFMI is *not* good for: imposing limits on yourself and making baseless steroid accusations.

Next Steps

Fat-free mass index is pretty straightforward, so complex study designs aren't really needed at this time. Instead, we need more data using large data sets, with samples that include people who have been hitting the weights hard for a long time, and (ideally) some genetically gifted lifters. As more studies like this come out, we should continue to develop a better understanding of exactly how big the typical male or female lifter can realistically hope to get over the course of their lifting career.

References

1. Harty PS, Zabriskie HA, Stecker RA, Currier BS, Moon JM, Jagim AR, et al. Upper and lower thresholds of fat-free mass index in a large cohort of female collegiate athletes. J Sports Sci. 2019 Oct;37(20):2381–8.
2. Kouri EM, Pope HG, Katz DL, Oliva P. Fat-free mass index in users and nonusers of anabolic-androgenic steroids. Clin J Sport Med Off J Can Acad Sport Med. 1995 Oct;5(4):223–8.
3. Trexler ET, Smith-Ryan AE, Blue MNM, Schumacher RM, Mayhew JL, Mann JB, et al. Fat-Free Mass Index in NCAA Division I and II Collegiate American Football Players. J Strength Cond Res. 2017;31(10):2719–27.
4. Mountjoy M, Sundgot-Borgen JK, Burke LM, Ackerman KE, Blauwet C, Constantini N, et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. Br J Sports Med. 2018 Jun 1;52(11):687–97.
5. Rosen LW, Hough DO. Pathogenic Weight-Control Behaviors of Female College Gymnasts. Phys Sportsmed. 1988 Sep;16(9):140–4.
6. Blue MNM, Hirsch KR, Pihoker AA, Trexler ET, Smith-Ryan AE. Normative fat-free mass index values for a diverse sample of collegiate female athletes. J Sports Sci. 2019 Aug;37(15):1741–5.
7. Currier BS, Harty PS, Zabriskie HA, Stecker RA, Moon JM, Jagim AR, et al. Fat-Free Mass Index in a Diverse Sample of Male Collegiate Athletes. J Strength Cond Res. 2019 Jun;33(6):1474–9.
8. Abe T, Buckner SL, Dankel SJ, Jessee MB, Mattocks KT, Mouser JG, et al. Skeletal muscle mass in human athletes: What is the upper limit? Am J Hum Biol Off J Hum Biol Counc. 2018;30(3):e23102.
9. Hattori K, Kondo M, Abe T, Tanaka S, Fukunaga T. Hierarchical differences in body composition of professional Sumo wrestlers. Ann Hum Biol. 1999 Apr;26(2):179–84.

Study Reviewed: The Placebo and Nocebo Effect on Sports Performance: A Systematic Review. Hurst et al. (2019)

The Placebo Effect Impacts Performance More Than You Might Expect

BY GREG NUCKOLS

The placebo effect is a well-known psychological phenomenon, but we often forget about it in exercise research. We focus on how much a supplement, device, or treatment improves performance relative to a placebo, but a lot of the "real-world" improvements in performance may actually be attributable to placebo effects.

KEY POINTS

1. In a meta-analysis, placebo and nocebo effects both have small but meaningful impacts on performance.
2. The placebo effect is larger if someone thinks they're ingesting a banned substance, such as anabolic steroids or erythropoietin (EPO).
3. Interestingly, for caffeine, up to two-thirds of the "real-world" increase in performance with caffeine usage may be attributable to the placebo effect (though caffeine itself absolutely still has a real physiological effect).

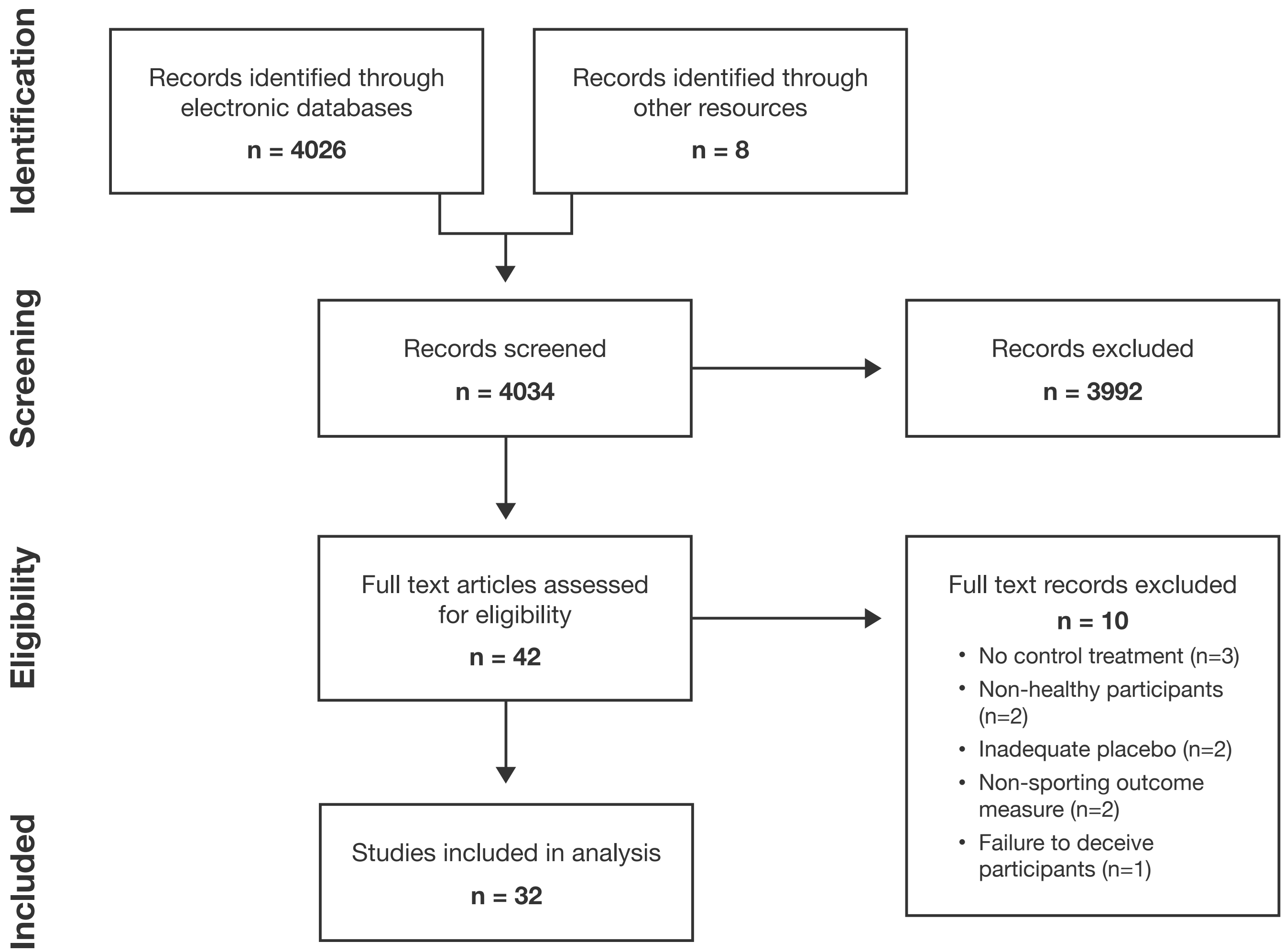
Any time a new supplement study drops, savvy readers know that one of the first things to check is whether the study was placebo-controlled. Placebo controls are important, because part of the effect you get from any treatment is the effect you get from simply receiving a treatment, even if that treatment doesn't actually do anything. If you can do 10 reps with a given weight without taking a pill, 12 reps with a sugar pill, and 13 reps with a caffeine pill, then simply taking a pill gives you 2 reps, with caffeine only giving you 1 extra rep, not 3.

We mostly focus on how much a given supplement, device, or treatment improves performance in excess of the boost provided by a placebo. We generally pay less attention to the boost provided by the placebo itself. However, a recent meta-analysis (1) fills that gap, analyzing the research that has investigated the magnitude of the placebo effect on exercise performance. Overall, it seems that placebo and nocebo effects (the placebo effect's evil twin – worsening performance when given a treatment you expect to harm performance) have a small but notable effect on physical performance. Furthermore, when people think they're consuming a banned substance, or when they're manipulated into believing the placebo has already provided them a performance boost, the placebo effect is even larger.

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

Figure 1 Exclusion process of identified studies



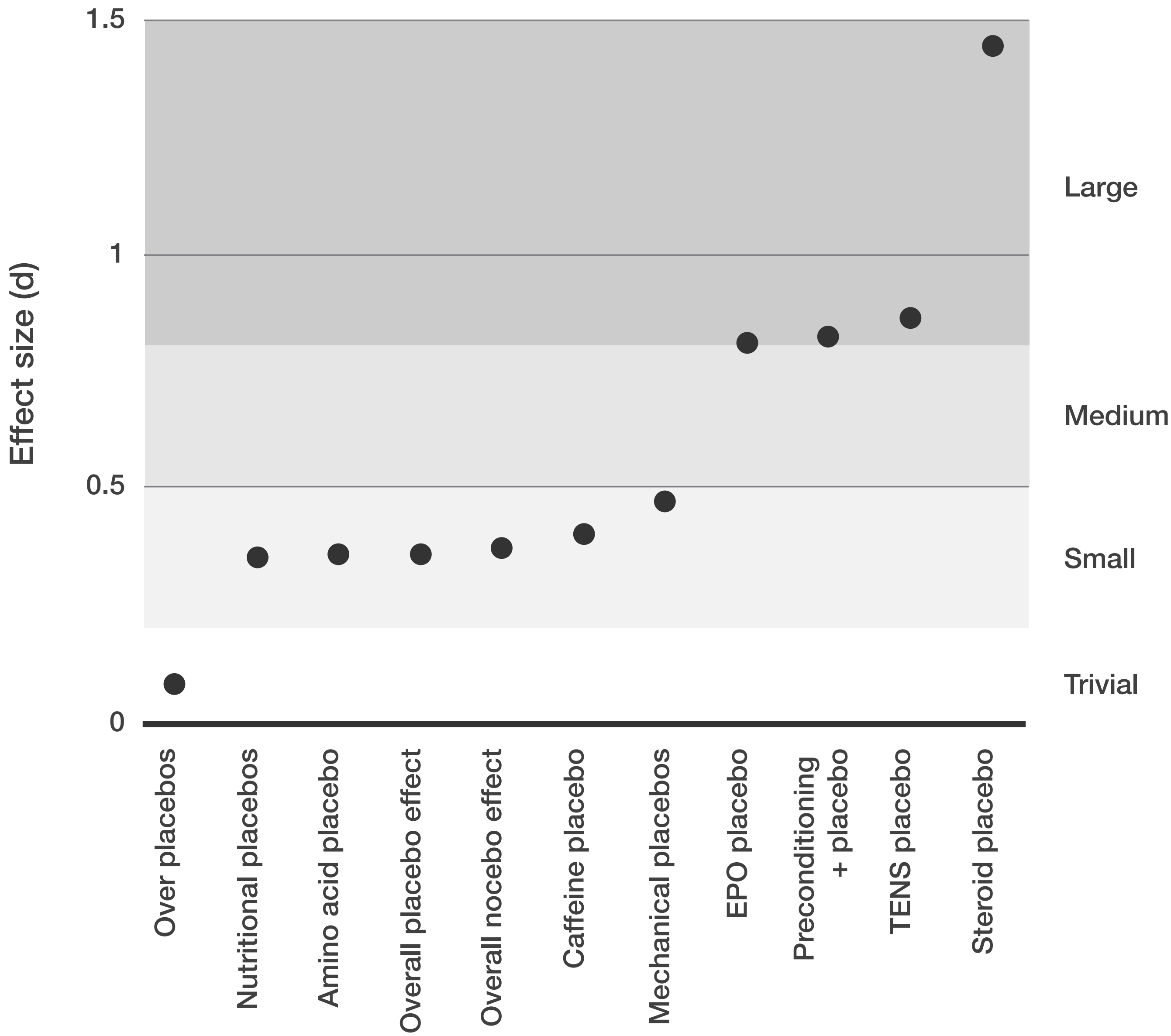
Purpose and Hypotheses

Since this was a meta-analysis, the purpose was simply to statistically analyze all of the research investigating the effects of placebos and nocebos on exercise performance. No hypotheses are stated for meta-analyses.

Methods

The authors ran a search for studies investigating the effects of placebos and nocebos on physical performance. To be included in the meta-analysis, a study needed to be published in an English-language peer-reviewed journal, include healthy subjects, and re-

Figure 2 Effect sizes for various placebo interventions



port at least one objective measure of performance (i.e. not just pain, fatigue, or perceived exertion). Furthermore, to quantify the placebo effect, studies needed to include a no-treatment control condition or measurement.

Once the studies were collected, the authors calculated effect sizes for each study and "aggregated" them. The authors don't actually explain how they performed the meta-analysis itself (Fixed effects model? Random effects model? Simply taking a weighted average of the effect sizes?), which is odd.

ONE OF THE MOST LOGICAL AND INTERESTING FINDINGS OF THIS META-ANALYSIS WAS THE MORE "SERIOUS" PLACEBOS INDUCED LARGER PLACEBO EFFECTS.

Findings

More than 4,000 studies were screened, with 32 eventually meeting the inclusion criteria, representing 1,513 total participants. Of those 32 studies, 20 investigated nutritional placebos or nocebos, while 12 investigated mechanical placebos. Most of the studies investigated the placebo effect, while only 5 examined the nocebo effect. Of the studies using placebos, 4 used overt placebos (i.e. they told the subjects when they were giving them a placebo), while 5 augmented the placebos with preconditioning via augmented feedback (which I'll explain in the next section).

Overall, nutritional and mechanical ergogenic aids significantly improved performance. The effect sizes for both were small (d = 0.35 for nutritional placebos, and d = 0.47 for mechanical placebos). Unsurprisingly, the placebo effects generated by placebos claiming to be banned substances were larger (d = 1.44 for steroids, and d = 0.81 for EPO). Interestingly, preconditioning procedures also had a large effect on performance (d = 0.82). Sham transcutaneous electrical nerve stimulation (TENS) was also reported to have a large effect size (d = 0.86). Small effect sizes were noted for placebo amino acids and caffeine (d = 0.36 and 0.40, respectively). A completely fictitious sport supplement was found to have a small effect (d = 0.21). Cold water immersion, sodium bicarbonate, ischemic preconditioning, carbohydrate, beta alanine, kinesio tape, and magnetic wristbands were all found to have trivial or null effects. Overt placebos also had no significant effect.

The nocebo effect was also small (d = 0.37). Since there were only five studies investigating the nocebo effect, the researchers didn't investigate whether the type of nocebo used moderated the outcomes.

Interpretation

The first thing I'd like to reiterate is that the authors don't make it clear how they actually performed their meta-analysis, so it's hard to tell if they mucked something up. I *think* they just extracted the effect size from each study and either averaged them or took a weighted average. If that's what they did, their point estimates for each effect size may be pretty alright (just skimming the reported effect sizes, it doesn't seem like there's an extreme amount of variability within each sub-category), but they may have either more or less variance around the mean estimate than they "should." There are a few other statistical things I could complain about, but I feel like I probably do too much of that anyways, and the things I want to complain about probably wouldn't meaningfully change my interpretation of this meta-analysis. However, we probably need to be a little more cautious with these findings than we would otherwise need to be if their statistical approach was more transparent.

