MASS

MONTHLY APPLICATIONS IN STRENGTH SPORT

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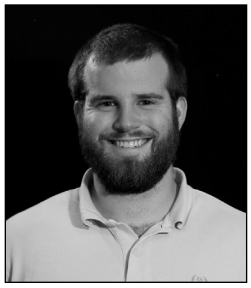
The Reviewers

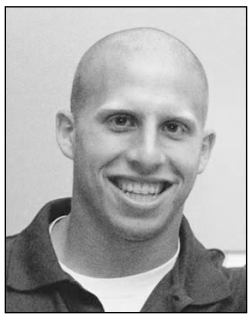
Eric Helms

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 is currently enrolled in the exercise science M.A. program at 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.



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Michael (Mike) C. Zourdos, Ph.D, CSCS, is an associate professor in exercise science at Florida Atlantic University (FAU) in Boca Raton, FL., USA, with a specialization 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. As an associate professor at FAU, Mike is the director of the FAU Muscle Physiology Research Laboratory. 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.

Letter from the Reviewers

Welcome to the 2018-2019 "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 25 issues – that's about 225 articles and videos, 2,000 pages of content, 150 audio roundtable episodes, 500 illustrative graphics, and 50 hours of video. We offer CEUs for two top organizations: NSCA and NASM. As of April 2019, we have more than 2,850 active subscribers. (Not a subscriber yet? Join here.)

And we're just getting started.

What you'll find in these pages is a glimpse at some of our favorite content from the second 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 blood-flow restriction training, failure training, energy availability during weight loss, the impact of hormonal contraceptives on strength gains, power training, and role of training in appetite and weight control, and the science of muscle memory.

In our unique video content, Mike examines how to troubleshoot training programs and make evidence-based adjustments went life gets in the way of training. Eric tackles the topic of sustainable motivation for sport and fitness, showing you what you should (and shouldn't focus) on if you want to stay in your sport long-term.

Each issue will tackle new questions 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, Greg, and Mike

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Study Reviewed: Type I Muscle Fiber Hypertrophy after Blood Flow-Restricted Training in Powerlifters. Bjørnsen et al. (2018)

Blood Flow Restriction Training Causes Type I Fiber Hypertrophy in Powerlifters

BY GREG NUCKOLS

A recent study found that just two weeks of low-load blood flow restriction training caused substantial quad growth in high-level powerlifters. If you're aiming to maximize growth, should you bust out some knee wraps and get your pump on? Well ... maybe.

KEY POINTS

1. In a sample of high-level powerlifters, 6.5 weeks of "normal" training failed to cause significant lower body hypertrophy, while a 6.5 week cycle with two concentrated one-week blocks of low-load blood flow restriction training caused significant quad growth.
2. In the group doing blood flow restriction training, type I ("slow-twitch") muscle fibers increased in size by roughly 12%, while type II ("fast-twitch") muscle fibers didn't grow, providing us with clear evidence of fiber type-specific hypertrophy.
3. Strength gains didn't significantly differ between groups.

Low-load blood flow restriction training involves using a knee wrap (or some other device that functions as a tourniquet) to cut off venous blood flow out of a muscle, while maintaining arterial blood flow into the muscle. The theory behind blood flow restriction training is that venous occlusion will enhance metabolite build-up in the exercising muscle, thus (hopefully) enhancing training adaptations. A couple of years ago, I wrote an article arguing that powerlifters should use low-load blood flow restriction training for a lot of their accessory work. The research at the time indicated that adding low-load blood flow restriction training to a "normal," heavier training program enhanced strength gains without making sessions much harder to recover from, but it didn't seem to cause additional hypertrophy.

Well, a recent study (1) on high-level powerlifters had the exact opposite findings. Two groups of lifters trained for 6.5 weeks, including two one-week blocks of front squats. One group did "normal," heavier front squat training, while the other group did only low-load blood flow restriction training during those front squat blocks. The group doing low-load blood flow restriction training experienced significant quad hypertrophy, including preferential type I fiber growth, while the group doing traditional, heavier training failed to grow. However, strength gains were similar (and negligible) in both groups. The unique approach of having concentrated, non-consecutive blocks of blood flow restriction training may explain why the results of this study differ from prior research.

Listen to Greg, Eric, and Mike discuss this study and topic in the audio roundtable.

Go to playlist in Soundcloud

Table 1 Descriptive characteristics of the participants

	Blood flow restriction group	Conventional group
Age (years)	24 (3)	26 (8)
Height (cm)	176 (5)	177 (9)
Weight (kg)	89 (14)	102 (18)
Powerlifting experience (years)	4 (2)	6 (4)
Muscle strength		
1RM in front squat (kg)	141 (25)	151 (26)
Personal record in squat (kg)	186.7 (42)	207 (40)
Personal record in deadlift (kg)	227 (44)	244 (36)
Personal record in bench press (kg)	135 (28)	154 (32)

The values are presented as mean ± standard deviation (SD). No statistically significant differences were seen between the two groups at baseline.

Purpose and Research Questions

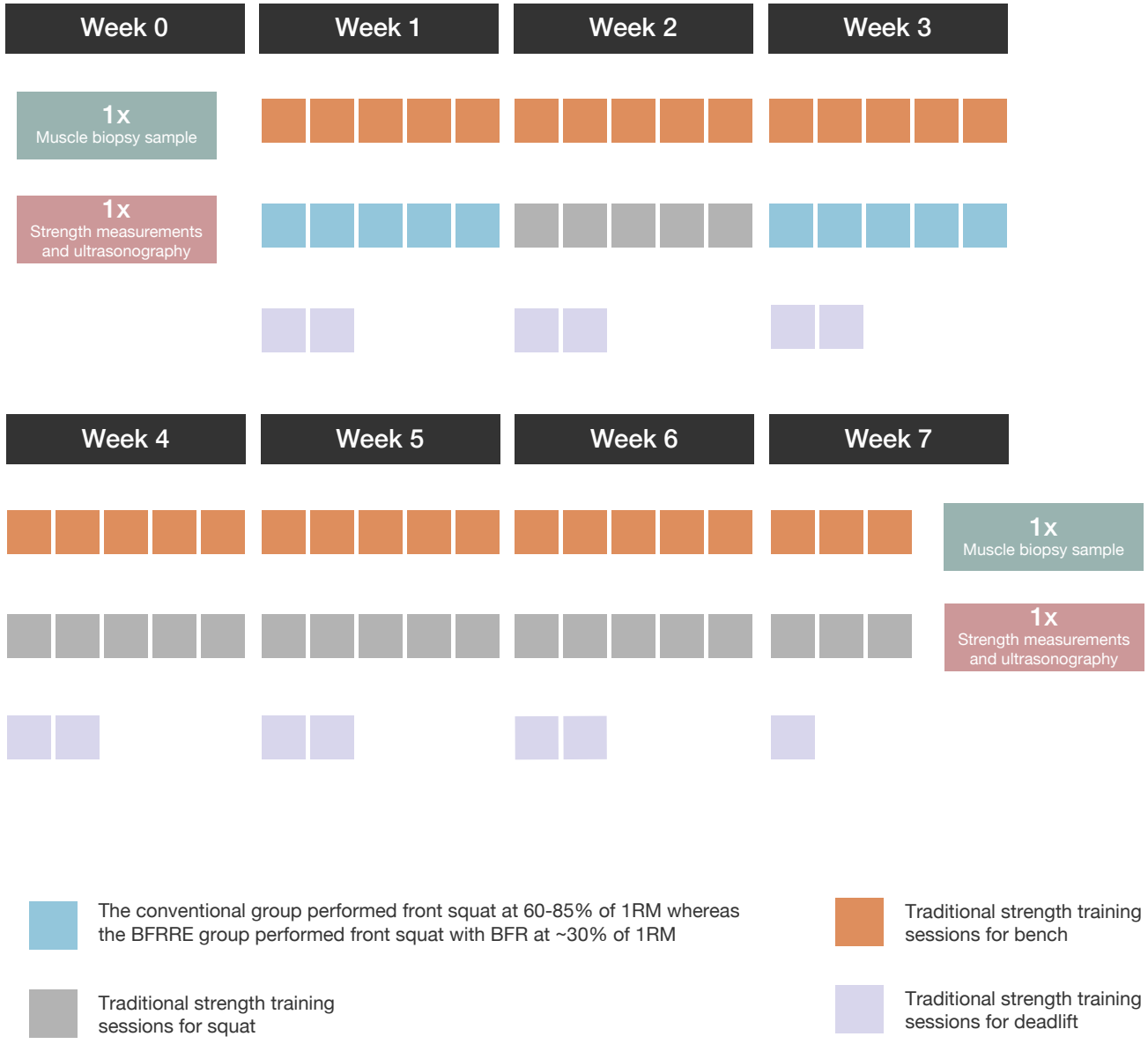
Purpose

The purpose of this study was to examine the effects of two one-week microcycles of low-load front squats with blood flow restriction, compared to traditional, heavier front squats.