One of the most logical and interesting findings of this meta-analysis was the more "serious" placebos induced larger placebo effects. Since the placebo effect is a psychological phenomenon based on expectancy (you think what you're taking will improve performance, and that belief is what actually improves performance), it makes since that placebos that you *think* will have a larger effect do actually have a larger effect. I think just about anyone would expect to improve their strength when taking steroids, or improve their endurance performance when taking EPO; those substances are banned in most competition because they do cause large improvements in performance, after all. And though just three of the studies in this meta-analysis investigated banned substances (2 for steroids, 1 for EPO; 2, 3, 4, 5[NOTE]), all three of those studies reported large effects, and the mean effect for those studies (d = 1.23) was more than three times larger than the mean effect for all of the studies in this meta-analysis (d = 0.37). I've always wondered about the degree to which the increase in strength people report when they start taking steroids is attributable to expectancy. People often claim that they build a lot more muscle and experience a disproportionate increase in strength. However, in placebo-controlled research (i.e. when people who aren't taking steroids are still given injections to make them think they're taking steroids), it seems that the boost steroids provide for strength gains is disproportionately smaller than the boost they provide for hypertrophy (6), with steroids helping subjects build 3.3 times more muscle (4.23% vs. 13.92% increases in combined triceps and quadriceps cross-sectional area), while only helping them build 1.7 times more strength (35 vs. 60 kg combined

> THE PLACEBO EFFECT OF CAFFEINE MAY BE JUST AS LARGE AS THE "TRUE" EFFECT OF CAFFEINE, IF NOT LARGER.

AS A GENERAL RULE, IT PAYS TO BE OPTIMISTIC; IF YOU EXPECT TO PERFORM WELL AND HAVE GOOD RESULTS FROM YOUR TRAINING, YOU PROBABLY WILL, AND IF YOU EXPECT TO PERFORM POORLY AND HAVE LACKLUSTER RESULTS, THAT WILL LIKELY BECOME A SELF-FULFILLING PROPHECY.

increase in squat and bench press 1RM). Since this meta-analysis found that the placebo effect itself generates a large improvement in performance when people think they're taking banned substances, it does make me think the reports of large, fast strength increases in strength reported when people start taking steroids may be based as much on expectancy as the actual physiological effects of the drugs.

Another interesting note is that the placebo effect of caffeine may be just as large as the "true" effect of caffeine, if not larger. Meta-analyses find that caffeine improves performance relative to placebo, with small effect sizes in the 0.2-0.4 range (7). This meta-analysis found that the placebo effect for caffeine was associated with an effect size of about 0.4. Thus, in the "real world," when people take caffeine before a workout, the total effect may actually be quite large (i.e. in the d = 0.6-0.8 range), with about one-half to two-thirds of the effect attributable to expectancy, and about one-third to one-half of the effect attributable to the actual physiological effects of caffeine.

I was surprised that placebo TENS treatment was so effective. TENS units consist of electrodes that are placed on the skin, with a current passed through the electrodes that is sufficient to stimulate the underlying nerves, but generally low enough that it does not cause muscular contraction. Generally you can feel a TENS unit working (it tingles at low voltage and can be mildly uncomfortable at higher voltages), though you can't feel the current if the voltage is low enough. For placebo TENS treatment, you turn the unit on, and you may even rig it up so that it will show that it's operating at a low voltage, but you don't run a current through it. While I suppose the show of placing the electrodes and turning the machine on may represent a "bigger" placebo treatment than simply giving someone an unmarked pill, I would have thought that people

APPLICATION AND TAKEAWAYS

The placebo effect has a small but notable effect on performance under most circumstances. While it's hard to placebo yourself, expectancy effects in general can influence performance, so try to maintain positive self-talk about your training, and try to avoid catastrophizing things that happen in your life, as doing so could harm performance by triggering negative expectancy effects.

would be *highly* skeptical that the machine was really doing anything if they didn't feel anything. However, it does appear that sham TENS treatment is quite an effective placebo. I doubt MASS readers can really do much with that information, but I thought it was cool.

The finding that preconditioning caused a large improvement in performance is fascinating. For a positive preconditioning study, you generally test subjects at baseline, test them again when giving them a placebo, while altering the test to make the subjects feel like they performed better, and then test them another time with the placebo but without the test alteration. For example, you could test someone's maximal bench press reps with 100kg on day 1. 48 hours later, you could give them a placebo pill, tell them it's caffeine, and test their maximal bench press reps with 90kg, while telling them it's 100kg (with altered plates so the bar still looks like it's loaded to 100kg). Their performance should be better, which they'll attribute to the placebo pill. 48 hours later, you give them the placebo pill again, and test their maximal bench press reps with 100kg. Since they already have the belief that the placebo pill (which they think is caffeine) improves their performance, they'll probably perform much better on this test with 100kg than they would have if you hadn't preconditioned them (i.e. session 1: 100kg with no placebo; session 2: 100kg with placebo). Preconditioning essentially works to amplify expectancy, and this meta-analysis found that when preconditioning is combined with a placebo treatment, the total effect is (on average) more than twice as large as the effect of the placebo alone.

Predictably, overt placebos didn't impact performance. An overt placebo is a placebo someone knows they're getting. If you give someone a pill and say, "this is a placebo pill that does nothing," that's an overt placebo (sometimes called an open label placebo). It may sound ludicrous that the effects of overt placebos even need to be researched, but interestingly, overt placebos have been found to improve subjective outcomes (such as pain or nausea) compared to a no-treatment control (8), which I find fascinating. However, when you're objectively measuring exercise performance, people don't get a placebo effect when they know they're taking a placebo.

Finally, I want to draw attention to the fact that the mean nocebo effect was just as large as the mean placebo effect. When people think something will improve performance, it generally does, and when people think something will harm performance it generally does. In a previous MASS article, I reviewed a study showing that in some cases, beliefs about your genetics can affect your physiology and perceptions more than your actual genetics themselves do (10). I'm not sure how relevant that finding is in this particular meta-analysis (I don't think many people intentionally take supplements or use interventions that they think will hinder their performance), but negative expectancy effects are a pretty generalizable concept. It's something to keep in mind with your self-talk and the interactions you have with your clients. For example, if you expect that your strength is going to drop when you go into a calorie deficit, it probably will. It may have done so anyways, but the drop will likely be larger if you expect it to take a hit. Or if you expect a night of bad sleep to hinder performance, your performance will probably be lower (even though research tends to find that one night of bad sleep doesn't have much of an impact on acute performance; it tends to negatively affect skill performance and tactical decision-making, but not things like force output or endurance [9]). As a general rule, it pays to be optimistic; if you expect to perform well and have good results from your training, you probably will, and if you expect to perform poorly and have lackluster results, that will likely become a self-fulfilling prophecy.

Next Steps

I'm still stuck on the idea of the placebo effect of steroids. A deception study would be really cool, involving four groups. All four groups are given pills. Group 1 is given oral steroids and are told they're being given oral steroids. Group 2 is given oral steroids and are told they're being given an inert control. Group 3 is given an inert control and are told they're being given oral steroids. Group 4 is given an inert control and are told they're being given an inert control. Since this meta-analysis found that overt placebos don't improve exercise performance, group 2 would show us the "true" physiological effect of steroids, group 3 would show us the placebo effect of thinking you're taking steroids, group 1 would show us the "stacked" effect (placebo + actual physiological effect), and group 4 would be the control group. I would predict that groups 1 and 2 would easily gain the most muscle, but I wouldn't be shocked if groups 2 and 3 gained similar amounts of strength.

References

1. Hurst P, Schipof-Godart L, Szabo A, Raglin J, Hettinga F, Roelands B, Lane A, Foad A, Coleman D, Beedie C. The Placebo and Nocebo effect on sports performance: A systematic review. Eur J Sport Sci. 2019 Aug 20:1-14.
2. Ariel G, Saville W. Effect of anabolic steroids on reflex components. J Appl Physiol. 1972 Jun;32(6):795-7.
3. Maganaris CN, Collins D, Sharp M. Expectancy Effects and Strength Training: Do Steroids Make a Difference? The Sport Psychologist. 200;14(3):272-278.
4. Heuberger JAAC, Rotmans JI, Gal P, Stuurman FE, van 't Westende J, Post TE, Daniels JMA, Moerland M, van Veldhoven PLJ, de Kam ML, Ram H, de Hon O, Posthuma JJ, Burggraaf J, Cohen AF. Effects of erythropoietin on cycling performance of well trained cyclists: a double-blind, randomised, placebo-controlled trial. Lancet Haematol. 2017 Aug;4(8):e374-e386.
5. I think the authors accidentally cited the wrong study for EPO. I think citation 4 above is the study they meant to cite.
6. Bhasin S, Storer TW, Berman N, Callegari C, Clevenger B, Phillips J, Bunnell TJ, Tricker R, Shirazi A, Casaburi R. The effects of supraphysiologic doses of testosterone on muscle size and strength in normal men. N Engl J Med. 1996 Jul 4;335(1):1-7.
7. Grgic J, Grgic I, Pickering C, Schoenfeld BJ, Bishop DJ, Pedisic Z. Wake up and smell the coffee: caffeine supplementation and exercise performance-an umbrella review of 21 published meta-analyses. Br J Sports Med. 2019 Mar 29. pii: bjsports-2018-100278.
8. Charlesworth JEG, Petkovic G, Kelley JM, Hunter M, Onakpoya I, Roberts N, Miller FG, Howick J. Effects of placebos without deception compared with no treatment: A systematic review and meta-analysis. J Evid Based Med. 2017 May;10(2):97-107.
9. Kirschen GW, Jones JJ, Hale L. The Impact of Sleep Duration on Performance Among Competitive Athletes: A Systematic Literature Review. Clin J Sport Med. 2018 Jun 14.
10. Turnwald BP, Goyer JP, Boles DZ, Silder A, Delp SL, Crum AJ. Learning one's genetic risk changes physiology independent of actual genetic risk. Nature Human Behavior. 2019; 3:48-56.

Concept Review:
Using the RIR-Based RPE Scale in Your Training

RPE and RIR: The Complete Guide

BY MICHAEL C. ZOURDOS

MASS and many others have discussed RPE at length. However, there are many uses of RPE outside of just basic load prescription. This article gives a brief historical context and then provides a section and example of every single way that RPE has been used in the literature.

KEY POINTS

1. Although most of us know rating of perceived exertion (RPE) as a scale that measures repetitions in reserve (RIR) during a set of resistance training, that is a relatively new usage of the term.
2. There are many ways to use RPE outside of just basic load assignment. These strategies include autoregulating volume, load progression over time, tracking progress, and predicting a 1RM.
3. While there are limitations and valid critiques of using RPE, using the RIR-based RPE scale requires little effort and comes at no cost. In most cases, even if RPEs are not perfectly accurate, they are still quite useful.

In today's fitness industry, we tend to discuss RPE as it relates to RIR during a resistance training set. While the RIR-based RPE scale is the focus of this article, we should understand that the concept of RPE has been around since before the launch of Apple, Inc. in the early 1970s. The *original* RPE scale was created by Gunnar Borg and was designed to gauge light, moderate, and heavy efforts during aerobic training (1). RPE, as most lifters use it today, is quite new when considering the historical context and actually out of step with mainstream academics. Nonetheless, the popularity of RIR-based RPE has exploded over the last 10+ years due to its great utility and ease of use. However, RPE tends to be viewed in a binary fashion as either "RPE training is good" or "RPE training is bad." This binary view tends to only take into account using RPE for basic load prescription because the ratings are subjective. The criticism of subjectivity is fair; however, what many still seem to be unaware of is that the utility of RPE extends far beyond basic load prescription. In this concept review, I aim to provide detailed insight into every possible way to use RPE including: load prescription, autoregulating volume, predicting 1RM, tracking progress over time, and weekly load/set/rep progression, among other uses. This article will

What's a concept review?

A written concept review is similar to our signature video reviews. The aim of this article type is to review a cornerstone topic in physiology or applied science research.

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

provide specific practical examples in the form of tables for each use, so that you can directly implement these strategies into training programs for yourself or your clients. This article will also discuss some of the common criticisms of RPE and how someone can improve their rating accuracy. First, let's start with the aforementioned historical context.

Brief History

Traditionally, RPE has been used in a different context than how the fitness community uses it today. Originally, Gunnar Borg created a 6-20 RPE scale in 1970 (1), during Helms's freshman year of high school. The scale was created for aerobic exercise, and the ratings were intended to correspond with a runner's heart rate. For example, the idea was that a rating of 6 would correspond to a heart rate of 60 beats per minute, and 20 with 200 beats per minute; this range of 60 to 200 beats per minute roughly represents a typical spectrum from resting to maximal heart rate. Therefore, the original iteration of RPE was to gauge effort levels during aerobic exercise. Borg then added the C (category) R (Ratio) 10 (CR10) scale in 1982 (2), which gauged effort levels in a simpler 0-10 rating system. Both Borg scales accomplished the same thing, which was allowing aerobic exercisers to gauge a general sense of light, moderate, or heavy effort. However, for resistance training, the application is relatively limited. Fast forward to 2006 and the OMNI scale was created for resistance exercise. The 1-10 OMNI scale was very similar to the CR10 Borg Scale except visual descriptors were added to the "easy," "moderate," and "heavy" descriptors. While the visual descriptors were an improvement, these RPE scales still lacked precision for resistance training. The two original Borg scales can be seen in Tables 1 and 2.

Table 1 Borg 6-20 Scale

RPE Score	Exertion Level
6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

Adapted from Borg (1) 1970
RPE = Rating of Perceived Exertion

The lack of precision of the Borg scales for resistance training was first revealed in the scientific literature in 2012 from Hackett et al (3). Hackett had bodybuilders perform 5 sets to failure at 70% of 1RM on the squat and bench. After 10 reps on each set, the bodybuilders recorded both a Borg RPE (i.e. light, moderate, or heavy effort), noted how many repetitions they felt they had left, and then continued the set to failure. The bodybuilders estimated their reps left until failure (what we now call RIR) with pretty good accuracy; however, they tended to record moderate effort even when going to failure on the Borg scale. Thus, it was concluded that the Borg or "traditional RPE" scales lacked utility for resistance training, as lifters tend to gauge cardiovascular effort when looking at the descriptors (Tables 1 and 2 above) on traditional scales. Although the Hackett study was the first time this concept had been broached in the scientific literature, it would be inaccurate to say that this was the first time it had been presented in general. In fact, around the time of his gold medal powerlifting performance at the World Games, Mike Tuchscherer published the Reactive Training Systems Manual (4), in which he created the idea of having RIR descriptors for RPE. Our laboratory took the work of Tuchscherer and Hackett et al and formalized the RIR-based RPE scale within the scientific literature in 2016 by validating it against velocity (Table 3) (5). The idea, however,

Table 2 Borg CR10 scale

RPE Score	Exertion Level
0	Nothing at all
0.5	Extremely light/weak
1	Very weak / light
2	Weak / light
3	Moderate
4	Somewhat strong
5	Strong (heavy)
6	
7	
8	Very strong
9	
10	Extremely strong
*	Maximal

Adapted from Borg (2) 1982
RPE = Rating of Perceived Exertion

Table 3 Resistance training-specific RIR-based RPE scale

RPE Score	RIR / Description
10	Maximal effort
9.5	No RIR, but could increase load
9	1RIR
8.5	Definitely 1, maybe 2RIR
8	2RIR
7.5	Definitely 2, maybe 3RIR
7	3RIR
5-6	4-6 RIR
3-4	Light effort
1-2	Little to no effort

Adapted from Zourdos et al. (5) 2016
RPE = Rating of Perceived Exertion; RIR = Repetitions in Reserve

originates with Mike T., and it's important to make sure he gets the credit. Further, this is an excellent example of how the practical realm is sometimes ahead of science and how what is already done in practice can, and sometimes should, impact scientific research.

Although not the focus of this concept review, I would be remiss to exclude in this historical discussion the usage of the session RPE scale. Session RPE was brought to the forefront by Dr. Carl Foster in 2001 (6). It's a 0-10 scale typically administered 30 minutes following exercise and asks the individual about their global fatigue level using descriptors such as: "easy," "moderate," "hard," and "maximal." Let's look at an example of the utility of this scale in the context of resistance training: If two programs produced the same long-term hypertrophy and strength, but one produced a lower session RPE, then it might make sense to recommend the program that caused less fatigue or even consider adding volume or intensity to the less-fatiguing program (we've previously written about this).