Research Questions

1. Would low-load front squats with blood flow restriction lead to more quad hypertrophy than heavier front squats without blood flow restriction in well-trained powerlifters?
2. Would low-load front squats with blood flow restriction lead to larger strength increases than heavier front squats without blood flow restriction in well-trained powerlifters?
3. Would low-load front squats with blood flow restriction lead to larger changes in molecular mechanisms associated with hypertrophy (myonuclei, satellite cells, RNA expression, etc.) than heavier front squats without blood flow restriction?

Figure 1 Schematic Illustration of the Study Design



Hypotheses

The authors hypothesized that the low-load front squats with blood flow restriction would lead to more hypertrophy and larger strength gains and that changes in strength and muscle size would be related to the molecular mechanisms associated with hypertrophy.

Subjects and Methods

Subjects

The subjects were 19 Norwegian powerlifters – 16 men and 3 women – who were at least strong enough to qualify for the national championship in Nor-

way's IPF affiliate. The average Wilks score was 369, and six of the participants (three in each group) regularly participated in international competitions for the Norwegian team (16). Two participants dropped out (one quit the study, and one didn't show up to post testing), and there were measurement issues for a couple of other participants, so either 16 or 17 subjects were included in all analyses. Details about the participants can be seen in Table 1.

Experimental design

Details of the training program can be seen in Figure 1. Briefly, the participants completed a 6.5-week training block with some squat variation and some bench variation five days per week, and some deadlift variation twice per week. During weeks 1 and 3, the subjects in both groups performed front squats for all five squatting sessions. The subjects in one group (n=9) performed low-load front squats with blood flow restriction, and the subjects in the other group (n=8) performed heavier front squats without blood flow restriction.

The group doing front squats with blood flow restriction used approximately 24% of 1RM for week 1, and approximately 31% of 1RM for week 3. They performed four sets with 30 seconds between sets in each low-load BFR session, with the first and last sets taken to voluntary failure, and rep targets of 15 and 12 reps for sets 2 and 3. The blood flow restriction wraps remained on between sets (i.e. they didn't release the pressure and re-wrap between sets).

The group doing heavier front squats performed 6-7 sets of 1-6 reps with 60-85% of 1RM. Details of each session weren't provided, but the training programs were designed by the national team coaches and were part of the lifters' annual periodized plan. Overall, this group performed more sets of front squats in each session, but it doesn't seem that they were taking any sets to failure.

For the group training with blood flow restriction, venous occlusion was accomplished using elastic knee wraps. The lifters were first trained so that they could apply the correct amount of pressure (~120 mmHg). The researchers would put a lightly inflated pressure cuff around the lifters' thighs, and the lifters would apply the knee wraps; the pressure reading on the cuff would tell the lifters and researchers about the actual pressure applied. The lifters practiced applying the wraps until they could reliably achieve a pressure of approximately 120 mmHg. During weeks 1 and 3, the powerlifting coaches randomly checked the lifters' wrapping jobs to ensure that they were still applying their wraps with the correct amount of pressure. At 120 mmHg of pressure, venous blood flow (blood flow out of the limb) should be almost entirely occluded, while arterial blood flow should only be partially occluded (so blood is still allowed into the limb).

Measures

Measurements were taken 2-3 days before the start of the training program and 2-3 days after the end of the training program in both groups.

Strength was assessed via a 1RM front squat and maximum isokinetic torque at 60º/sec.

Hypertrophy was assessed three different ways. Mean fiber area of type I and type II muscle fibers was assessed via biopsy, muscle thickness of all quad muscles was assessed via ultrasound, and cross-sectional area (CSA) of the vastus lateralis and rectus femoris was also assessed using ultrasound.

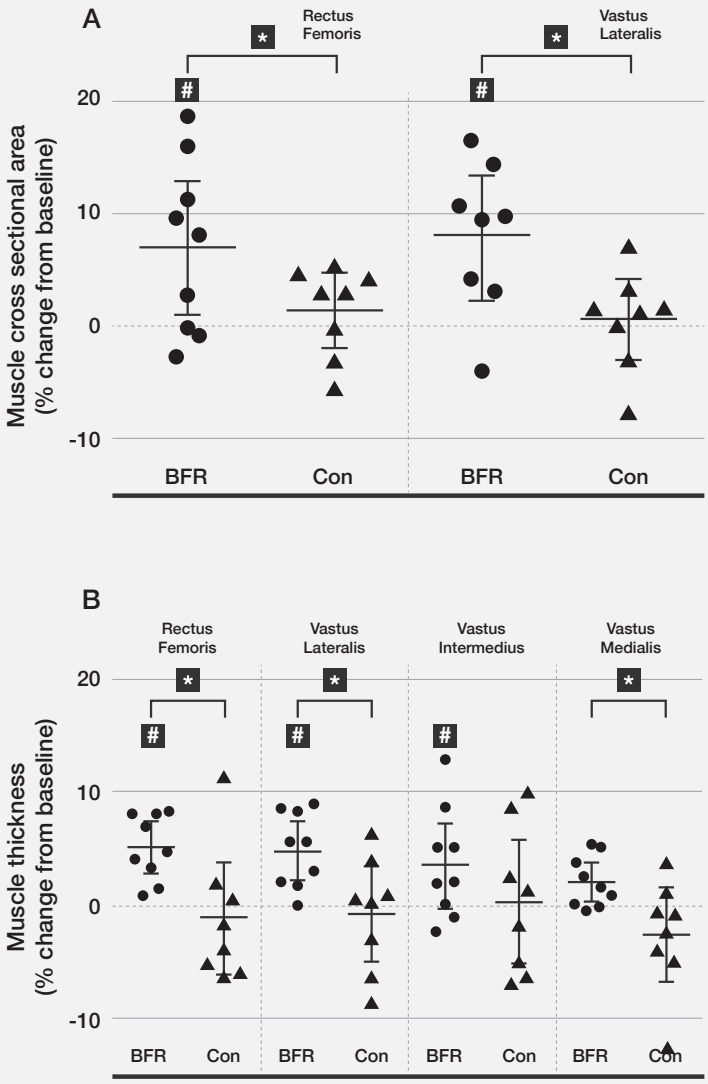
Muscle capillarization, myonuclear number, and satellite cell content were also assessed from the biopsy samples. The biopsies were also used to test for cellular markers associated with hypertrophy signaling and ribosome biogenesis.

Finally, vastus lateralis EMG was assessed in a sub-sample of six participants. These participants performed 2 sets of 3 front squats at 80% of 1RM, and 4 sets of front squats with 30% of 1RM with blood flow restriction (using the same protocol as was used in the rest of the study).

Findings

Front squat volume load didn't differ between groups in week 1, but the blood flow restriction group had a significantly greater volume load in week 3. However, I'm not sure how much that actually matters, since volume load tends to be higher with low-load training anyways, and I don't think volume load is a particularly useful metric in the first place.