Recently, I've seen people suggest there could be a difference in RPE and RIR. In reality, if using the RIR-based RPE scale, there isn't a difference. They are the same. However, historically, there is indeed a difference, but this doesn't mean that the terminology of "RPE" and "RIR" won't have different connotations to people even though the scale treats them the same way. Toward the end of this article, we will return to the terminology and discuss how sometimes it may be better to use one term over the other, even if they are technically referring to the same thing. Now that we understand the history, the remainder of this article will focus on the exact usage of the RIR-based RPE scale and will be chock-full of practical examples.

Specific Ways to Implement RPE

The remainder of this article will explain every way in which RPE can be used so that you can implement it into your training in the appropriate manner. Before we get to each subsection, please remember that RPE is just one form of autoregulation, which is defined as gathering feedback about training to make informed decisions (7). For example, a form of autoregulation outside of RPE is flexible training templates. Additionally, thanks to two recent studies, one from Dr. Helms (8) and the other from Graham and Cleather (9 – reviewed in MASS), we know that autoregulating training load with RPE/RIR over the long-term is also a good idea for strength when compared strictly to percentages. Let's get started with a breakdown of each way to use RPE, starting with the most basic.

Basic Load Prescription with RPE

Table 4 Basic RPE loading example

	Mesocycle 1 (About 4-6 weeks)	Mesocycle 2 (About 4-6 weeks)	Mesocycle 3 (About 4-6 weeks)	Mesocycle 4 (About 4-6 weeks)
Day 1	3 x 10 @ 5-7 RPE	3 x 8 @ 6-8 RPE	3 x 6 @7-9 RPE	3 x 5 @8-9.5 RPE
Day 2	4 x 8 @ 5-7 RPE	4 x 6 @ 6-8 RPE	4 x 4 @7-9 RPE	3 x 3 @8-9.5 RPE
Day 3	5 x 6 @ 5-7 RPE	5 x 4 @ 6-8 RPE	4 x 2 @7-9 RPE	Single @9.5 RPE

RPE = Rating of Perceived Exertion

The most basic way to implement RPE is to simply replace percentage-based load prescription with RPE-based load prescription. For example, instead of programming 4 sets of 8 at 70% on a compound movement, you could program 4 sets of 8 at 6-8 RPE. This means that you would simply choose a load that would land you within the 6-8 RPE range. The utility of this is that since strength fluctuates daily due to various readiness factors (i.e. poor sleep, anxiety, scheduling issues, etc.), using RPE allows you to increase or decrease the load as needed. In the same way, RPE also allows you to lift heavier when you are feeling good. In fact, the Helms (8) and Graham and Cleather (9) studies found that allowing lifers to choose a load based upon RPE allowed subjects to train at a higher average intensity throughout the respective 8- and 12-week training studies, which led to greater 1RM strength in various compound movements.

Additionally, the number of reps that can be performed at a given intensity is highly individual. Recent studies have reported ranges of 6-28 (average = 16 ± 4) (10) and 6-26 (average = 14 ± 4) (11) reps performed on the squat at 70% of 1RM. Therefore, replacing percentages with RPE can account for the between-individual differences. Even if you like to use percentages as a coach, using RPE in the first few weeks with a new client (if they are well-trained and reasonably accurate with RPE) will give you an idea of how many reps a lifter can do at a certain percentage. Then, based on how many reps are performed at a specific load, you can now individualize a percentage program. Table 4 shows a basic RPE load prescription example.

Table 4 is just one example of a basic RPE-based load prescription. The example is quite practical, as it takes into account long-term periodization and programming strategies outlined here, in that there is an overall decrease in the number of repetitions (i.e. decrease in volume) and an increase in RPE as intensity increases from block to block.

When incorporating assistance work with the main lifts in the above example, I would generally follow a similar RPE format, but keep the reps a bit higher so that you are not doing triples and singles on single-joint movements in the final mesocycle. Overall, this example takes into account day-to-day fluctuations in strength levels, the individual ability to perform reps, and it allows for intra-session load adjustments. When adjusting load intra-session, if you miss the RPE target on a set, I think it's good to have a guide for how to adjust load. Therefore, in Table 5, I have included the table from Dr. Helms's dissertation that provides this load adjustment guide. You don't have to follow this table exactly, but it provides the general idea of how to change the load for the next set when you miss the target RPE range. Lastly, I find it a good idea to include an RPE range as your target rather than an exact RPE target when performing multiple sets with RPE-load prescription, as it will become difficult to hit an exact RPE target each time. Besides, it's just not that important to maintain an exact proximity to failure; rather, it's important to understand the intent of RPE, which is generally to either be far from failure, a few reps from failure, or at failure. A range saves you from having to adjust the load every single set. Table 5 essentially stipulates that if your set is within the RPE range, you can choose what you would like to do for your next set; however, for every 0.5 RPE points outside of the range your set lands, then you should adjust the load 2% up or down for your next set. Table 5 uses an example of a target RPE range of 6-8, but of course this concept can be applied with any target RPE range.

Table 5 Basic load adjustment chart if missing the RPE range

Actual RPE	Next set load adjustment if assigned RPE range is 6-8
1	Increase load by 20%
2	Increase load by 16%
3	Increase load by 12%
4	Increase load by 8%
5	Increase load by 4%
6	Lifter's choice
7	Lifter's choice
7.5	Lifter's choice
8	Lifter's choice
8.5	Decrease load by 2%
9	Decrease load by 4%
9.5	Decrease load by 6%
10	Decrease load by 8%

Adapted from Helms et al. 2018
RPE = Rating of Perceived Exertion

Autoregulating Volume (RPE Stop)

Autoregulation can also be implemented to achieve the appropriate volume. Using RPE to autoregulate volume has been described using the term "RPE stop." You can implement RPE stops in two ways: 1) to autoregulate the number of reps in a set, or 2) to autoregulate the number of sets in a session for a specific exercise. These strategies are similar to velocity loss (12), which we have discussed before.

Table 6 RPE stop examples

RPE stop method 1 (Autoregulating reps in a set)	RPE stop method 2 (Autoregulating total sets)
1. Choose exercise (squat)	1. Choose exercise (squat)
2. Choose load (let's use 70% of 1RM for volume)	2. Choose load (let's use 70% of 1RM for volume)
3. Choose RPE to stop at per set (let's use 6-8 for volume and to avoid fatigue spilling into next session)	3. Choose number of reps per set (let's use 8)
4. Choose number of sets, which fits within your individual volume needs (let's use 4 sets)	4. Choose RPE to stop squatting for the day (let's use 8.5, assuming this person can do more than 12 reps at 70%)
	5. Choose a number of sets to cap the volume (let's use 5)
Programming for the day 4 sets of squats at 70% of 1RM and stop each set @6-8 RPE	**Programming for the day** Sets of 8 reps on squats at 70% of 1RM for as many sets (up to 5) until an 8.5RPE is reached

To implement the first strategy and autoregulate reps in a set with an RPE stop, you would pick a percentage or an exact weight and do as many reps as possible on each set but stop each set when you reach a predetermined RPE. If aiming to accumulate volume, you would stop at about a 7-8RPE, and if training with low volume at high intensities (i.e. 2-4 reps at 85-90% of 1RM), you may stop a set at around a 9 RPE. I actually think RPE stops have utility over velocity loss. Specifically, and as we pointed out last month, if your first rep on a 70% of 1RM squat set is 0.65 m/s, a 40% loss puts you at 0.39 m/s, which probably lands someone between a 5 and 8 RPE. However, your first (or fastest rep velocity) will be lower on subsequent sets. So, let's say on your fourth set, your first rep velocity is 0.55 m/s, then a 40% velocity loss has you stopping the set at 0.33 m/s, which is much closer to failure than the 0.39 m/s on the first set. Therefore, an RPE stop will always have you stop the set at the desired amount of RIR (assuming the RPE is accurate), and it is inherently individualized, whereas individual velocity profiles must be determined, otherwise velocity can be misapplied across a group.

The second usage of an RPE stop is to autoregulate total set volume. In this model, we take a certain load (i.e. 70% of 1RM) and perform 8 reps per set, but without a predetermined number of sets. Now, we would perform as many sets as

Table 7 Total reps and rest-pause example

Total reps and rest-pause	Results, reps after each set
1. Choose exercise (dumbbell shoulder press)	**Set 1** 11 reps
2. Choose load (if you are in a volume block, maybe choose something you can do for a 12-15RM)	**Set 2** 20 reps
3. Choose RPE to stop at per set (let's use 8-9 RPE)	**Set 3** 28 reps
4. Choose total number of reps (let's use 35)	**Set 4** 35 reps

To do the above rest-pause style just take 20-30 seconds between sets and this will serve as a really efficient time saving strategy when needed.
RM = repetition maximum

we can until we exceed a predetermined RPE, let's say an 8 RPE. I would recommend capping the number of sets so that you don't end up performing 10 sets of high rep squats one session if you have a high work capacity. This might be 5 sets for compound lifts when accumulating volume (i.e. moderate reps and lower peak RPE) or 3 sets for compound lifts when in an intensity block (i.e. low reps and high peak RPE). Of course, the amount of sets is also dependent on an individual's recovery. Table 6 shows an example of both RPE stop methods.

Another advantage of RPE stops is that they have built-in progressive overload. For example, in method one, if your goal is volume you can just stick with the same weight for a few weeks and aim for more reps. Then, once you hit a certain number of total reps across all 4 sets (as per the example), you can increase the load. In method two, if your goal is 5 sets at an 8 RPE or less, you can continue to perform this load each week until you complete all 5 sets successfully, then increase the load.

Total Reps and Rest-Pause with RPE

For assistance work, RPE can be just as valuable as it is for the main lifts. Even though strictly using generalized percentages isn't great for programming the main lifts, a lifter could create an individualized percentage chart. However, for assistance work, we typically do not know our 1RM; thus, we are left with our perception of difficulty to determine loading. For assistance work, you can certainly use basic load assignment (Table 4); however, a total rep or non-failure rest-pause strategy works well and

Table 8 Using RPE to predict daily 1RM or gauge progress			
Method	**Training example**	**Application**	**Outcome**
Predicting intensity or a daily 1RM	150kg squat for 1 rep @9RPE	1 rep @9RPE is about 95%. Thus, divide 150 / 0.95	157.5 kg can be concluded as the daily 1RM
Gauging strength progress over time	150kg squat for 1 rep @9RPE	After 8 weeks of training, 150kg is squatted for 1 rep @7RPE	We can conclude progress and projected 1RM went from 157.5 kg to 172.5 kg
Gauging volume progress over time	Completed 3 sets of 8 at 105kg on squat without exceeding 8	After 4 weeks of training, 6 sets of 8 at 105kg was completed with a last set RPE of 7	We can conclude significant volume progress and greater hypertrophy should follow over time

is similar to an RPE stop. For example, if your 12RM on dumbbell bench press is about 40kg, you could set a threshold of 35 total reps and perform each set to an 8-9 RPE until you reach the threshold of 35 reps. In week 1 of your training block, it might take you 4 sets to reach the 35 reps. You could continue each week with the same load until you reach the 35 reps in 3 sets, then increase the total reps threshold or increase the load to achieve progressive overload. The above example can also be used in the rest-pause variety, just simply take 20-30 seconds between sets. Using RPE during rest-pause allows you to avoid the typical failure training associated with rest-pause training, so it should cause less fatigue in the 48 hours following training than going to failure (13). In reality, the above examples are really variations of RPE stops, but as this is a concept review, I wanted to clearly explain every possible RPE load assignment strategy.

Tracking Progress and Predicting a 1RM

Something I've hit on before in MASS is that even if you don't use RPE to program load, you should still track it. Tracking RPE would itself be scored as an RPE 1, meaning it takes little to no effort to do, and it provides you with a gauge of progress over time. From one week to the next, one block to the next, or even year after year, you can look back and say "I did 175kg for a single at 9 RPE and now I can do it at 5 RPE." This is clearly progress and you can track it without having to consistently do fatiguing 1RM tests. If you perform some sort of fatiguing test after every training block, then you might have to take a deload or elongated intro week following the test. However, tracking RPE gives you a metric of how successful the block was from a strength (or volume performance) perspective and even allows you to gauge progress during the middle of a training block by comparing RPEs at a given load to RPEs at similar loads during previous weeks. Table 8 gives specific examples of how to track progress and predict a 1RM with RPE. The first two columns of Table 8 originally appeared in an article from Volume 2,

IT'S JUST NOT THAT IMPORTANT TO MAINTAIN AN EXACT PROXIMITY TO FAILURE; RATHER, IT'S IMPORTANT TO UNDERSTAND THE INTENT OF RPE, WHICH IS GENERALLY TO EITHER BE FAR FROM FAILURE, A FEW REPS FROM FAILURE, OR AT FAILURE.

Issue 11, and the third row was newly created for this article.

Progression Schemes with RPE.

Another often overlooked aspect of RPE is achieving progressive overload. We are oftentimes fixated on saying we are going to increase 2.5kg each week on a main lift; however, that is just not typically feasible. If you are programming load prescription with RPE like the basic RPE loading example (Table 4), then progressive overload takes care of itself; over time, you should be able to load more on the bar to meet the prescribed RPE. However, if you have a predetermined load and you simply track RPE (Table 8) in a percentage-based program, then you can use your RPE scores from each session or each week to progress load, sets, or reps for the following week. The most basic way to use RPE to progress load is creating an inverse relationship between RPE and progress (i.e. the lower the RPE, the greater the increase in load). Additionally, if you bench 100kg for 3 sets of 10 reps at an average RPE of 9, then you can repeat this until your average RPE is 8, and then add load or add a set. In the previous example, you could continue adding sets until you complete 5 X 10 at an average RPE of 8 and then increase the load on the bar and go back to 3 sets (i.e. 3 X 10 at 102.5kg). There are many other ways to do this, which could be an article in and of itself. The good news is that we already have it in video form. You can watch the detailed presentation of using RPE to achieve progressive overload, and remember that strategies laid out in the video are not mutually exclusive.

To Implement a Flexible Template in the Warm-Up

Using RPE to assess readiness is not as popular as the strategies discussed above, but it still has merit. A foundational principle of using RPE for load prescription is that it can take into account low daily readiness and high fatigue. So, why not use RPE to account for readiness in the warm-up? Specifically, if you are going through a busy time, then it might make sense to im-

Table 9 Using RPE during the warm-up to determine flexible template decisions

RPE	Choice of session-type
> 8	Off day
7-8	Light session
7	Moderate or light session
6	Moderate session
5	Heavy or moderate session
< 5	Heavy session

5 = this individual's normal RPE at 85% of 1RM

plement a flexible training template. In short, you might have three training sessions per week in which you have a heavy training day, a moderate day, and a light day, but the order in which you do them is flexible to meet your readiness levels. You then need to have a metric that you use to decide which training day to do. The most common readiness metric is the perceived recovery status scale, which is essentially a 1-10 Likert scale with 1 being "poorly recovered and expecting declined performance" and 10 being "well-recovered and expecting improved performance." In short, a high rating on this scale suggests you should do the heavy session that day, while a low recovery rating indicates you should do the light session. However, data have shown that pre-training recovery ratings do not always correlate with performance in that day's session; rather, RPE during the warm-up might be a better indicator (14). Thus, if you are using a flexible template, you could simply warm up to a decent load (i.e. 80%-85% of 1RM) and take an RPE. If your normal RPE at an 85% bench press is 6 (4 RIR), and you record an 8 RPE or higher, then you might opt for the light day. If your RPE is 5-7, you might opt for the moderate session, and if your RPE is <5, you might opt for the heavy session. I don't think you have to be that strict with it, and RPE is only going to fluctuate so much, but – in general – it's probably better to decide which workout you are going to do in a flexible template after you start warming up rather than before the warm-up. Table 9 presents a "less-strict" version of using RPE during the warm-up to choose session-type.

Final Thoughts

Before we finish up, we should address the common critique of using RPE, which is that the rating is subjective and may be inaccurate. Some people's ratings may indeed be inaccurate, but how much does that matter? Well, it can matter a lot if someone is attempting to use RPE to autoregulate a heavy double or single. In this case, if someone records a 200kg squat at an 8 RPE while working up to a single at 9 RPE, that would translate to about a 220kg max. However, if the 200kg X 1 was actually a 9 RPE, this calculation may erroneously cause the lifter to attempt 210kg on the next set, and it could be a true max or the lifter could even fail the attempt. While there would be fatigue consequences to overshooting the RPE at such a high intensity, it is highly unlikely that someone experienced is that inaccurate with RPEs during low-rep sets at heavy loads. It's more likely that some personality types don't lend well to programming something like "singles at 9 RPE." Some lifters might simply rationalize a way to max out when heavy singles are programmed using RPE. In this case, as a coach, I would just prescribe a heavy load for a single that I know the lifter can hit at an 8 RPE and give them an option for a second rep to ensure failure doesn't occur. On the other hand, a lifter might be too cautious when working up and end up only working to an 8 RPE; however, that is less consequential than overshooting the RPE.

FURTHER, IF USING RPE DURING MODERATE- TO HIGH-REP SETS, THE POINT ISN'T TO BE PERFECTLY PRECISE. RATHER, THE PURPOSE IS TO SIMPLY BE WITHIN A RANGE (I.E. 5-8) TO ENSURE YOU ARE AN APPROPRIATE PROXIMITY FROM FAILURE.

While RPEs are typically quite accurate during low-rep/high-intensity sets, RPEs can indeed be inaccurate during high-rep sets. In fact, well-trained lifters were on average 5.15 ± 2.92 reps off when asked to verbally call out an intra-set RPE when they believed they were at a 5 RPE (5 RIR), a 7 RPE (3 RIR), and 9 RPE (1 RIR), and then continue to failure during a set at 70%

APPLICATION AND TAKEAWAYS

1. RPE is just one of many tools to implement the concept of autoregulation. In its most basic form, RPE can be utilized to prescribe daily training load, which takes into account the limitations of percentages such as daily readiness and the large between-lifter variation in reps that can be performed at a given percentage of 1RM.
2. Although RPE is most commonly used to prescribe load (i.e. 3 sets of 5 at 7-9RPE) it can also be used to autoregulate volume (RPE Stops), program assistance work, progress weekly load, sets, or reps, and simply be used to track progress over time. The utility of RPE is widespread and not limited to the narrow box we usually put it in.
3. The common critique that RPE is subjective, and thus not perfectly accurate, is correct. However, the point isn't to always be perfectly accurate. Further, using RPE to predict RIR is quite accurate, based upon the existing literature, during low-rep and high-intensity sets, which is when accuracy is most important.

of 1RM on squats in which the average reps performed were 16 ± 4 (11). That is a considerable amount of error; however, that was also overrating RPE, meaning subjects actually had about 10 reps left, which is far less consequential than underrating RPE. Further, if using RPE during moderate- to high-rep sets, the point isn't to be perfectly precise. Rather, the purpose is to simply be within a range (i.e. 5-8) to ensure you are an appropriate proximity from failure. Overall, as previously mentioned, RPE ratings tend to be strikingly accurate in well-trained lifters during low-rep sets (15), and there is no evidence that velocity provides a better gauge of RIR in this case. During high-rep sets intended to be shy of failure, I don't see some degree of inaccuracy to be too much of a problem, especially if you are overrating RPE. Besides, if you consider the large variance of reps performed at moderate intensities mentioned earlier (i.e. 6-28 at 70%), then a percentage program could lead a lifter to a much greater programming error than an RPE rating inaccurately predicting RIR by just a few reps.

If you are unsure if your RPEs are accurate, try rating an RPE after a few reps and then continuing to failure to see how precise you are. I would recommend doing this with a weight ≥80% of 1RM on a compound movement when you perceive you are at about a 7 RPE. If you are a coach and a client is unsure that there RPEs are accurate, you can advise them to also gauge an intra-set RPE before continuing a set to failure. Additionally, as a coach, I would still have a client rate RPE, even if you aren't using it to program or progress load, and have them send you videos of those lifts. Then, you can evaluate, although not perfectly, if you think those RPEs are accurate. Of course, you can always validate RPE with velocity, but we do not all have access to accurate velocity devices. If you do have access to an accurate velocity device, then just about everything written in this article can also be accomplished with velocity.

Over the past year, I've seen some people prefer to simply rate RIR as opposed to rating RPE. If you are at a 5 RPE or higher (1-5 RIR), then that is perfectly fine. Terminology is just terminology; it's the intent and implementation that matters. So, if someone prefers to just use the right side of the scale in Table 3 (i.e. RIR), then that will work just fine, as the terms are interchangeable when the RPE is at least 5. However, in the literature, even the RIR-based scale uses RPE on the low end of the scale, as scores ≤4 RPE quantify effort and are not associated with an RIR. It is difficult to determine a precise RIR when so far from failure. It is also possible that even though RPE and RIR are intended to be interchangeable when close to failure, the term RIR simply resonates more with some. So, whichever terminology someone prefers is fine. They are the same thing in the context of this specific scale.

References

1. Borg G. Perceived exertion as an indicator of somatic stress. Scand j rehabil med. 1970;2:92-8.
2. Borg GA. Psychophysical bases of perceived exertion. Med sci sports exerc. 1982 Jan 1;14(5):377-81.
3. Hackett DA, Johnson NA, Halaki M, Chow CM. A novel scale to assess resistance-exercise effort. Journal of sports sciences. 2012 Sep 1;30(13):1405-13.
4. Tuchscherer M. The Reactive Training Manual: Developing your own custom training program for powerlifting. Reactive Training Systems. 2008;15.
5. Zourdos MC, Klemp A, Dolan C, Quiles JM, Schau KA, Jo E, Helms E, Esgro B, Duncan S, Merino SG, Blanco R. Novel resistance training–specific rating of perceived exertion scale measuring repetitions in reserve. The Journal of Strength & Conditioning Research. 2016 Jan 1;30(1):267-75.
6. Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA, Parker S, Doleshal P, Dodge C. A new approach to monitoring exercise training. The Journal of Strength & Conditioning Research. 2001 Feb 1;15(1):109-15.
7. Ormsbee MJ, Carzoli JP, Klemp A, Allman BR, Zourdos MC, Kim JS, Panton LB. Efficacy of the repetitions in reserve-based rating of perceived exertion for the bench press in experienced and novice benchers. The Journal of Strength & Conditioning Research. 2019 Feb 1;33(2):337-45.
8. Helms ER, Byrnes RK, Cooke DM, Haischer MH, Carzoli JP, Johnson TK, Cross MR, Cronin JB, Storey AG, Zourdos MC. RPE vs. Percentage 1RM loading in periodized programs matched for sets and repetitions. Frontiers in physiology. 2018 Mar 21;9:247.
9. Graham T, Cleather DJ. Autoregulation by" Repetitions in Reserve" Leads to Greater Improvements in Strength Over a 12-Week Training Program Than Fixed Loading. Journal of strength and conditioning research. 2019 Apr.
10. Cooke DM, Haischer MH, Carzoli JP, Bazyler CD, Johnson TK, Varieur R, Zoeller RF, Whitehurst M, Zourdos MC. Body Mass and Femur Length Are Inversely Related to Repetitions Performed in the Back Squat in Well-Trained Lifters. The Journal of Strength & Conditioning Research. 2019 Mar 1;33(3):890-5.
11. Zourdos MC, Goldsmith JA, Helms ER, Trepeck C, Halle JL, Mendez KM, Cooke DM, Haischer MH, Sousa CA, Klemp A, Byrnes RK. Proximity to Failure and Total Repetitions Performed in a Set Influences Accuracy of Intraset Repetitions in Reserve-Based Rating of Perceived Exertion. Journal of strength and conditioning research. 2019 Feb.
12. Pareja-Blanco F, Rodríguez-Rosell D, Sánchez-Medina L, Sanchis-Moysi J, Dorado C, Mora-Custodio R, Yáñez-García JM, Morales-Alamo D, Pérez-Suárez I, Calbet JA, González-Badillo JJ. Effects of velocity loss during resistance training on athletic perfor-

mance, strength gains and muscle adaptations. Scandinavian journal of medicine & science in sports. 2017 Jul;27(7):724-35.

13. Pareja-Blanco F, Rodríguez-Rosell D, Aagaard P, Sánchez-Medina L, Ribas-Serna J, Mora-Custodio R, Otero-Esquina C, Yáñez-García JM, González-Badillo JJ. Time Course of Recovery From Resistance Exercise With Different Set Configurations. Journal of strength and conditioning research. 2018 Jul.
14. Zourdos MC, Dolan C, Quiles JM, Klemp A, Jo E, Loenneke JP, Blanco R, Whitehurst M. Efficacy of daily one-repetition maximum training in well-trained powerlifters and weightlifters: a case series. Nutrición Hospitalaria. 2016;33(2):437-43.
15. Sousa CA. Assessment of Accuracy of Intra-set Rating of Perceived Exertion in the Squat, Bench Press, and Deadlift (Doctoral dissertation, Florida Atlantic University).

Study Reviewed: Effects of Vitamin D3 Supplementation on Serum 25(OH) D Concentration and Strength in Athletes: A Systematic Review and Meta-analysis of Randomized Controlled Trials. Han et al. (2019)

Shedding Some Light on Vitamin D Supplementation: Does It Increase Strength In Athletes?

BY ERIC TREXLER

Vitamin D deficiency is shockingly common in athletes, and low levels are associated with reduced strength. A recent meta-analysis suggested that vitamin D supplementation failed to enhance strength in athletes, but there's more to this paper than meets the eye. Read on to figure out if vitamin D supplementation might be worth considering.

KEY POINTS

1. While much of the vitamin D literature focuses on the general population, the current meta-analysis (1) sought to determine if vitamin D supplementation enhances strength performance in athletes
2. In order to make the results a bit more intuitive and interpretable, I re-crunched the numbers. Overall, there was a small effect of vitamin D supplements (d = 0.20, p = 0.34). If you divide up the results by performance outcome, the effect size for bench press was -0.12 (p = 0.54), and the effect size for isokinetic leg extension was 0.63 (p = 0.01).
3. The largest effects of vitamin D supplementation were observed in the samples who started out with the lowest blood vitamin D levels. While there appears to be a discrepancy between upper body and lower body outcomes, this might be due to the methods used to measure each strength outcome.

It probably shouldn't be too controversial to suggest that, in general, vitamin deficiencies aren't a positive thing. However, vitamin D has a special status in the eyes of most lifters, as researchers have previously suggested that vitamin D supplementation could potentially enhance aerobic performance, strength performance, muscle growth, and recovery from exercise (2). Unfortunately, there's also a bit of uncertainty associated with the management of blood vitamin D levels; there's an active debate about whether the optimal range is above 50 nmol/L or 75 nmol/L, blood levels are meaningfully influenced by latitude and magnitude of sun exposure (which is difficult to practically quantify), and excessively high blood levels are also problematic. Dr. Helms has previously discussed vitamin D supplementation in two previous MASS articles, but this month, there's a new vitamin D meta-analysis (1) to report and interpret. Authors of the current paper (1) specifically evaluated the effects of vitamin D supplementation on strength outcomes in athletes. Results indicated that supplementation significantly increased blood vitamin D levels, but effects on bench press strength (effect size [d] = -0.07, p = 0.72) and isokinetic leg extension strength (d = 2.14, p = 0.12)

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

were not statistically significant, nor was the overall effect on both strength outcomes pooled together (d = 0.75, p = 0.17). Having said that, I think these numbers should be taken with a grain of salt. This article explains why I feel that way and discusses whether or not vitamin D is an advisable supplementation strategy.

Purpose and Hypotheses

Purpose

The purpose of this meta-analysis was "to investigate the effects of vitamin D3 supplementation on skeletal muscle strength in athletes." As an additional outcome, they also analyzed the effects of vitamin D3 supplementation on serum vitamin D levels.

Hypotheses

The authors hypothesized that the meta-analysis would find that vitamin D3 supplementation significantly increases serum vitamin D levels and significantly improves strength performance.

Subjects and Methods

Subjects

As a meta-analysis, this paper pooled the results of multiple studies. A defining characteristic of this meta-analysis was that it only included studies that recruited athletes. The sports represented included taekwondo, soccer, judo, rugby, and football. Strength data were available for a total of 80 athletes, with bench press data for 49 athletes and isokinetic leg extension data for 31 athletes.

Methods

The whole point of a meta-analysis is to search the literature systematically, then mathematically pool the results together to summarize the collective findings. The authors searched the common research databases and only included randomized controlled trials that specifically evaluated strength outcomes in athletes taking oral vitamin D3 supplements. They excluded any potential studies that involved non-athletes, vitamin D2 supplementation, interventions utilizing multivitamins, and studies that included athletes with illnesses or medical conditions that could have potentially altered outcomes of interest.

With meta-analyses, you are working toward calculating a pooled effect size. In order to do that, an effect size (Cohen's d, or some similar form of standardized mean difference) is calculated for each study included. Typically, for this type of literature, you'd calculate the effect size based on the change in the placebo group (from pre-testing to post-testing), the change in the vitamin D group, and then some form of standard deviation for each group– either the standard deviation of the pre-test or post-test value,

Table 1 Baseline and follow-up serum 25(OH)D concentrations

Reference	Latitude	Time	Vitamin D3 daily dosage IU	Baseline (ng/mL)	N=149	1 week (ng/mL)	4 weeks (ng/mL)	6 weeks (ng/mL)	8 weeks (ng/mL)	12 weeks (ng/mL)
Jung 2018	33.3° N	Jan-Feb	5000 0	10.9 ± 0.5 12.4 ± 0.8	20 15		38.4 ± 1.5 13.1 ± 1.0			
Fairbairn 2018	45-46.5° S	Mar-May	3570 0	37.2 ± 7.6 38 ± 6.8	28 29			44.4 ± 7.2 34 ± 6.8		45.6 ± 7.6 32 ± 8.4
Close 2013b	53° N	Nov-Jan	5000 0	11.6 ± 10.0 21.2 ± 11.6	5 5				41.3 ± 10.0 29.6 ± 9.6	
Close 2013a	53° N	Jan-Apr	5714 2857 0	20.4 ± 10.4 21.2 ± 10.4 20.8 ± 10.8	6 10 9			39.3 ± 5.6 31.7 ± 5.6 14.8 ± 7.2		36.5 ± 9.6 34.1 ± 4.0 16.4 ± 8.8
Wyon 2016	52.3° N	Feb	18750 0	13.2 ± 3.8 16.3 ± 2.7	11 11	16.8 ± 3.2 16.3 ± 2.6				

Data Calculated from Han Q, Li X, Tan Q, Shao J, and Yi M. 2019 (1)
Data are mean ± SD unless stated otherwise
Measurements are in ng/mL

or the standard deviation of the change from pre- to post-testing.

For the current meta-analysis, they took a very different approach. Effect sizes were calculated using only the pre-test value in the vitamin D group, the post-test value in the vitamin D group, and the standard deviations at each time point. This is quite atypical, and totally ignores a key, defining feature of these studies, which is that they included a placebo group. The strength of the placebo-controlled design is that we can directly evaluate the effect of the treatment above and beyond the effect of the placebo; to ignore this in the effect size calculation is to adopt a less informative interpretation of each study's individual results. Off the top of my head, I can only specifically recall seeing one other recent meta-analysis use this approach (3), and it was subsequently retracted, accompanied by a message stating that, "The authors have retracted this article because after publication it was brought to their attention that the statistical approach is not appropriate." I believe they're referring to the manner in which the effect sizes were calculated, but unfortunately no details were provided.