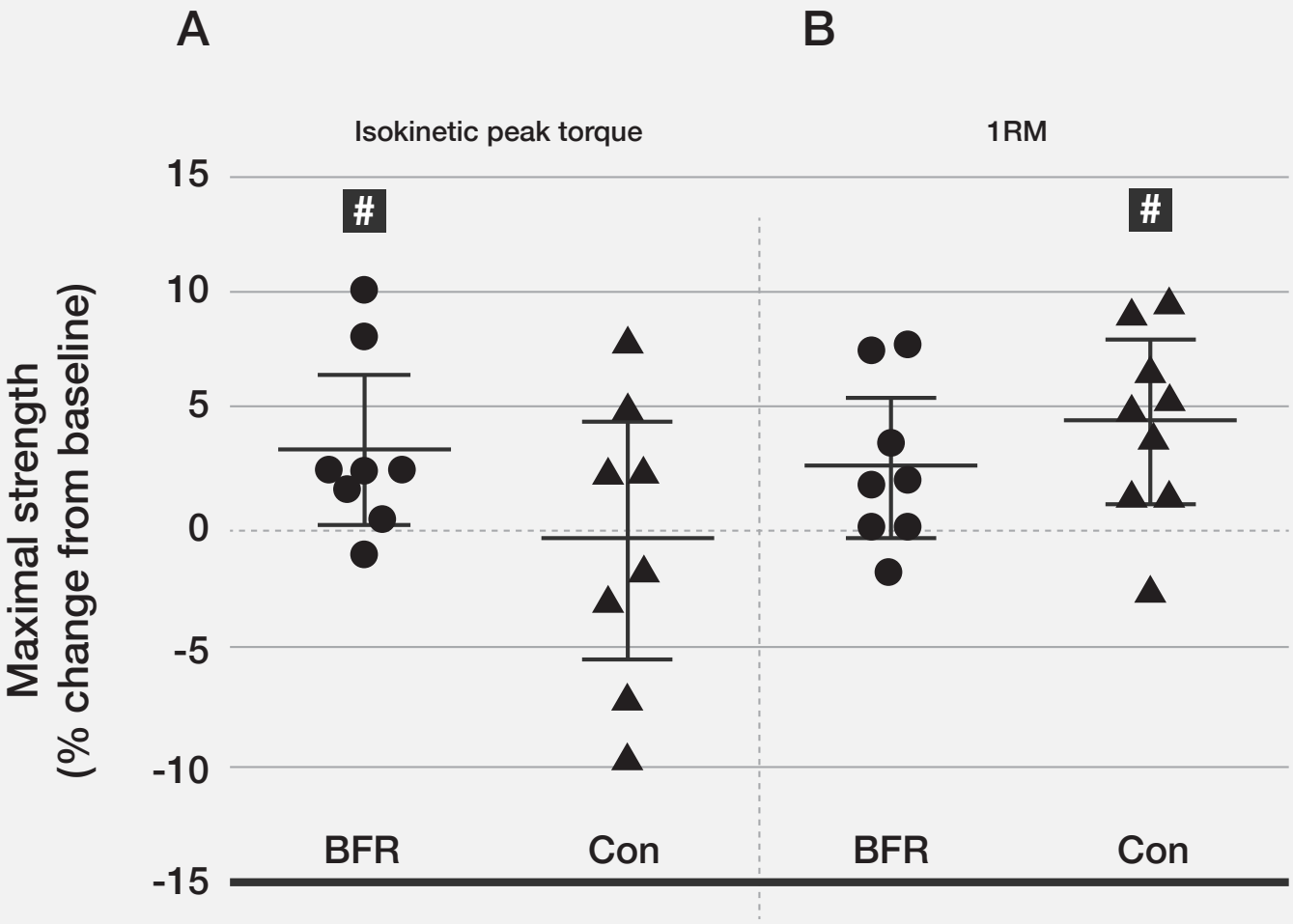
Figure 2



*BFR = blood flow restriction; * = significant difference between groups; # = significant within-group change from pre- to post-training*

Measures of hypertrophy

Figure 3



BFR = blood flow restriction; # = significant within-group change from pre- to post-training

Type I muscle fiber CSA, vastus lateralis CSA, rectus femoris thickness, vastus lateralis thickness, and vastus medialis thickness increased significantly more in the blood flow restriction group.

Rectus femoris CSA increased significantly pre- to post-training in the blood flow restriction group but not the traditional group, but the difference between groups wasn't quite significant (p=0.09).

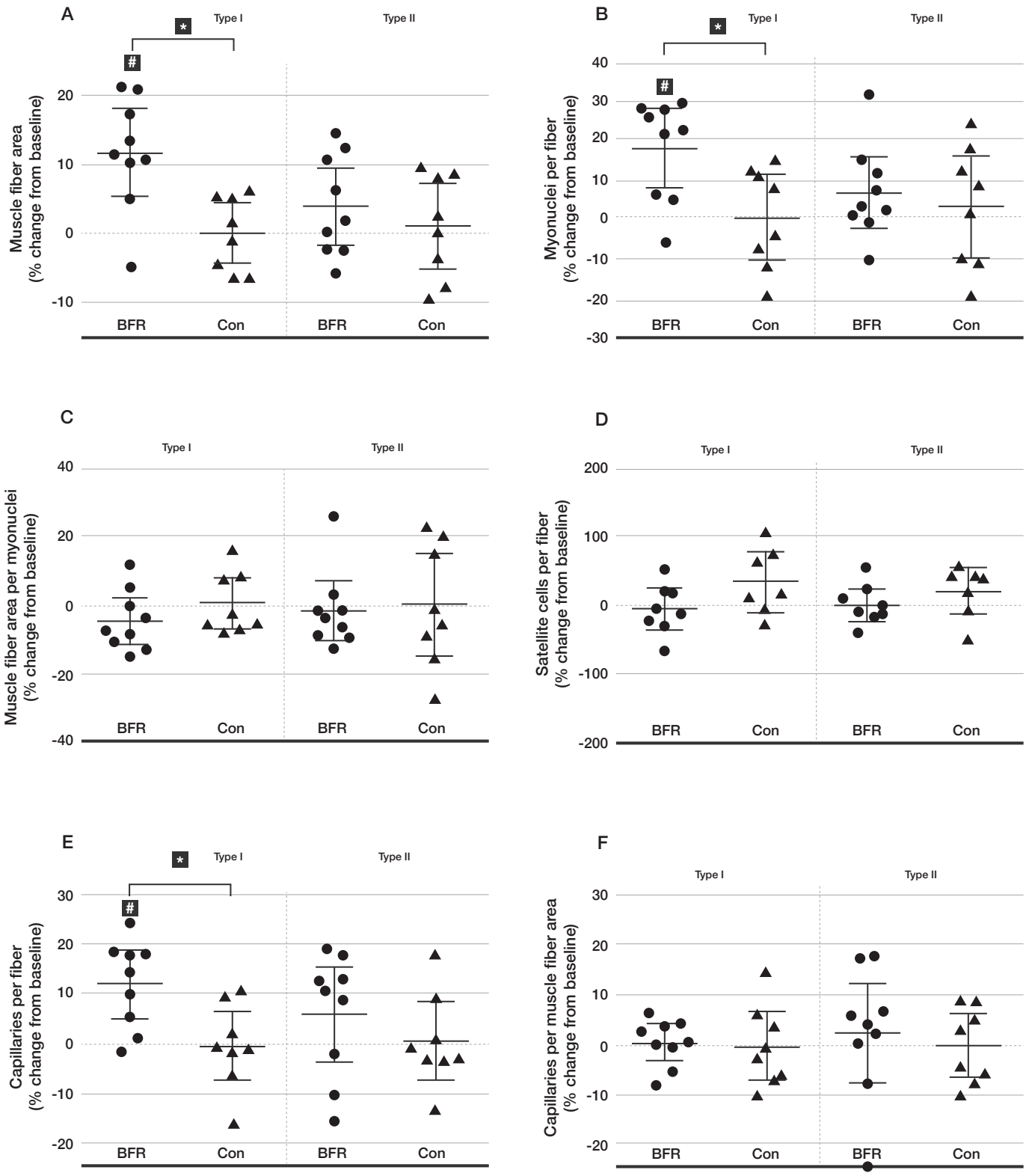
Type II muscle fiber CSA and vastus intermedius thickness didn't significantly increase in either group.