Findings

As one would expect, oral vitamin D supplementation significantly increased blood vitamin D levels; the effect size was d = 3.0 for all studies together, and d = 1.18 after removing one study with a fairly high dropout rate. I have used informal language up to this point, so I should clarify that "blood vitamin D levels" refers more specifically to serum levels of 25-hydroxyvitamin D, or 25(OH)D. The liver converts vitamin D3 into 25(OH)D, which is then converted to 1,25-hydroxyvitamin D. While 1,25-hydroxyvitamin D is technically the active form of vitamin D, researchers typically measure 25(OH)D because it has a longer half-life in the blood, and its circulating levels are about 1,000 times higher (4). Table 1 shows the baseline and post-test values for blood vitamin D levels, along with some key study characteristics related to vitamin D levels, such as latitude and time of

Reference	Vitamin D daily dosage IU	N = 149	1RM BP (kg)			Leg extension (N·m)		
			Pre	Post	Change	Pre	Post	Change
Jung 2018	5000 0	20 15				323.6 ± 32.6 329.9 ± 32.5	350.4 ± 33.5 339.2 ± 33.7	26.8 9.3
Fairbairn 2018	3570 0	28 29	126 ± 17 122 ± 17	122 ± 15 123 ± 16	-4 1			
Close 2013b	5000 0	5 5	82 ± 14 82 ± 14	88.5 ± 14 84.5 ± 14	6.5 2.5			
Close 2013a	5714 2857 0	6 10 9	91 ± 22 90 ± 13 79 ± 17	90 ± 20 92 ± 15 79 ± 18	-1 2 0			
Wyon 2016	18750 0	11 11				232 ± 37.4 239 ± 65.9	265 ± 45.6 239 ± 63.7	33 0

Table 2 Strength outcome measures

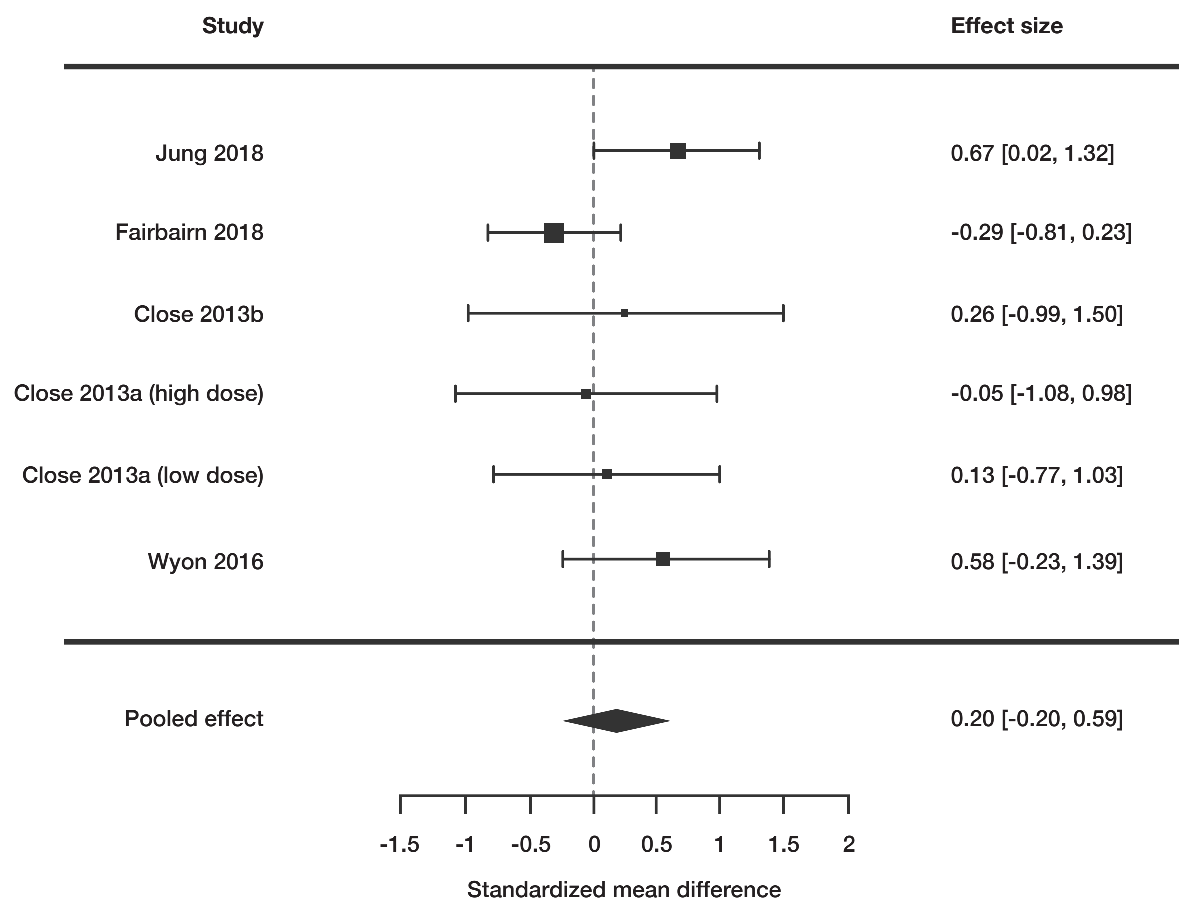
Data Calculated from Han Q, Li X, Tan Q, Shao J, and Yi M. 2019 (1). Note: We corrected some values from the original text.

year. To assist with interpretation, keep in mind that 1 ng/mL is equivalent to 2.5 nmol/L, and some scientists suggest that the ideal blood vitamin D range is 20-40 ng/mL (50-100 nmol/L), whereas others suggest that it's 30-50 ng/mL (75-125 nmol/L).

For strength outcomes, the authors found that supplementation did not significantly alter performance, with an overall effect size of d = 0.75. When they looked at each performance outcome (bench press or isokinetic leg extension) in isolation, the bench press effect size was -0.07, and the leg extension effect size was 2.14 (but still not statistically significant, with a p-value of 0.12). The results from each individual study, along with the vitamin D dosages used, are presented in Table 2.

If you read my previous review of a fairly recent creatine meta-analysis, you know that I tend to be really picky about how meta-analyses are done. At first glance, it might seem like I'm splitting hairs and making a huge deal out of minor differences. However, the current meta-analysis presents us with an awesome example of why meta-analytic methods matter, big time. There was another vitamin D meta-analysis by Tomlinson et al in 2015 (5), which included some of the same data. Both included a 2013 study by Close et al (6), which evaluated two different vitamin D doses, looking at bench press as an outcome variable. For the same exact data, Tomlinson et al calculated an effect size of d = 0.75 for the low-dose treatment, whereas the current meta-analysis calculated an effect size of d = 0.14. To contextualize that gap of around 0.6, it's worth noting that caffeine typically improves strength and power outcomes with an effect size of around 0.2-0.3 (7), and a 2003 meta-analysis found that the effect of creatine on 1RM outcomes was around 0.32 (8). Furthermore, the current study computed an effect size of 3.55 for Jung et al (9), while the true value should be less than 1 if you use virtually any commonly accepted method of effect size calculation. So, while I

Figure 1 Re-calculated forest plot



admittedly enjoy exploring these details more than anyone should, it's safe to say that these things matter.

As I noted in the *methods* section, the authors of the current meta-analysis took a very unconventional statistical approach. It should also be noted that they got standard deviation and standard error mixed up for Jung et al (9) and omitted the higher-velocity leg extension data from Jung et al (9) and Wyon et al (10) without explanation or justification. Taken together, we have multiple justifiable reasons to disregard the values calculated in the original paper.

With these considerations in mind, I went ahead and re-crunched the numbers in a way that I find to be more informative. I calculated Hedges' g as my effect size metric, but its interpretation is extremely similar to Cohen's d, so I'm just going to use "d" as my general effect size symbol throughout this article. To calculate effect sizes, I compared the change (pre to post) in the supplement group to the change (pre to post) in the placebo group. I used the baseline standard deviation for effect size calculations, and I assumed a within-study correlation of 0.8 when aggregating multiple effect sizes from a single sample. With this approach, the overall pooled effect size ends up being 0.20 (p = 0.34), and the forest plot is presented in Figure 1.

The authors of the current meta-analysis also split the data set to independently look at bench press results and leg extension results. If we do that with the re-calculated values, the effect size for bench press outcomes is -0.12 (p = 0.54), and the effect size for leg extension outcomes is 0.63 (p = 0.01).

Interpretation

Vitamin D supplementation has become somewhat popular among lifters, likely because low vitamin D levels tend to be quite common. For example, even among healthy athletes, one recent meta-analysis reported that 56% of subjects sampled had inadequate vitamin D levels, which was operationally defined as blood 25(OH)D levels below 32 ng/mL (80 nmol/L) (11). Other studies have shown up to 57%, and even 62%, of athlete samples to have deficient or insufficient vitamin D levels (12). That's pretty troubling, as low vitamin D levels have been linked to depression, cognitive decline, poor bone health, and decreased neuromuscular function (2). Specifically, vitamin D levels tend to be lower during the winter months, and in individuals who have minimal direct exposure to sunlight, live at high latitudes, or frequently wear sunblock with a high sun protection factor. It's frequently said that vitamin D levels are typically lower in individuals with darker skin pigmentation, but Dr. Helms made me aware of some research indicating that it might not be that simple (13). In short, individuals with darker skin pigmentation may have lower levels of *total* blood 25(OH)D concentrations, despite having similar bone density and similar levels of *bioavailable* 25(OH)D. This is important, because not all of the 25(OH)D in our blood is bioavailable, and it's the bioavailable 25(OH)D that's really driving the positive effects of vitamin D.

As noted by Dahlquist et al (2), there are very plausible reasons to believe that correcting vitamin D deficiency or insufficiency would have a positive impact on performance. For example, multiple studies have found correlations between blood vitamin D levels and aerobic fitness (VO2max), and one study found that vitamin D supplementation increased VO2max (14), possibly by influencing oxygen's binding affinity with hemoglobin. There is also observational and experimental evidence linking vitamin D to muscle force production, which may be related to an increase in the size and number of type II muscle fibers or enhanced calcium sensitivity of the sarcoplasmic reticulum. Much of this research has been conducted in samples of older adults; such studies typically observe notable deficits in neuromuscular function as a result of vitamin D deficiency, which is robustly restored following vitamin D supplementation (15). Other observational and experimental studies have linked vitamin D to higher testosterone levels, reduced post-exercise inflammation, and more rapid recovery from intense exercise (2). In summary, there is reason to believe that vitamin D may positively impact a variety of exercise performance outcomes due to its effects on sarcoplasmic reticula, testosterone, hypertrophy, recovery, and even oxygen delivery.

When I first saw the results of the current meta-analysis, I was pretty skeptical. The effect sizes just seemed way too large and inconsistent, and further digging verified that some additional number-crunching was warranted. After re-running the analysis, this literature is much more in line with what I would have expected. The overall effect size is a very realistic d = 0.20, and the overall analysis was not statistically significant. However, if we look a little bit closer at the results, a couple of interesting patterns appear.

The first pattern is pretty intuitive: The studies with the three largest effect sizes were the studies reporting the lowest baseline vitamin D levels in the supplement group. In each of these studies, the baseline value for the supplement group was under 14 ng/mL, while the other three studies had baseline values above 20 ng/mL. By far, the least impressive results were reported by Fairbairn et al (16), with an effect size of -0.29. Their supplement group had the highest baseline vitamin D levels by far, with an initial value of over 37 ng/mL. To contextualize that, no other group receiving supplements in this meta-analysis had a baseline value over 21.2 ng/mL. In addition, while there is some ongoing debate on exactly what the "ideal" range of blood vitamin D levels is, 37 ng/mL is considered sufficient under every set of recommendations that I've come across.

The second pattern is pretty intriguing: The studies within this meta-analysis seem to indicate that vitamin D was beneficial for lower-body exercise, but not upper-body exercise. This was also reported in a recent meta-analysis by Zhang et al (17), but a 2015 meta-analysis by Tomlinson et al (5) reported nearly identical effects for a variety of upper-body and lower-body exercises following vitamin D supplementation. Of course, for the current set of data, it's possible that the explanation is simply that the studies looking at lower-body exercises coincidentally happened to, on average, report lower baseline vitamin D levels. It's also possible that the observed difference between upper-body and lower-body results may relate to physiological explanations. For example, Zhang et al (17) speculated that lower-body musculature could be more responsive to vitamin D supplementation due to greater vitamin D receptor density in those particular muscle groups, or due to physiological differences related to the fact that the lower-body musculature is much more heavily involved in activities of daily living. Despite these possibilities, I have a hunch that this apparent difference could be explained by research methods.

In the current study, upper-body strength was exclusively defined as bench press strength, whereas lower-body strength was defined as isokinetic leg extension. Isokinetic leg extension doesn't really mimic the way we train in the gym or compete on the platform, but it's awesome for research purposes. You can control the speed of contraction, the range of motion, and every joint angle imaginable, for a measurement that can be reliably replicated at multiple visits. In addition, you're taking a sensitive, granular torque measurement, down to the exact Newton meter. With bench press, things are different. Setups can vary from day to day. Participants know the load on the bar, have some degree of an emotional connection to it, and deep down, they probably want their post-test value to be higher than their pre-test value, despite not knowing what treatment group they're in. It's much more difficult to standardize the movement and to ensure that you're getting a perfectly equivalent, maximal effort at all visits. In addition, how much can we realistically expect an athlete's (note: typically well-

VITAMIN D SUPPLEMENTATION CAN HAVE A SMALL BUT POSITIVE EFFECT ON STRENGTH OUTCOMES, PARTICULARLY IF SUPPLEMENTATION IS BRINGING YOU FROM INSUFFICIENT VITAMIN D LEVELS TO SUFFICIENT VITAMIN D STATUS.

trained, but not hyper-focused on bench pressing) bench press to increase from vitamin D supplementation over the span of a fairly short-term study? Many labs lack fractional plates, so for a number of subjects, testing probably approximated a categorical variable: they could either add another 1.25kg plate to each side, or maybe two, or maybe none.

Perhaps the strongest evidence supporting this theory is presented by Tomlinson et al (5). They looked at a variety of upper body and lower body outcomes following vitamin D supplementation, using a broader selection of studies than the current meta-analysis. The pooled effect sizes for upper body results and lower body results were virtually identical. However, both the upper body and lower body categories included ex-

IF YOU SUSPECT THAT YOUR VITAMIN D IS LOW, THE BEST APPROACH IS TO GET YOUR BLOOD TESTED AND WORK WITH A QUALIFIED HEALTHCARE PROFESSIONAL TO GET A SUPPLEMENTATION PLAN TOGETHER.

ercise tests that used gym machines, free weights, and dynamometers (handgrip or isokinetic). For the upper body outcomes (eight total), the two lowest effect sizes were from tests using gym machines or free weights, with the six highest effect sizes coming from dynamometry. For the lower body outcomes (eight total), the three lowest effect sizes were from tests using gym machines or free weights, with the five highest effect sizes coming from dynamometry. Further, a 2013 study (18) sought to determine if blood vitamin D levels correlated with upper body or lower body strength measurements in a sample of 419 men and women aged 20-76 years. Notably, all measurements were taken via dynamometry. When controlling for age and sex, blood vitamin D level was significantly associated with both arm and leg strength. If anything, the relationship was more consistent for upper-body strength than lower-body strength after controlling for additional covariates.

The studies included in this meta-analysis suggest that vitamin D supplementation can have a small but positive effect on strength outcomes, particularly if supplementation is bringing you from deficient or insufficient vitamin D levels to sufficient vitamin D status. While it's true that effects appear to be more pronounced in lower body exercise than upper body, I'm inclined to believe that this is an artifact of the measurement techniques used rather than a "real" physiological difference. As I noted previously, there's a bit of a debate regarding what "sufficient" really is; some people suggest that blood levels of 25(OH)D should be above 20 ng/mL (50 nmol/L), while other suggest it should be above 30 ng/mL (75 nmol/L). However, as with most things in physiology, more is not always better. Vitamin D enhances calcium absorption from the gut, in addition to increasing mineralization and bone resorption (that is, the process by which bone tissue is broken down and its minerals are released into the blood) by stimulating bone cells to produce receptor activator nuclear factor-kB ligand. As a result, chronically high vitamin D levels could potentially lead to excessive blood calcium levels, which could increase the risk of kidney stones or cardiovascular issues

APPLICATION AND TAKEAWAYS

Maintaining sufficient blood vitamin D levels definitely seems like a good idea, both for health and performance. The studies within this meta-analysis suggest that vitamin D supplementation can have a small but meaningful effect on strength performance, but only if supplementation is bringing your suboptimal baseline vitamin D levels up into the optimal range. There is some evidence suggesting that effects are more pronounced in lower-body strength tasks than upper-body tasks, but I suspect this is more of a methods issue than a physiology issue. Finally, it's important to remember that more vitamin D isn't always better. If you suspect that your vitamin D is low, the best approach is to get your blood tested and work with a qualified healthcare professional to get a supplementation plan together.

related to vascular calcification (2). To be fair, serum 25(OH)D concentrations below 140 don't seem to be associated with high blood calcium levels, and acutely observable adverse effects typically aren't reported until blood 25(OH)D levels get up around 200 nmol/L, which would probably require a daily vitamin D dose of around 40,000 IU per day (2). Nonetheless, the take-home point remains the same: You don't want your vitamin D levels to be too low or too high. Finally, individuals with a relatively high degree of skin pigmentation might want to rely on metrics other than total blood 25(OH)D levels to determine if they should consider supplementation. In my opinion, the best approach to managing your vitamin D levels with confidence is to get some valid blood testing done, and put a supplementation plan together with your doctor or otherwise qualified healthcare practitioner.