In the blood flow restriction group, changes in type I fiber CSA were strongly and positively associated with changes in vastus lateralis CSA (r=0.81).

Strength

There were no significant between-group differences for changes in

Figure 4



*BFR = blood flow restriction; * = significant difference between groups; # = significant within-group change from pre- to post-training*

front squat 1RM or changes in maximum isometric contraction force of the knee extensors.

However, isometric knee extension strength significantly increased pre- to post-training in the blood flow restriction group, but not the traditional group. The opposite was true with the front squat – a significant pre- to post-training increase in the traditional group, but not the blood flow restriction group (though the increase was nearly significant in the blood flow restriction group: p=0.08).

In the blood flow restriction group, changes in isometric knee extension force were correlated with changes in summed rectus femoris and vastus lateralis CSA (r=0.68) and changes in type I fiber CSA (r=0.79).

Myonuclei and satellite cells

The number of myonuclei in type I fibers increased more in the blood flow restriction group than the traditional group, while no significant myonuclear accretion was observed in type II fibers in either group. The average myonuclear domain size did not significantly change in either fiber type in either group. There also weren't significant changes in the number of satellite cells per fiber in either group.

Capillarization

Neither group saw increased capillarization (capillaries around each fiber) pre- to post-training for type II fibers.

THIS WAS A REALLY SURPRISING STUDY, AS IT PROVIDED THE FIRST STRONG EVIDENCE FOR FIBER TYPE-SPECIFIC HYPERTROPHY.

Capillaries per type I fiber increased pre- to post-training in the blood flow restriction group, but not the control group. However, the between-group difference wasn't quite significant (p=0.07).

RNA

The specifics here wouldn't really be of use for athletes or coaches, so to briefly summarize the results: RNA for some regulators of satellite cell differentiation and fusion, and RNA for some markers of ribosomal capacity (ribosomes are what synthesize proteins), increased more in the blood flow restriction group than the traditional group. Overall, however, the RNA findings were a bit muddled, and probably aren't directly relevant for the vast majority of MASS readers.

EMG

Vastus lateralis EMG was higher during heavy (80% 1RM) front squats

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without blood flow restriction than low-load (30% 1RM) front squats with blood flow restriction.

Interpretation

This was a really surprising study, as it provided the first strong evidence for fiber type-specific hypertrophy. That's a topic we've covered before in MASS (2), and the prior evidence was … opaque at best. However, the results of this study are both clear and striking: An increase in type I fiber CSA of 12%, without any type II fiber hypertrophy, after just *two weeks* (10 sessions) of front squats with blood flow restriction.

Before digging deeper into these results, I first want to address a common misconception about fiber types. Many strength athletes believe that type II fibers are substantially better for strength sports than type I fibers, and that selective type I hypertrophy may even be detrimental (adding mass without much of an increase in performance). This belief is based on the fact that type II fibers have a higher shortening velocity than type I fibers, and thus produce more power. However, type I and type II fibers produce about the same amount of *force* per unit of cross-sectional area (3). In other words, type II fibers are almost certainly beneficial for tasks like sprinting or jumping and may be beneficial for power-based strength sports (like weightlifting or highland games), but fiber type proportions probably don't impact performance in a sport like powerlifting or in most strongman events. In fact, previous research (4) has shown that powerlifters have roughly the same proportion of type I and type II fibers as people in the general population, indicating that the sport doesn't seem to preferentially select for people with a specific fiber type profile.

So, with that out of the way, let's dig in to some of these findings.

So, to this point, prior research suggested that adding low-load blood flow restriction training to normal, heavier training may enhance strength gains but not promote additional hypertrophy. The results of the present study run the opposite direction – no strength benefits, but a sizeable hypertrophy benefit.

I was surprised by the amount of hypertrophy observed in such a short period of time in the blood flow restriction group. The fiber type-specific findings may seem a bit esoteric, but whole-muscle CSA of the rectus femoris and vastus lateralis increased by 7-8% as well. Muscle CSAs barely changed in the traditional group, so it seems that the growth is almost entirely attributable to the two weeks of blood flow restriction training. And honestly, I'm having a hard time coming up with a clear explanation for this result. Low-load training with blood flow restriction doesn't seem to be any better for hypertrophy than heavier training without blood flow restriction (5), so one would *assume* that subbing out heavier front squat training for low-load training with blood flow restriction wouldn't lead to any additional growth.

The two most likely explanations seem to be a) training to failure or b) novelty. The details of the traditional, heavier training weren't provided (beyond a very rudimentary description), but it doesn't seem that the traditional group trained to failure. Some research indicates that training to failure may be better than non-failure training for hypertrophy (6), while other studies find no significant differences (7, 8), so I'm not sold on failure as the differentiating factor. I'm even less sold on novelty as the differentiating factor; people constantly posit "novelty" as a reason for hypertrophy in response to a non-traditional training stimulus, but I'm not aware of any research actually indicating that novelty makes much of a difference. If it did, you'd expect low-load training (i.e. 30+ reps per set with <50% of 1RM) to produce substantially greater hypertrophy than heavier, "normal" training (since most people don't do that many reps with that light of a load during "normal" training), but that's largely not what you see in the research (9, 10). However, the population may make a difference. In a sample of well-trained powerlifters, low-load training with blood-flow restriction may have been such a large departure

from their normal training (presumably quite heavy, and not to failure) that there was some sort of novelty effect (exploiting an untapped avenue of adaptation), whereas the typical gym-goers used as subjects in most studies may still train light enough and close enough to failure that low-load training with blood flow restriction isn't novel enough to have a big additive effect. I don't personally find that to be an intellectually satisfying explanation, but it's the best I can come up with.

The results of this study are even more surprising when stacked up against prior research. A study by Yamanaka et al (11) looked at the effects of adding low-load squat and bench press training, with or without blood flow restriction, to the offseason strength training program of Division I football players. That study found larger strength gains in the group using blood flow restriction, but the only major hypertrophy difference was in chest girth (which you wouldn't expect to be affected by blood flow restriction applied to the arms); changes in thigh girth were similar between groups, changes in right arm girth were similar between groups, and changes in left arm girth were significantly different, but the mean difference was tiny (0.7cm). A study by Luebbers et al (12) used a similar design and had similar findings: no differences in hypertrophy, but larger strength gains in the squat for the group using blood flow restriction. Finally, a Masters Thesis by O'halloran (13) compared heavy training (>70% of 1RM) to a program consisting of about two-thirds heavy training and one-third low-load training with blood flow restriction. It found no significant differences in strength gains or hypertrophy. So, to this point, prior research suggested that adding low-load blood flow restriction training to normal, heavier training may enhance strength gains but not promote additional hypertrophy. The results of the present study run the opposite direction – no strength benefits, but a sizeable hypertrophy benefit.

I can see three possible explanations for the difference. The present study used well-trained powerlifters, while prior research had used football players (Yamanaka and Luebbers) or just generally trained subjects (O'halloran). As previously mentioned, if there is a "novelty effect," it may only apply to populations, such as powerlifters, who typically carry out ultra-specialized training. Furthermore, the present study assessed hypertrophy via direct measures (fiber CSA, muscle CSA, and muscle thickness), while two of the prior studies (Yamanaka and Luebbers) just assessed limb circumferences. The study by O'halloran measured limb circumferences and muscle CSAs and demonstrates how those two methods of assessment can arrive at different conclusions: muscle CSAs in both groups decreased non-significantly (by about 7mm^2), while thigh circumfer-

ences increased significantly (by about 1.5cm). So, it's possible that the Luebbers and Yamanaka studies would have found hypertrophy differences if they directly assessed hypertrophy.

The third possible explanation deals with the training protocol itself. When I first skimmed the abstract of this study, I thought the intervention seemed somewhat bizarre. During a 6.5-week training program, low-load front squats with blood flow restriction were only performed for two nonconsecutive weeks. However, the authors' rationale was based on prior work (14), which suggested that the positive impact of low-load blood flow restriction training on hypertrophy and satellite cell proliferation reached a plateau after eight days. The authors thought that two nonconsecutive weeks of low-load training with blood flow restriction would allow for two small growth spurts, whereas the benefits of continuous low-load training with blood flow restriction may plateau after the first week. In other words, one week of low-load training with blood flow restriction may be just as good as multiple consecutive weeks, but multiple nonconsecutive weeks may offer an additional benefit, by allowing a wash-out period to re-sensitize the lifters to the stimulus. That's certainly an interesting idea that I'd love to see explored in future research.

Moving on to the strength findings, the authors note that some of the athletes weren't well-familiarized with front squats, which may have muddied the water. They state that the members of the traditional group (which performed 16 heavy front squat sessions, versus just 6 heavy sessions in the group doing low-load blood flow restriction training) seemed to improve their technique and core strength more. In spite of this, mean strength gains in the front squat were similar between groups (~4% for the traditional group versus ~3% for the blood flow restriction group). It would have been nice if they also tested back squat 1RM to see how well both groups' training transferred to the athletes' competition lift.

IT MAY BE BEST TO USE BLOOD FLOW RESTRICTION TRAINING EITHER EVERY OTHER WEEK, OR EVERY THIRD WEEK.

Most of the other findings were either unsurprising (EMG) or not incredibly useful for MASS readers (markers of ribosome biogenesis). However, it's worth speculating about why this study found clear preferential type I fiber-specific hypertrophy, while prior research did

APPLICATION AND TAKEAWAYS

If you've been struggling to grow, concentrated one-week (non-consecutive) blocks of blood flow restriction training may help you make some quick progress. However, the results of this study are at odds with previous research, so they should be viewed with some caution.

not. The authors speculate that using a bilateral compound exercise involving a large amount of muscle mass induced a lot of central fatigue, hindering recruitment of type II fibers. While that's certainly possible, prior research using compound lower-body exercises hasn't found fiber-type specific hypertrophy (9). It's also possible that the powerlifters in this study had accrued type II fiber-specific hypertrophy over years of heavy, non-failure training, leading to "catch-up growth" of type I fibers when exposed to a metabolically stressful stimulus. However, I find that unlikely, as these lifters' type I fibers weren't disproportionately small, compared to their type II fibers (they were 10-15% smaller, which is pretty typical). Lars Samnøy, one of the Norwegian powerlifting coaches, also informed us that many of these lifters regularly did accessory lifts in moderate rep ranges, so it's unlikely that they were completely neglecting their type I fibers during their normal training. Finally, it's possible that the increased capillarization allowed for type I fiber growth in the blood flow restriction group. Capillary density has been found to be predictive of hypertrophy in elderly subjects (15), so it's possible that insufficient capillarization places a cap on type I fiber size, and thus increased capillarization allows for type I fiber hypertrophy. I feel like this explanation would raise more questions than answers (What's the mechanism? Why didn't capillarization of type II fibers increase as well?), but we can't rule it out as a possibility.

In terms of application, *if* the hypertrophy responses in this study were due to the fact that the blood-flow restriction training took place on non-consecutive weeks, it may be best to use blood flow restriction training either every other week, or every third week. You could use a compound exercise (as this study did) or use blood flow restriction on accessory lifts. A wider wrap doesn't require as much pressure to occlude blood flow (which is beneficial for both comfort and safety), so use knee wraps instead of exercise bands, and aim for a pressure of tight-but-not-painful. If you get an insane pump, you wrapped to the appropriate pressure. If your limbs start turning purple and you don't get an insane

pump, you wrapped too tight and you're cutting off too much arterial blood flow. Use a weight between 20-40% of 1RM, knock out 3-4 sets with 15-45 seconds between sets, and enjoy the burn.

I really enjoyed this study because it gave me a lot to stew on. Were the hypertrophy differences due to novelty? Were they the result of training to failure? Why did the results of this study differ from those of previous low-load blood flow restriction studies in athletes? Why did this study find very clear type I-specific fiber hypertrophy when other studies failed to? Those are questions for future research to expand on, but I'll be mulling over this study for quite some time.

Next Steps

First and foremost, I'd like to see the results of this study replicated and extended. If the "trick" was having blood flow restriction training on non-consecutive weeks, I'd like to see another longer duration study in powerlifters, with blood flow restriction training once per three weeks, over a 12-week span (four three-week blocks, with one week of blood flow restriction training and two weeks of normal, heavier training). In that study, it would be good for one-third of the training in the control group to be taken to failure in order to rule out the possibility that the results of this study were simply due to failure training. I'd also like to see this finding replicated in another population (such as recreationally trained lifters) who don't have a background of specialized training. That would help rule out the possibility that the results of this study were attributable to novelty.

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Study Reviewed: Time Course of Recovery From Resistance Exercise With Different Set Configurations. Pareja-Blanco et al. (2018)

Leave the Gym with a Little Left in the Tank

BY MICHAEL C. ZOURDOS

We've written about failure training a few times, but this study was a monster. It compared 10 different conditions and the time course of recovery with five conditions to failure and five conditions ranging between a 5-8RPE. So how close to failure should you train?

KEY POINTS

1. This paper examined the time course of recovery between training to failure and leaving some reps in the tank across different repetition ranges.
2. In general, training to failure elongated recovery time versus non-failure training, with higher rep sessions to failure being particularly fatiguing.
3. When putting this study into context, it provides evidence that it may be wise to avoid failure training, at least in some sessions, to meet weekly volume and frequency recommendations.

Designing a program for hypertrophy and strength is multi-faceted, in that variables such as volume, intensity, and frequency cannot be considered in isolation. Therefore, after the overall trends (i.e. periodization) of these variables are decided, we then must consider how these variables shall be configured with meso- and microcycles. Variables cannot be considered in isolation because they are all interrelated. For example, if someone wishes to train the squat three times per week but packs excessive volume into Monday's session, leading to a 96-hour recovery time, then three squat sessions won't happen. However, even if volume was lower in Monday's session, the proximity to failure at which someone trains also affects recovery time. Specifically, Moran-Navarro et al (2017) (reviewed in MASS) showed that when session volume is equated, training to failure with 10 reps elongates recovery (2) versus training to ~5RPE. Therefore, it seems that staying shy of failure, at least early in the training week, is wise to ensure all weekly training variables are optimized. However, we don't yet know if failure training would still exacerbate fatigue during low rep sets and how different proximities to failure (i.e. ~5RPE versus ~8RPE) would affect recovery. The present study (1) was a monster in that it examined fatigue and muscle damage for 48 hours following 10 different program configurations among 10 men with 2-4 years of training experience. Subjects performed sessions of three sets on the Smith machine bench press and squat in each session. Sets were either performed to failure (i.e. 12 reps when 12 were possible) or with

Listen to Greg, Eric, and Mike discuss this study and topic in the audio roundtable.

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Table 1 Subject Characteristics

Subjects	Age (yrs.)	Body Mass (kg)	Height (cm)	Training Age (yrs.)	Squat 1RM (kg)	Bench 1RM (kg)
14 Men	22.1 ± 3.5	73.5 ± 10.7	175 ± 7	2-4	101.7 ± 14.4	88.4 ± 19.0

Data are mean ± SD
Subjects characteristics from Pareja-Blanco et al. 2018 (1).

half the number of possible reps (i.e. 6 reps when 12 were possible) using two sessions each of 4, 6, 8, 10, and 12 reps. In general, lifters had longer-lasting measures of fatigue and indirect markers of muscle damage following the sessions in which they trained to failure. Fatigue was particularly exacerbated following the higher rep sessions when training to failure (10 and 12 reps). It seems quite clear that training to failure, especially with high reps, should be carefully placed within a training week so that it does not harm other training variables. This article will explore how these results impact the configuration of a training week and will provide context for failure versus non-failure training.