Next Steps

For lifters, it looks like there are two key questions to be answered in the near future. When it comes to blood vitamin D levels, there still isn't a consensus about how much is enough. So, it'd be great to more conclusively identify the optimal range of blood vitamin D levels in which neuromuscular performance is optimized. In addition, I'd like to see some follow-up work investigating the apparently differential responses between upper-body and lower-body musculature. Ideally, we'd see studies that involve both upper-body and lower-body measurements within the same subjects, using both free weights and dynamometry, to figure out if the observed difference in the current meta-analysis is attributable to physiology or measurement precision. For the free weight measurements, it'd be great if researchers blind the loads being used and utilize fractional plates, which would serve to minimize confounding effects from psychological factors and enhance the precision of 1RM estimates.

References

1. Han Q, Li X, Tan Q, Shao J, Yi M. Effects of vitamin D3 supplementation on serum 25(OH)D concentration and strength in athletes: a systematic review and meta-analysis of randomized controlled trials. J Int Soc Sports Nutr. 2019 Nov 26;16(1):55.
2. Dahlquist DT, Dieter BP, Koehle MS. Plausible ergogenic effects of vitamin D on athletic performance and recovery. J Int Soc Sports Nutr. 2015;12:33.
3. Siddique U, Rahman S, Frazer AK, Howatson G, Kidgell DJ. RETRACTED ARTICLE: Determining the Sites of Neural Adaptations to Resistance Training: A Systematic Review and Meta-Analysis. Sports Med. 2019 Nov;49(11):1809.
4. Lukaszuk JM, Luebbers PE. 25(OH)D status: Effect of D3 supplement. Obes Sci Pract. 2017 Mar;3(1):99–105.
5. Tomlinson PB, Joseph C, Angioi M. Effects of vitamin D supplementation on upper and lower body muscle strength levels in healthy individuals. A systematic review with meta-analysis. J Sci Med Sport. 2015 Sep;18(5):575–80.
6. Close GL, Leckey J, Patterson M, Bradley W, Owens DJ, Fraser WD, et al. The effects of vitamin D(3) supplementation on serum total 25[OH]D concentration and physical performance: a randomised dose-response study. Br J Sports Med. 2013 Jul;47(11):692–6.
7. Grgic J, Grgic I, Pickering C, Schoenfeld BJ, Bishop DJ, Pedisic Z. Wake up and smell the coffee: caffeine supplementation and exercise performance-an umbrella review of 21 published meta-analyses. Br J Sports Med. 2019, ePub ahead of print.
8. Branch JD. Effect of creatine supplementation on body composition and performance: a meta-analysis. Int J Sport Nutr Exerc Metab. 2003 Jun;13(2):198–226.
9. Jung HC, Seo MW, Lee S, Jung SW, Song JK. Correcting Vitamin D Insufficiency Improves Some But Not All Aspects of Physical Performance During Winter Training in Taekwondo Athletes. Int J Sport Nutr Exerc Metab. 2018 Nov 1;28(6):635–43.
10. Wyon MA, Wolman R, Nevill AM, Cloak R, Metsios GS, Gould D, et al. Acute Effects of Vitamin D3 Supplementation on Muscle Strength in Judoka Athletes: A Randomized Placebo-Controlled, Double-Blind Trial. Clin J Sport Med. 2016 Jul;26(4):279–84.
11. Farrokhyar F, Tabasinejad R, Dao D, Peterson D, Ayeni OR, Hadioonzadeh R, et al. Prevalence of vitamin D inadequacy in athletes: a systematic-review and meta-analysis. Sports Med. 2015 Mar;45(3):365–78.
12. Ogan D, Pritchett K. Vitamin D and the Athlete: Risks, Recommendations, and Benefits. Nutrients. 2013 May 28;5(6):1856–68.
13. Owens DJ, Allison R, Close GL. Vitamin D and the Athlete: Current Perspectives and New Challenges. Sports Med. 2018;48(Suppl 1):3–16.
14. Jastrzębski Z. Effect of vitamin D supplementation on the level of physical fitness and blood parameters of rowers during the 8-week high intensity training. Facicula Educ Fiz Şi Sport.

2014;2:57–67.

15. Hamilton B. Vitamin D and Human Skeletal Muscle. Scand J Med Sci Sports. 2010 Apr;20(2):182–90.

16. Fairbairn KA, Ceelen IJM, Skeaff CM, Cameron CM, Perry TL. Vitamin D3 Supplementation Does Not Improve Sprint Performance in Professional Rugby Players: A Randomized, Placebo-Controlled, Double-Blind Intervention Study. Int J Sport Nutr Exerc Metab. 2018 Jan 1;28(1):1–9.

17. Zhang L, Quan M, Cao Z-B. Effect of vitamin D supplementation on upper and lower limb muscle strength and muscle power in athletes: A meta-analysis. PloS One. 2019;14(4):e0215826.

18. Grimaldi AS, Parker BA, Capizzi JA, Clarkson PM, Pescatello LS, White MC, et al. 25(OH) vitamin D is associated with greater muscle strength in healthy men and women. Med Sci Sports Exerc. 2013 Jan;45(1):157–62.

Study Reviewed: Delayed Myonuclear Addition, Myofiber Hypertrophy and Increases in Strength with High-Frequency Low-Load Blood Flow Restricted Training to Volitional Failure. Bjørnsen et al. (2018)

The First Clear Evidence of Delayed Hypertrophic Supercompensation

BY GREG NUCKOLS

The idea of delayed hypertrophic supercompensation – the idea that your muscles can keep growing for several days after you complete a grueling block of training – is very contentious. A recent study provides us with the first evidence that it's possible. However, there's quite a bit more to the story.

KEY POINTS

1. This study had untrained subjects complete two blocks of high-frequency blood flow restriction training, with 10 days between blocks.
2. Strength and muscle fiber cross-sectional area both appeared to follow a pattern of delayed supercompensation. Muscle fiber CSA decreased at first, and then increased until at least 10 days after the last session was completed. Maximal knee extension strength increased until at least 20 days after the last session was completed.
3. Interestingly, muscle fiber CSA and whole muscle size followed different patterns of adaptation. Whole muscle size didn't decrease initially, and it didn't keep increasing after the training was completed.

I recently reviewed a study from Bjørnsen and colleagues with some interesting findings: Just two weeks of low-load training with blood flow restriction (BFR) caused really robust hypertrophy of type I fibers, providing the clearest evidence we have for fiber type-specific hypertrophy (2). The same group is back with another eye-catching study (1), potentially demonstrating delayed hypertrophic supercompensation for the first time. Delayed supercompensation is the idea that beneficial adaptations can keep occurring after a period of training is completed. It's most often discussed in the context of overreaching: You train beyond your normal capacities for a time, but after several days of rest, you rapidly accrue beneficial adaptations. Most people think about delayed supercompensation from a performance perspective, and several theories of tapering and peaking are built around this idea. However, delayed hypertrophic supercompensation is much more controversial; the traditional view is that muscles stop growing when you stop training.

In this study, untrained subjects completed two five-day blocks of high-frequency, low-load training with blood flow restriction. The researchers measured maximal knee extension strength, muscle fiber cross-sectional area (CSA), and whole-muscle CSAs and thicknesses. While measures of whole muscle size increased quickly and potentially decreased a bit after the cessation of training (probably due to a reduction in

Listen to Greg Nuckols, Eric Trexler, Eric Helms and Mike Zourdos discuss this study in the audio roundtable.

Go to playlist in Soundcloud

Table 1 Subject characteristics				
Age (years)	**Body mass (kg)**	**Height (cm)**	**1RM knee extension**	**Sex**
24 ± 2	78 ± 12	179 ± 8	65 ± 14	9 men, 4 women

swelling), muscle fiber CSAs and knee extension strength kept increasing long after the second block of training finished. The continued increase in fiber CSA and discordance between changes in fiber size and whole muscle size are very interesting and certainly worth a closer look.

Purpose and Research Questions

Purpose

The purpose of this study was to "investigate the effects of two blocks with high frequency blood flow restricted resistance exercise, separated by 10 days of rest, on fiber and whole muscle areas, myonuclear and satellite cell numbers and muscle strength, and the time courses of those changes."

Hypotheses

In previous research (3), hypertrophy due to high-frequency BFR training plateaued after seven days of training. It was hypothesized that the 10-day rest period between training blocks would reset the subjects' responsiveness to the anabolic stimuli so that they'd experience hypertrophy, increases in satellite cell number, and myonuclear accretion during both blocks of training.

Subjects and Methods

Subjects

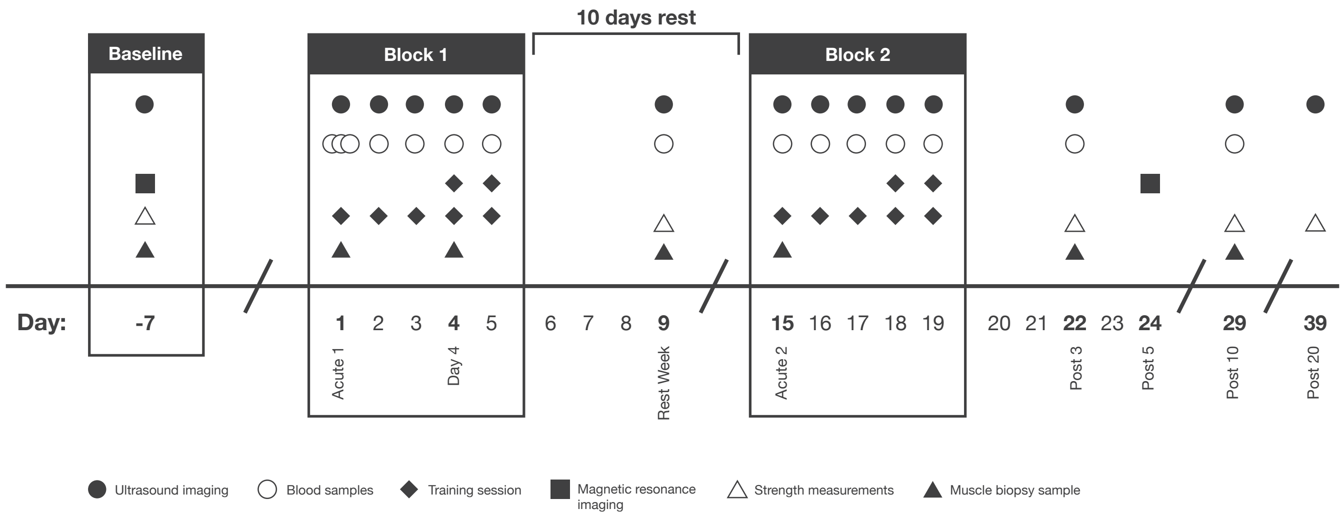
16 recreationally active adults with no resistance training experience participated in this study. Three subjects dropped out over the course of the study, so 13 subjects were included in the final analyses. Further details about the subjects can be seen in Table 1.

Study Overview

The whole study took place over 46 days for each participant. One week before training began, the subjects underwent baseline testing, including assessments of quad muscle size and strength, a blood draw, and a muscle biopsy.

The training itself consisted of two blocks of high-frequency, low-load knee extensions with BFR. Each block lasted for five days. During the first three days of each block, the subjects trained once per day, and they trained twice per day during the last two days of each block (accomplishing seven workouts in five

Figure 1 Overview of the study protocol



days). For all sessions, the subjects performed four sets of blood flow restricted unilateral knee extensions to failure with each leg, with 20% of 1RM and 30 seconds between sets. All four sets were completed on the right leg first, followed by four sets with the left leg. The pressure cuff used to achieve blood flow restriction (inflated to 90mmHg for women and 100mmHg for men) was left on between sets.

The subjects had a 10-day break between the two blocks of training, and follow-up measures were assessed at 3, 5, 10, and 20 days following the second training block. The authors assessed strength using 1RM knee extensions; they assessed hypertrophy with ultrasound scans, muscle biopsies, and MRIs; and they performed blood draws to measure blood markers of muscle damage (creatine kinase and myoglobin).

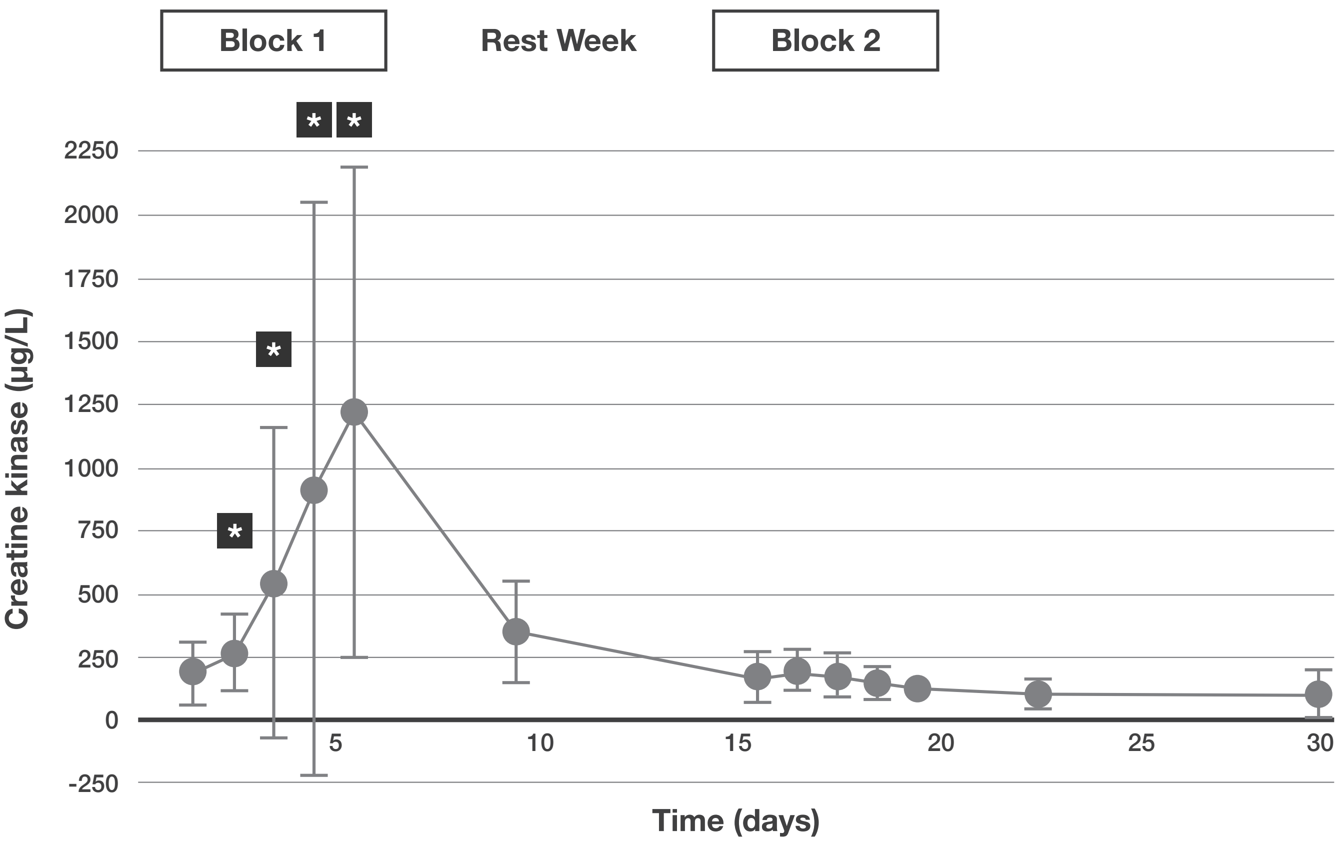
For a schematic of this study, see Figure 1.