Purpose and Research Questions

Purpose

The purpose of this study was to examine the time course of fatigue and recovery following training protocols involving failure and non-failure and training sessions with high reps and low reps.

Research Questions

How much does training to failure and the amount of reps performed per set affect the magnitude and duration of fatigue and recovery?

Hypothesis

While a formal hypothesis was not given, it seems that the authors expected training to failure, particularly during higher rep sessions, to elicit the greatest magnitude of fatigue and cause the longest recovery time.

Subjects and Methods

Subjects

Ten men with 2-4 years of training experience completed the study. Details of the subjects are available in Table 1.

Protocol

Over 20 weeks, subjects performed 10 different set and rep configurations of the bench press and squat. They had fatigue and muscle damage measured before training along with 6-, 24-, and 48-hours post-training. Bench press preceded squat

Table 2 List of Outcome Measures

Mechanical measurements of fatigue	Velocity against a load that could be performed at 1m·s^{-1} when fresh
	Vertical jump height
Biomechanical measurements of fatigue/muscle damage	Creatine kinase
	Testosterone
	Cortisol
	Prolactin
	Insulin-like growth factor-1
	Growth hormone

Outcome measures assessed in Pareja-Blanco et al. 2018 (1).

training in all sessions. All protocols consisted of three sets, with some protocols involving training to failure and others including different proximities to failure. The following represents the number of reps performed in each set with the predicted number of possible reps in parentheses; for example, 10(12) indicates 10 reps per set with 12 predicted, so this would be a non-failure protocol. The protocols were as follows: 12(12), 10(10), 8(8), 6(6), 4(4), 6(12), 5(10), 4(8), 3(6), 2(4). Therefore, the non-failure sessions ranged in proximity to failure from an 8RPE to a 5RPE (RPE = rating of perceived exertion). The approximate intensities used for each number of repetitions or possible repetitions were as follows: 12 reps: ~70%, 10 reps: ~75%, 8 reps: 80%, 6 reps: 85%, and 4 reps: 90%.

Fatigue and Muscle Damage Measurements

Vertical jump height and average velocity against a load that could be performed at 1 m·s^{-1} were tested to assess fatigue. Further, a myriad of biochemical parameters were assessed. The list of all fatigue measurements can be seen in Table 2.

Findings

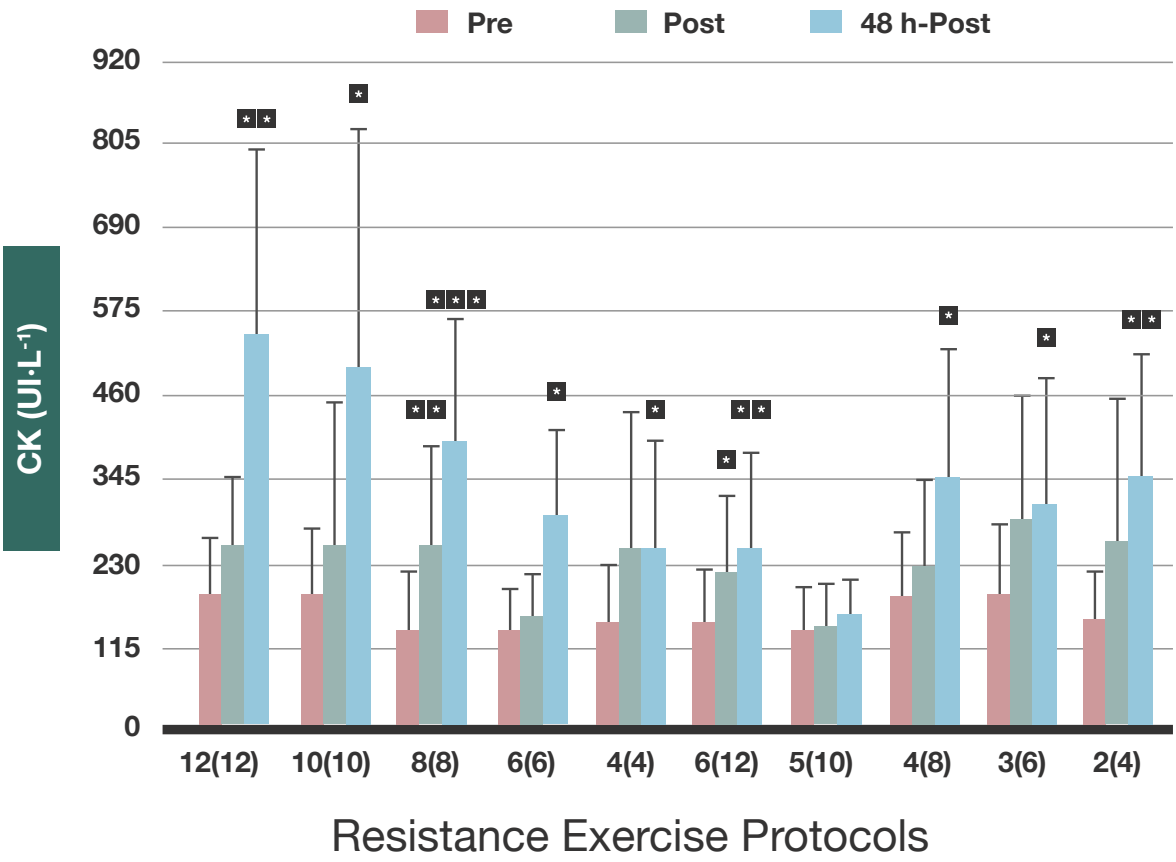
In general, the results were quite clear. The protocols to failure, and particularly those with higher repetitions (i.e. 6-12 reps per set), caused greater and longer lasting declines in vertical jump height and lifting velocity than non-failure protocols. Specifically, in Table 3, you can see the higher rep sets to failure led to diminished vertical jump and in some cases diminished velocity at 24 hours post-training.

Table 3 Changes in Mechanical Measures

Protocols	Post	6 h-Post	24 h-Post	48 h-Post
BP V_1-load (%)				
12(12)	55.4 ± 13.2*	92.9 ± 5.1*	94.5 ± 6.4	95.8 ± 4.6
10(10)	57.8 ± 12.9*	91.7 ± 13.4	93.5 ± 7.3	98.2 ± 8.9
8(8)	56.7 ± 14.7*	96.1 ± 7.4	96.2 ± 7.4	98.4 ± 6.2
6(6)	69.5 ± 8.7*	95.8 ± 6.2	94.4 ± 7.6	97.2 ± 3.3
4(4)	80.5 ± 8.5*	95.5 ± 7.4	95.7 ± 4.7	101.6 ± 5.0
6(12)	85.7 ± 6.5*	101.4 ± 5.8	101.2 ± 5.6	102.4 ± 7.3
5(10)	86.5 ± 6.3*	101.4 ± 5.1	102.0 ± 4.7	100.8 ± 3.7
4(8)	88.9 ± 5.4*	100.8 ± 6.8	100.9 ± 5.9	104.9 ± 5.2
3(6)	87.8 ± 5.4*	98.6 ± 5.5	98.5 ± 5.4	102.2 ± 7.6
2(4)	94.7 ± 6.5*	98.6 ± 4.2	100.9 ± 5.9	103.1 ± 6.9
SQ V_1-load (%)				
12(12)	70.0 ± 8.9*	96.2 ± 9.0	93.7 ± 7.3	97.3 ± 6.6
10(10)	77.8 ± 7.0*	95.9 ± 3.9	92.0 ± 2.6*	96.8 ± 4.0
8(8)	73.9 ± 6.6*	92.7 ± 7.9	95.5 ± 5.1	100.8 ± 5.5
6(6)	87.7 ± 10.6*	94.7 ± 5.0	93.3 ± 4.0*	98.5 ± 3.0
4(4)	80.2 ± 9.0*	98.3 ± 4.2	97.6 ± 4.3	101.1 ± 4.2
6(12)	86.3 ± 7.4*	101.3 ± 3.6	100.4 ± 5.2	99.2 ± 7.3
5(10)	91.0 ± 3.9*	97.2 ± 4.5	94.7 ± 3.4*	96.5 ± 4.1
4(8)	88.6 ± 6.5*	100.9 ± 7.8	98.8 ± 5.5	102.1 ± 7.6
3(6)	93.9 ± 6.7*	97.3 ± 4.1	101.5 ± 7.3	103.8 ± 5.8
2(4)	89.1 ± 4.9*	97.8 ± 8.9	96.2 ± 3.5	100.1 ± 6.1
VJ (%)				
12(12)	68.1 ± 11.2*	92.9 ± 4.6*	92.8 ± 5.3*	95.7 ± 5.0
10(10)	68.8 ± 7.3*	91.8 ± 6.4*	91.4 ± 3.9*	96.0 ± 4.5
8(8)	67.5 ± 5.3*	91.5 ± 5.0*	93.9 ± 4.6*	95.6 ± 5.8
6(6)	75.1 ± 6.2*	91.0 ± 7.7*	93.8 ± 2.2*	96.3 ± 3.8
4(4)	76.7 ± 2.9*	96.5 ± 4.2	95.6 ± 2.9*	101.6 ± 3.7
6(12)	78.5 ± 3.9*	99.5 ± 3.3*	98.3 ± 3.4	101.6 ± 3.2
5(10)	78.6 ± 3.5*	99.4 ± 2.9*	99.3 ± 3.0	101.5 ± 1.7
4(8)	78.0 ± 3.9*	99.3 ± 3.2*	99.5 ± 3.9	101.9 ± 3.7
3(6)	79.7 ± 3.6*	97.1 ± 2.8	96.9 ± 4.1	99.5 ± 3.1
2(4)	79.8 ± 3.4*	95.6 ± 3.2*	95.5 ± 4.0	99.3 ± 2.4