Findings

Training loads didn't change over the course of the study, but rep performance increased. The subjects completed 80 ± 14 reps per session during the first block, and 89 ± 13 reps per session during the second block.

Markers of muscle damage were significantly elevated during the first block of training, went back to baseline during the rest week, and then did not increase significantly above baseline during the second block of training. Soreness (assessed via a visual analog scale) peaked during the third day of the first block, whereas creatine kinase and myoglobin peaked on the last day of the first block of training.

Figure 2 Creatine kinase levels from the first day of training until 10 days post-training

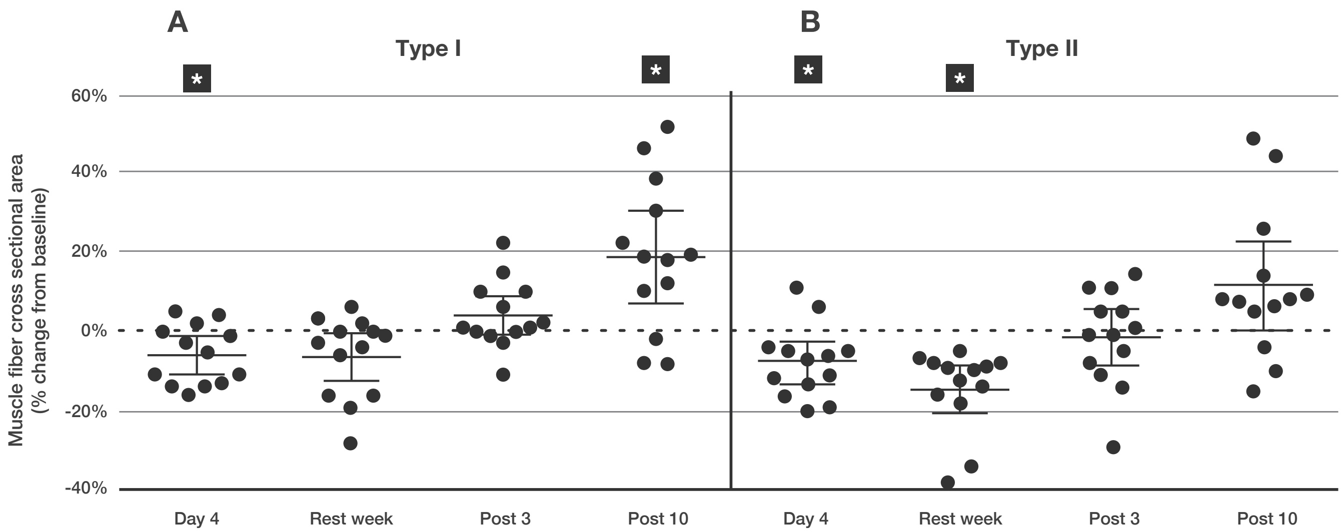


** = Significant increase in creatine kinase levels from baseline*

Muscle fiber CSA significantly *decreased* at first. The decrease was larger in type II fibers (-15% during the rest period) than type I fibers (-6% during the first block of training). After the initial decrease in fiber CSA, fiber size increased throughout the rest of the study. It was back around baseline for both fiber types three days after the last training session and was elevated above baseline 10 days post-training (+19% for type I, and +11% for type II). The difference from baseline at 10 days post-training was significant for type I fibers (p=0.01), but not quite significant for type II fibers (p=0.09).

Hypertrophy estimates from ultrasound scans tell a very different story. Rectus femoris CSA and vastus lateralis thickness increased significantly above baseline by the end of the first block of training (+6.8% and +5.6%, respectively), trended back toward baseline measures during the 10-day rest period (down to 1.5% and 3.4% above baseline), increased significantly again

Figure 3 Changes in fiber CSAs from baseline



* = Significant change in fiber CSA from baseline

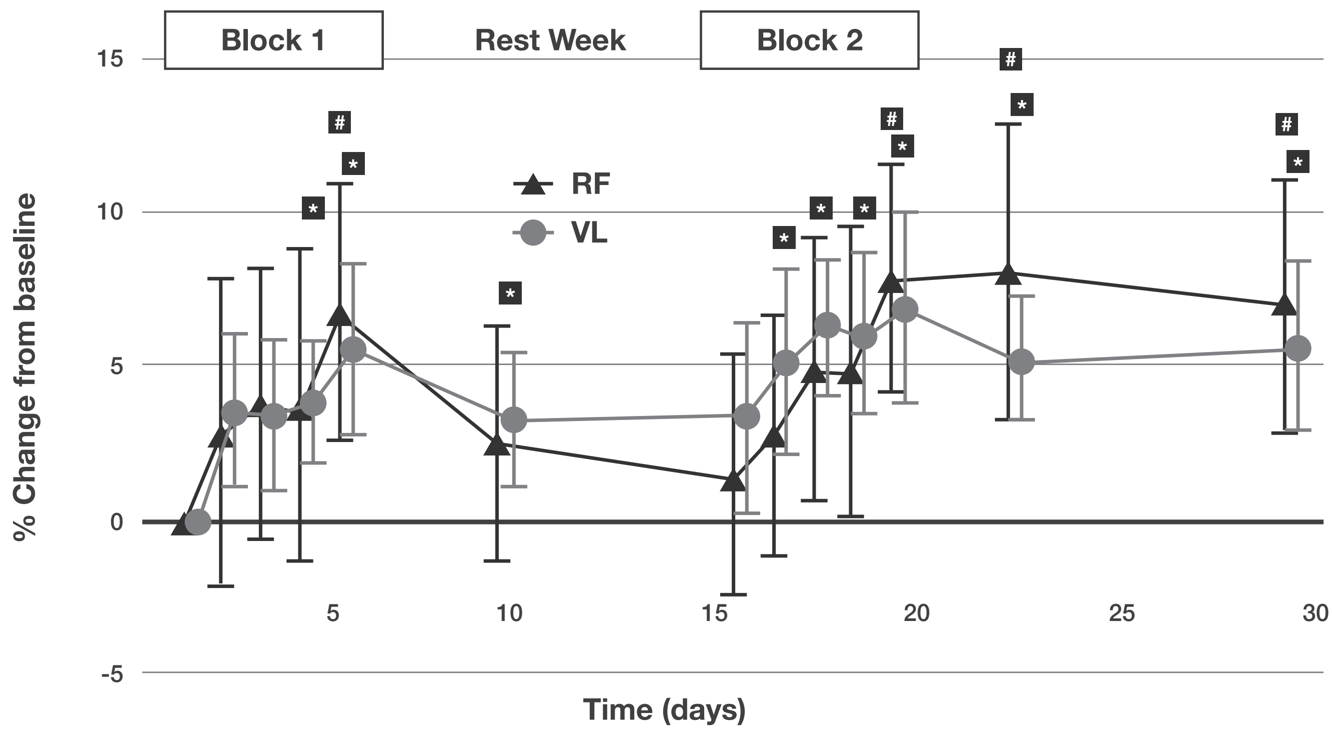
by the end of the second training block (up to 7.9% and 6.9% above baseline), and stayed elevated above baseline (decreasing non-significantly to 7.0% and 5.7% above baseline) during the 10 days following the last training session. MRI scans were only taken at baseline and five days post-training, but rectus femoris CSA, vastus lateralis CSA, and total quadriceps CSA all significantly increased as well. However, the relative increases tended to be smaller than those seen with either the ultrasound scans or the biopsies (+6.2% for rectus femoris CSA, +2.4% for vastus lateralis CSA, and +1.2% for quadriceps CSA).

Much like fiber CSA, 1RM knee extension strength initially decreased slightly, though significantly (-4%), from baseline to the rest period. Strength did not significantly differ from baseline at 3 and 10 days post-training, but was significantly elevated 20 days post-training (+6%). However, the total swing in mean strength was very modest, from 65kg at baseline, to 63kg during the rest period, to 69kg 20 days post-training.

Satellite cells per muscle fiber increased quickly in both fiber types (by ~70% in type I fibers and ~50% in type II fibers by day four of the first block of training). That increase more or less leveled off for type I fibers (peaking at an increase of 96% three days post-training), but satellite cells per type II fiber increased progressively (peaking at an increase of 144% 10 days post-training).

In both fiber types, myonuclei per fiber didn't increase between baseline and the rest week. However, myonuclei per

Figure 4 Relative increases in CSA of rectus femoris and muscle thickness of vastus lateralis



Relative increases in cross sectional area of rectus femoris and muscle thickness of vastus lateralis from the first day of training until the last measurements 10 days post-training.
** = Significant change in VL compared to baseline ($p < 0.05$); # = significant change in RF compared to baseline ($p < 0.05$)*
Data is reported as pooled means between the left and right leg and expressed as mean ± SD

fiber then increased following the second training block, peaking at 10 days post-training for both fiber types (+30% for type I fibers, and +31% for type II fibers). Interestingly, myonuclear domain tended to decrease in both fiber types.

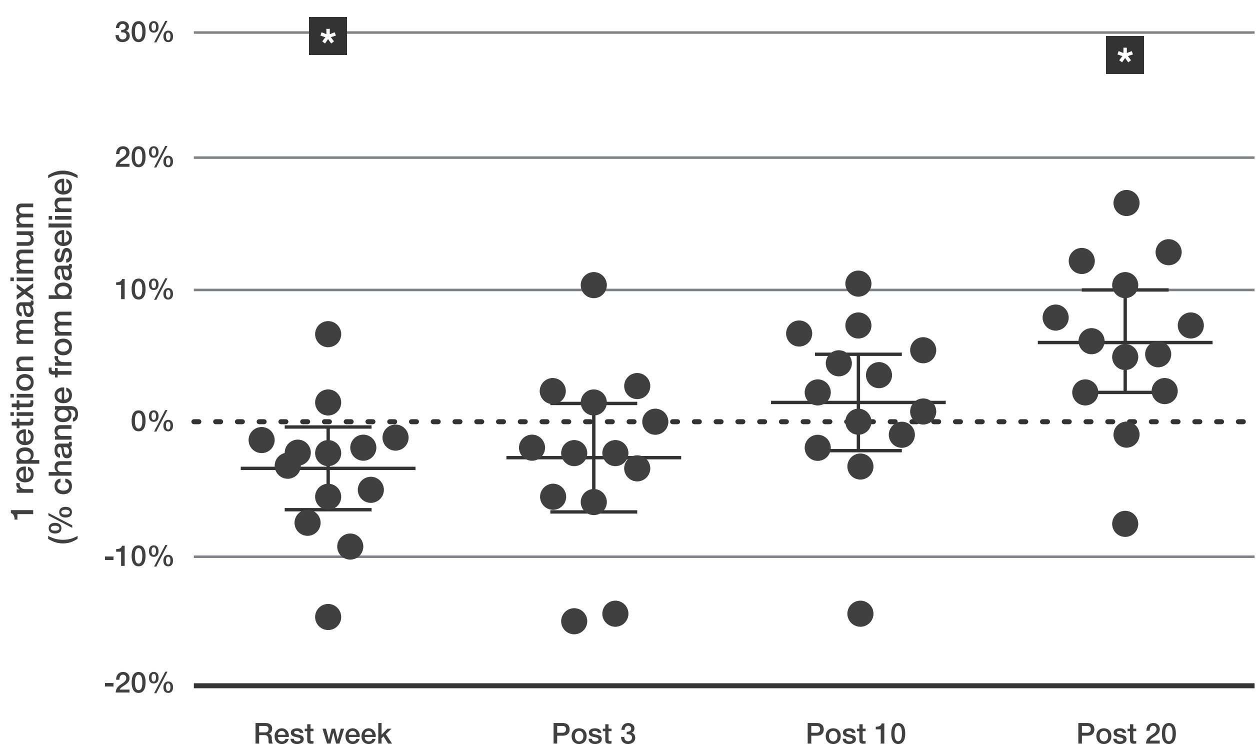
Since this is a research review for strength athletes and coaches, I won't belabor the cellular signaling markers, except to say that the pattern of gene expression looked to be most in favor of anabolism 10 days post-training.

Interpretation

There are a few interesting things about these findings.

To start with, as a word of caution, one of the subjects had to withdraw from the study during the first block of training with symptoms that looked like the possible onset of rhabdomyolysis (pain, extreme weakness, and huge elevations in creatine kinase). More generally, the subjects had large increases in creatine kinase, myoglobin, and soreness during the first block. This stands in opposition

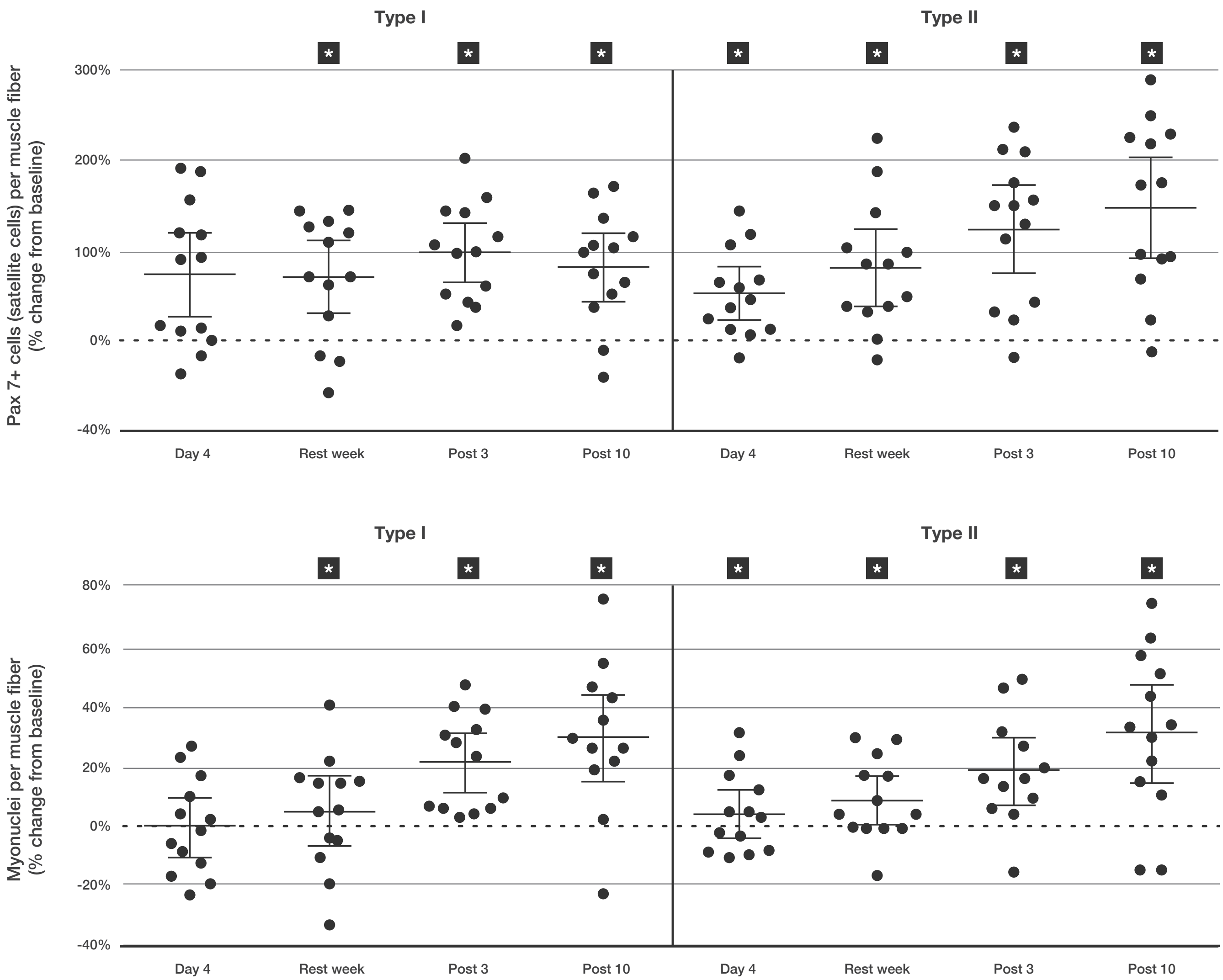
Figure 5 Increase in 1RM of knee extension



* = significant change in 1RM knee extension from baseline

to the popular idea that low-load training with blood flow restriction causes minimal muscle damage (4). However, it seems like the devil is in the details. *Most* early blood flow restriction studies had people perform sets of 30, 15, 15, and 15 reps with a load equal to 30% of 1RM. Even with blood flow restriction, 30 reps at 30% of 1RM will be well shy of failure, and you may not hit failure until the last set with the 30/15/15/15 protocol (if you hit failure at all). The majority of the research using this rep scheme finds little to no muscle damage with low-load blood flow restricted training, even with untrained subjects. However, it seems that low-load blood flow restriction training *can* cause substantial muscle damage if all sets are taken to failure (5, 6). On a related note, this study also demonstrates the outcomes of the repeated bout effect (7). During the first block of training, the subjects experienced increases in soreness and substantial elevations in myoglobin and creatine kinase. During the second block of training (after just seven prior exposures to that style of training), there were no elevations in any of the markers of muscle damage assessed.

Figure 6 Changes in satellite cells and myonuclei per fiber from baseline



** = Significant change from baseline*

It's difficult to parse the actual training outcomes of this study. A simple takeaway is that this study beautifully demonstrated training specificity. Strength endurance (total reps completed during each session) increased by a bit over 10% from the first block to the second block of training. However, maximal strength didn't significantly increase between baseline testing and the 1RM test that occurred three days after the end of the second block of training. The training loads were very light, so they were effective at increasing strength endurance, but didn't do much for maximal strength.

The maximal strength findings are a

bit confusing. Maximal knee extension strength didn't significantly increase over baseline values until *20 days* after the last training session. On one hand, you could interpret that as a delayed supercompensation effect. On the other hand, we could just be seeing the effect of learning the test. The subjects were familiarized at the start of the study, but seeing as the subjects were untrained, they still hadn't done many maximal knee extensions in their lives by the time the second block of training wrapped up. Simply having a few more sessions to learn the test could explain the modest increase in maximal strength (~6%), especially in these untrained subjects. I think that's especially likely, since strength wasn't significantly elevated at 10 days post-training. The training protocol in this study was certainly challenging, but I just can't imagine that it was grueling enough that fatigue wouldn't have dissipated after 10 days of rest.

Unlike maximal strength, muscle fiber size *did* seem to undergo delayed supercompensation. Between 3 and 10 days post-training, the CSAs of both fiber types increased by more than 10%. As noted, the anabolic signaling milieu (i.e. decreased p21 abundance and increase myogenin and cyclin D2 abundance) seemed to be the most favorable for hypertrophy at 10 days post-training, so that may contribute. As we learned in a previous issue of MASS, training, detraining, and retraining also causes epigenetic changes (8) that seem to be favorable for hypertrophy. That may contribute as well. However, it's still very interesting, and I'm not sure that those two potential explanations can fully explain these results. As far as I'm aware, this is the first study showing this type of delayed hypertrophic supercompensation following resistance training. I'd love to see these results replicated.

IT SEEMS THAT LOW-LOAD BLOOD FLOW RESTRICTION TRAINING CAN CAUSE SUBSTANTIAL MUSCLE DAMAGE IF ALL SETS ARE TAKEN TO FAILURE.

It's interesting to note that whole-muscle hypertrophy and fiber hypertrophy displayed very different patterns. Early on, whole muscle size (thickness or CSA) increased, while fiber CSA decreased. During the actual training phases, the increases in whole muscle size were likely at least partially due to local swelling (edema). However, whole muscle size tended to still be elevated above baseline four days into the rest period between training blocks (which should have been enough time for edema to dissipate), and

> UNLIKE MAXIMAL STRENGTH, MUSCLE FIBER SIZE DID SEEM TO UNDERGO DELAYED SUPERCOMPENSATION. BETWEEN 3 AND 10 DAYS POST-TRAINING, THE CSAS OF BOTH FIBER TYPES INCREASED BY MORE THAN 10%.

it tended to decrease slightly between the end of the second training block and 10 days post-training. Because of that, I doubt the delayed supercompensation of fiber size would matter much to a physique athlete. If your muscle fibers are growing a bit, but the whole muscle isn't changing in size, I doubt that would really affect your appearance of muscularity.

The fact that the different hypertrophy assessments came to different conclusions is intriguing. As mentioned, the time course of gains was completely different; however, the magnitude of changes was different as well. By 10 days post-training, average fiber CSA had increased by ~15%, whereas the change in whole muscle size was closer to 6-7%. This is similar to a recent study by Haun et al (9), where different hypertrophy measures again painted substantially different pictures. At this point, I'm not sure what we can do with that information, but I'd like to see more research investigating the disconnect between changes in fiber size and changes in whole muscle size. One possibility is just that the fibers that are biopsied are not representative of the fibers in the muscle as a whole (possibly due to regional hypertrophy). Muscle swelling should affect whole-muscle size more than fiber size as well. However, I'd bet that there's more to the picture.

As always, it's important to note the variability in individual responses. Type I fiber size increased by ~50% in one subject, while decreasing by ~5% in two subjects. The same is true of type II fibers. One individual saw an increase slightly exceeding 40%, while another individual saw a decrease in excess of 10%. As for strength, one individual got about 8% weaker, while another individual got about 15% stronger. In this study, as with any study, don't blindly assume that everyone gets results that are tightly clustered around the mean.

The authors of this study noted that the satellite cell response in this study was larger than the satellite cell response typically seen in untrained subjects doing conventional, heavier resistance training. However, the increase wasn't as large as the increase previously seen in another low-load blood flow restriction study (3). The authors speculate that differences in training stress could explain the difference. The subjects in this study trained to true failure, whereas the authors of the prior study noted that they didn't push their subjects quite as hard for their first few training sessions. The authors of the present study proposed that the muted satellite cell response and the initial decreases in both fiber size and strength may have been due to simply pushing their subjects past the point they could truly recover from during the first block of training.

Finally, it's interesting to note that we may be seeing preferential type I fiber growth again in this study, as the increase in type I fiber size (19%) tended to be a bit larger than the increase in type II fiber size (11%). A previous paper from this same research group involving low-load blood flow restriction training in powerlifters provided the first strong evidence (in my opinion) for fiber type-specific hypertrophy (2). However, I'm less sold that we're seeing fiber type-specific hypertrophy in this study. Fiber CSA of both fiber types increased by about the same amount between the 10-day rest period and 10 days post-training. The difference was that, during the initial training block, type II fibers *atrophied* more. So, maybe you could make a case for fiber type-specific *atrophy*, but I'm less sold on the premise of preferential type I growth in this study.

I DOUBT THE DELAYED SUPERCOMPENSATION OF FIBER SIZE WOULD MATTER MUCH TO A PHYSIQUE ATHLETE. IF YOUR MUSCLE FIBERS ARE GROWING A BIT, BUT THE WHOLE MUSCLE ISN'T CHANGING IN SIZE, I DOUBT THAT WOULD REALLY AFFECT YOUR APPEARANCE OF MUSCULARITY.

I'm sure some people will read this review and get fired up to try an overreaching block. After all, we now have some direct evidence for delayed supercompensation, which is the idea that overreaching blocks are built on. However, I'd be wary of that interpretation. For starters, the total strength gains in this study weren't anything to write home about, and whole muscle size didn't show delayed supercompensation. More importantly, we can't know if the delayed supercompensation (likely due

APPLICATION AND TAKEAWAYS

I'm wary of extracting any concrete takeaways from this study, since we don't know whether the results would generalize to the type of training most MASS readers would be doing. However, we do now know that delayed hypertrophic supercompensation is at least possible, which is something that was debatable (and that I was personally doubtful of) before this study.

to overreaching) allowed the subjects to make larger gains, in total, compared to non-overreaching training. For example, fiber size initially decreased, and then increased dramatically between the 10-day rest period and 10 days post-training, including a pretty large increase between 3 days post-training and 10 days post-training. If you just pay attention to the actual period of delayed supercompensation, the results are impressive indeed: Fiber CSA increased by around 1% per day, which is an insane rate of progress. However, it's very possible that these subjects would have grown more, in total, had they not overreached and experienced a bit of atrophy during and following the first part of the study. Without clear evidence of better results with overreaching, it seems like a high risk (increasing your odds of overtraining, and potentially increasing your injury risk) strategy with an unclear (likely small, at best) payoff. Finally, it's clear that delayed supercompensation isn't fully understood. Maybe it happens in trained subjects after moderate-to-heavy loading, or maybe it's a phenomenon that could only occur in new lifters training at very low intensities. There's just a lot that we don't yet know about delayed supercompensation.

As a final note, some of the results of this study could be used to argue for the efficacy of occasional blocks of high-frequency, low-load blood flow restriction training for people interested in maximizing hypertrophy. As we learned previously (2), low-load, high-rep front squats with blood flow restriction can cause impressive quad growth in powerlifters after just two one-week blocks. The authors of the present study note that low-load training with BFR leads to a larger satellite cell response than is typically seen with traditional, heavier training, and the decrease in myonuclear domain size hints at the possibility that the subjects in this study were primed for future growth. Myonuclear domains tend to increase initially with training (up to a relatively fixed point, called the myonuclear domain limit), as fiber CSA initially increases faster than new myonuclei can be added (10). Hypertrophy is generally easier when lifters' fibers are below their myonuclear domain limit, so a decrease in myonuclear domain size suggests that the subjects in this study were primed for additional rapid growth. However, the same caveat applies: We don't know if we'd see the same myonuclear domain outcomes in well-trained lifters.

Next Steps

I'd love to see these results replicated in trained lifters, specifically the delayed supercompensation of fiber size. I'd also love to see changes in muscle protein subfractions during and after a block of training. Maybe contractile proteins initially increase during training, and then structural and metabolic proteins increase after training cessation to support the increase in contractile proteins (i.e. myofibrillar hypertrophy followed by sarcoplasmic hypertrophy), or maybe structural and metabolic proteins increase initially to meet the initial demands of training, followed by contractile proteins (sarcoplasmic hypertrophy followed by myofibrillar hypertrophy). Finally, I'd like to see a comparison of "concentrated" training like this (two blocks of seven session in five days) that likely caused acute overreaching compared to more dispersed training (maybe those same 14 sessions spread over 28 days). I think strength gains would be greater with the dispersed training, but it's possible that concentrating training could be beneficial for hypertrophy, as a way to overload the muscles enough to force adaptation.

References

1. Bjørnsen T, Wernbom M, Løvstad AT, Paulsen G, D'Souza RF, Cameron-Smith D, Flesche A, Hisdal J, Berntsen S, Raastad T. Delayed myonuclear addition, myofiber hypertrophy and increases in strength with high-frequency low-load blood flow restricted training to volitional failure. J Appl Physiol (1985). 2018 Dec 13.
2. Bjørnsen T, Wernbom M, Kirketeig A, Paulsen G, Samnøy L, Bækken L, Cameron-Smith D, Berntsen S, Raastad T. Type 1 Muscle Fiber Hypertrophy after Blood Flow-restricted Training in Powerlifters. Med Sci Sports Exerc. 2018 Sep 4.
3. Nielsen JL, Aagaard P, Bech RD, Nygaard T, Hvid LG, Wernbom M, Suetta C, Frandsen U. Proliferation of myogenic stem cells in human skeletal muscle in response to low-load resistance training with blood flow restriction. J Physiol. 2012 Sep 1;590(17):4351-61.
4. Loenneke JP, Thiebaud RS, Abe T. Does blood flow restriction result in skeletal muscle damage? A critical review of available evidence. Scand J Med Sci Sports. 2014 Dec;24(6):e415-422.
5. Umbel JD, Hoffman RL, Dearth DJ, Chleboun GS, Manini TM, Clark BC. Delayed-onset muscle soreness induced by low-load blood flow-restricted exercise. Eur J Appl Physiol. 2009 Dec;107(6):687-95.
6. Cumming KT, Paulsen G, Wernbom M, Ugelstad I, Raastad T. Acute response and subcellular movement of HSP27, αB-crystallin and HSP70 in human skeletal muscle after blood-flow-restricted low-load resistance exercise. Acta Physiol (Oxf). 2014 Aug;211(4):634-46.
7. Hyldahl RD, Chen TC, Nosaka K. Mechanisms and Mediators of the Skeletal Muscle Repeated Bout Effect. Exerc Sport Sci Rev. 2017 Jan;45(1):24-33.
8. Seaborne RA, Strauss J, Cocks M, Shepherd S, O'Brien TD, van Someren KA, Bell PG, Murgatroyd C, Morton JP, Stewart CE, Sharples AP. Human Skeletal Muscle Possesses an Epigenetic Memory of Hypertrophy. Scientific Reports. vol. 8, Article number: 1898 (2018)
9. Haun CT, Vann CG, Mobley CB, Roberson PA, Osburn SC, Holmes HM, Mumford PM, Romero MA, Young KC, Moon JR, Gladden LB, Arnold RD, Israetel MA, Kirby AN, Roberts MD. Effects of Graded Whey Supplementation During Extreme-Volume Resistance Training. Front Nutr. 2018 Sep 11;5:84.
10. Bruusgaard JC, Johansen IB, Egner IM, Rana ZA, Gundersen K. Myonuclei acquired by overload exercise precede hypertrophy and are not lost on detraining. Proc Natl Acad Sci U S A. 2010 Aug 24;107(34):15111-6.

VIDEO: All About Plus Sets

BY MICHAEL C. ZOURDOS

Everybody seems to program a set or two per week for as many reps as possible (AMRAP) or also known as a plus set. These sets have quite a bit of utility, but they also have some drawbacks and are oftentimes overused. This video covers when to use plus sets and provides strategies to individualize their usage.

Click to watch Dr. Zourdos's video.

References

1. Load Progression Part 1. Volume 3 Issue 2.
2. Load Progression Part 2. Volume 3 Issue 3.
3. Assistance Work in Periodization and Loading Options. Volume 2 Issue 2.
4. RPE and RIR: The Complete Guide. Volume 3 Issue 9.

VIDEO: Sticking Points – What Do We Know?

B Y E R I C H E L M S

It is extremely common for powerlifters to focus their training on the "sticking point" in a given lift through various methods. But many unanswered questions exist: Why do sticking points occur? Should we even be focusing on them? If so, which strategies have merit? Eric answers these questions in this video.

Click to watch Dr. Helms's video.

MASS Videos

1. Explaining the Length Tension Relationship

References

1. Kompf J, Arandjelović O. Understanding and overcoming the sticking point in resistance exercise. Sports Medicine. 2016 Jun 1;46(6):751-62.
2. Kompf J, Arandjelović O. The sticking point in the bench press, the squat, and the deadlift: Similarities and differences, and their significance for research and practice. Sports Medicine. 2017 Apr 1;47(4):631-40.
3. Vigotsky AD, Contreras B, Beardsley C. Biomechanical implications of skeletal muscle hypertrophy and atrophy: a musculoskeletal model. PeerJ. 2015 Nov 30;3:e1462.
4. Peltonen H, Walker S, Hackney AC, Avela J, Häkkinen K. Increased rate of force development during periodized maximum strength and power training is highly individual. European journal of applied physiology. 2018 May 1;118(5):1033-42.

Thanks for reading the 2020 "Best Of" issue of MASS.

Subscribe to MASS now to get a new issue just like this one every month.

Graphics by Katherine Whitfield, and layout design by Lyndsey Nuckols.