Data are mean ± standard deviation and represent the percentage performance compared to baseline. For example, 90% represents a 10% decline in the that measure from baseline. BP V_1-load (%) = Bench press average velocity against a load which was performed at 1 $m \cdot s^{-1}$ at baseline. SQ V1-load (%) = Squat average velocity against a load which was performed at 1 $m \cdot s^{-1}$ at baseline. VJ = Vertical Jump. *$p<0.05$ for decreased performance from baseline.

Figure 1 Changes in Creatine Kinase



Data are mean ± standard deviation.
On the x-axis the numbers represent reps performed (possible reps predicted)
CK = creatine kinase, * = p<0.05 for an increase, ** = p<0.01 for an increase, *** = p<0.001 for an increase

Out of the biochemical markers (Figure 1), the most pertinent to our analysis is creatine kinase (an enzyme which is commonly used to assess the time course of muscle damage). Creatine kinase was still significantly elevated at 48 hours in all failure training protocols, but not in all non-failure protocols.

Further, the higher rep failure protocols [12(12) and 10(10)] had the largest creatine kinase elevations. For the other hormones, most have a half-life of a few hours, so they don't tell us much regarding the time course of fatigue and damage. In general, though, the high rep protocols to failure (i.e. highest volume) elicited the greatest acute response in these markers, which is not a new or surprising finding.

Interpretation

First, let's give a quick applause to the authors for carrying out a study with 10 different conditions. Hats off to them. That's a lot of work. With that said, I find these results unsurprising, but important. They're unsurprising because the highest rep sets to failure (12 and 10 reps) produced the greatest amount of damage and longest lasting fatigue. At first glance, someone could simply say that the greater damage in the 10(10) versus the 5(10) is simply due to more volume; however, even though volume may account for some of this difference, previous data have shown that when volume is equated between failure and non-failure training, failure training still elongates recovery (2).

ALL PROTOCOLS – EVEN THE LOW REP 4(4) PROTOCOL – HAD A SIGNIFICANT DECREASE IN VERTICAL JUMP AT 24 HOURS (BETWEEN -4.4 TO -7.2%), WHILE NONE OF THE NON-FAILURE PROTOCOLS HAD A STATISTICAL DECLINE IN VERTICAL JUMP AT 24 HOURS POST-TRAINING.

When looking at Table 3, we can see it's clear that training to failure elongates recovery time. Specifically, all protocols – even the low rep 4(4) protocol – had a significant decrease in vertical jump at 24 hours (between -4.4 to -7.2%), while none of the non-failure protocols had a statistical decline in vertical jump at 24 hours post-training. At 48 hours, none of the failure protocols were associated with a statistical decrease in vertical jump, but vertical jump was still decreased by about 4% in all failure protocols with at least six reps, while vertical jump wasn't decreased more than 0.7% at 48 hours across all non-failure conditions. Again, this phenomenon is unlikely to be explained by volume; if we take the 5(10) condition and 4(4) condition using a theoretical lifter with a 100kg squat max, this individual would have performed a total volume of 1,050kg and 1,080kg, in their respective conditions. That's essentially the same volume, yet the failure condition, even when low reps were used, lengthened recovery time. Average concentric velocity tended to be diminished more in the failure versus non-failure protocols at 24 hours; however, it wasn't as clear as vertical jump. Further, since average velocity was tested at a load that could

TRAINING TO FAILURE MAY LEAD TO MORE VOLUME IN ONE SESSION, BUT IT COSTS YOU VOLUME THROUGHOUT THE WEEK, AND THE LATTER IS A MORE VITAL GOAL.

be completed at 1.0 $m \cdot s^{-1}$ at pre-training, which is about 40-50% of 1RM, I don't hold that metric in as high regard as vertical jump, since vertical jump declines have been directly related to squat performance (3).

Specifically, Watkins et al (2017) examined vertical jump as a measure of readiness in both trained men and women and found that an 8% decrease in vertical jump height led to a 28% decrease in squat reps performed at 80% of 1RM (r=0.65). In other words, when vertical jump was decreased in the Watkins study by 8%, that cost the subjects an average of more than two reps per squat set. In the present study (1), the 10(10) and 12(12) conditions had declines in vertical jump of -6.3% and -8.0% at 24 hours (respectively), and declines of about 3% at 48 hours. Certainly, an actual test of squat and bench reps performed at 24 and 48 hours is the best metric of performance; however, vertical jump gives us a decent indication that training to failure harms performance for longer than non-failure training.

Confirming the vertical jump results, creatine kinase was increased during all failure conditions at 48 hours post-training, and the greatest elevations in creatine kinase were achieved in the high rep failure protocols [12(12), 10(10), and 8(8)]. However, it is worth noting that in conditions with reps performed below eight, creatine kinase elevations were pretty similar regardless of failure or non-failure except for the 5(10) condition, which did not show any increase in creatine kinase. Despite this, I believe the vertical jump results and high rep failure training CK increases drive home the point that high rep sessions to failure are the most damaging.

This study gives us our first look at the time course of recovery following various proximities to failure. There were five non-failure conditions: 6(12), 5(10), 4(8), 3(6), and 2(4). Although this is interesting, this study wasn't truly designed to answer the question "*What are the various recovery times regarding different submaximal proximities to failure?*" The reason being, the reps in each set here were quite different. So, even though the 2(4) condition was an 8RPE and the 5(10) was a 5RPE, it's difficult to make a direct comparison between the different RPEs for time course of recovery. A follow-up study should compare conditions train-

ing to a 9RPE, 7RPE, and 5RPE while performing the same reps, and thus, using different loads. Additionally, this study did not show any difference between the non-failure conditions at any time point, and our best metric of performance capabilities, vertical jump, was recovered at 24 hours in all non-failure conditions. It certainly seems likely to me that in the proposed scenario above, the 9RPE condition would have a longer recovery period than the 5RPE condition if number of sets were equated, and I would still expect the 9RPE condition to lead to longer lasting fatigue even if the 5RPE condition did more sets, and thus, more volume (more on this in "Next Steps"). That last point is speculation on my part, but it does lead us into a discussion on the consequences of failure.

Eric has written about the consequences of failure here. Since training to failure on the major compound exercises at a normal volume diminishes performance for at least 48 hours (maybe longer), this lingering fatigue could impact training sessions later in the week and could lead us to fail to meet our overarching training guidelines. Let's say your individualized volume is 12 sets on squat over a frequency of three times per week. Training on Monday with five sets to failure at 70% of 1RM might yield 15 reps, 10 reps, 7 reps, 6 reps, and 5 reps across all five sets, but the cost of that would be an inability to effectively train again until about Friday. Now you've still got seven sets to go to get to your set volume threshold, and you've already missed the mark of three squat sessions. However, 4 sets of 8 reps on Monday would likely have yielded RPEs between 5-8 on all sets (2-5 RIR) and allowed for effective training again on Wednesday and Friday. Thus, training to failure may lead to more volume in one session, but it costs you volume throughout the week, and the latter is a more vital goal. Importantly for strength, it's quite clear that failure training does not lead to larger strength gains than not training to failure (4). Finally, as we hypothesized above, when reps are equated, there may be more fatigue with training at 9RPE than at 5RPE, so not training to failure shouldn't always mean stopping only one rep short. In fact, Eric's dissertation (5) showed that well-trained lifters training to a 5-6RPE three times per week for eight weeks on the squat and bench press produced similar hypertrophy compared to training to about an 8RPE. This doesn't mean that training should always be to a 5RPE; in fact, it shouldn't, as training to that low of an RPE is likely leaving strength on the table (5). Therefore, when fitting the concept of proximity to failure into your periodized plan, you should likely stay further from failure when accumulating volume (i.e. volume blocks) and train closer to failure when peaking strength (i.e. intensity blocks).

APPLICATION AND TAKEAWAYS

1. Training to failure causes more fatigue and elongates recovery compared to non-failure training, particularly when higher reps are performed in a set.
2. The main consequence of always training to failure is that the longer recovery time could decrease volume and frequency throughout the rest of the week. Importantly, always training to failure does not seem to lead to greater hypertrophy or strength when looking at the literature. Leaving some reps in the tank shouldn't hinder your gains and will probably leave you more recovered for the next session.
3. Moving forward, we don't yet know if training to a 9RPE elongates recovery compared to a 5 or 7RPE. We need more data examining the time course recovery between different submaximal proximities to failure.

The above paragraph isn't to say failure training can never be used. For one, it's much easier to train to failure on assistance movements. If you keep your squats and benches short of failure, but you take leg extensions and dumbbell lateral raises to failure, that's probably OK. The consequences of failure are lower for single-joint assistance movements. Also, if you have a training frequency on a muscle group of three times per week, you could probably train to failure before your longest training break. For example, if you train Monday, Wednesday, and Friday, then you could probably get away with training to failure on Friday, as you'd have 72 hours to recover before your next session. Lastly, training to failure doesn't have to be an all-or-none principle. You could simply perform only your last set of squats to failure in the Friday session and then add some selective assistance work. That strategy shouldn't negatively affect your next session. If you do take a main lift to failure, just make sure to keep your technique as sound as possible, as technique breakdown when training to a 10RPE is also another consequence of failure.

Next Steps

In my mind, it is pretty clear that training to failure all the time is not the way to go. However, it would be nice to have a true comparison of the time course of recovery when comparing different proximities to failure. The next study I'd like to see is a crossover design study such as this one with repetitions equated (as described earlier), but each condition taken to a different RPE. Lastly, let's say that training to a 9RPE causes more damage and recovery time than training to a 5RPE. Then, in theory, it's easier to add sets (and thus, volume) if you are training to a 5RPE. So, if that's

true (I'm speculating), how much more volume is needed (i.e. how many more sets) when training at a 5RPE to cause the same damage as training at a 9RPE? I don't know, but I'd sure like to know.

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Study Reviewed: Prevalence of Indicators of Low Energy Availability in Elite Female Sprinters. Sygo et al. (2018)

Energy Availability in Strength and Power Athletes

BY ERIC HELMS

We sometimes view body fat simply as tissue to lose to improve the ratio of fat to lean mass and subsequently improve performance. But, we forget that the process of losing fat, and sometimes maintaining a lean physique, can harm performance.

KEY POINTS

1. The term "energy availability" refers to whether or not you have adequate energy to maintain not only the energy demands of exercise or sport, but also of normal physiological function.
2. You can be at energy balance, maintaining a stable body mass, but be in a state of "relative energy deficiency" where reproductive and metabolic function are downregulated to maintain energy balance. Prolonged low energy availability can have long-term negative health consequences.
3. In this study, elite female sprinters and hurdlers had their energy availability assessed at the start of a season after a break and after five months of training and competing. Surprisingly, three athletes began the season exhibiting signs of low energy availability, and after five months, this number increased to more than half of the cohort.

Energy availability in sport refers to the "left over" caloric intake for an athlete after training is subtracted for physiological function. This easily calculated value is expressed relative to lean mass but has limitations that I'll discuss in this article. As an example, a 10% body fat, 100kg athlete (90kg of lean mass) consuming 3000kcal and expending 400kcals on average in training (2600kcal "left over") has an energy availability of 28.9kcal/kg (2600kcal divided by 90kg). The present study (1) is one of the only studies to assess energy availability in female power athletes, though previous studies have linked low energy availability to negative health, performance, and psychological outcomes in athletes with very high energy expenditures or those who restrict their energy intake (aesthetic athletes like gymnasts). Symptoms of low energy availability in 13 national-level female sprinters and hurdlers were assessed at the start of the season and five months into the season via DXA scans, questionnaires, blood samples, metabolic testing, and blood pressure tests. Three athletes already displayed signs of low energy availability at pre-testing despite coming out of off-season rest. Five months into the in-season, this increased to 7 out of the 13 (54%). In this article, I will discuss the implications of these findings and their broader relevance to physique and strength athletes.

Listen to Greg, Eric, and Mike discuss this study and topic in the audio roundtable.

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Purpose and Research Questions

Purpose

The purpose of this study was to assess the prevalence of signs and symptoms of low energy availability in a group of elite (world class and top national-level) sprinters and hurdlers during the first five months of the competitive season.

Hypothesis

The authors provided no hypotheses, which is understandable given that this was an observational study on a variable rarely assessed in this population.

Subjects and Methods

Subjects and Study Design

Thirteen athletes competing in the 100-400m sprint or 100m hurdles completed this observational study. Subject characteristics are shown in Table 1.

Table 1 Baseline subject characteristics for female elite sprint athletes

	n=13
Age	21 ± 3
Height (m)	1.67 ± 0.06
Weight (kg)	60.4 ± 4.3
BMI (kg/m^2)	21.6 ± 1.4
Years training (years)	8 ± 4
Training (hours/week)	15 ± 6.2
Previous stress fractures (n)	5
Black or mixed/white (n)	8/5

Data are mean ± SD

Shortly after commencing training following an offseason recuperation period (i.e. the beginning of the season), pre-testing occurred. Then five months later, in their competitive season, post-testing was carried out.

Outcome Testing

Both pre- and post-testing occurred after an overnight fast at least 18 hours after the athletes' last training session. Athletes were instructed to not consume caffeine the morning of testing or calcium supplements within 48 hours of their DXA scans, as acute calcium supplementation can artificially augment bone density results derived from DXA. Hydration status was also controlled prior to the DXA scans.

The goal of testing was to assess various known primary and secondary signs and symptoms of low energy availability and also to assess anthropometric and self-reported data to see if there were any correlations between variables. Table 2 displays the primary and secondary signs and symptoms of low energy availability, the testing method, and a description of each.

Table 2 Primary and secondary signs and symptoms of low energy availability

Primary	Method	Description
LEAF-Q score	Questionnaire	Subjective signs of energy deficiency if high
Bone mineral density	DXA	Low bone density
Resting metabolic rate	Indirect calorimetry	Low resting energy expenditure
Amenorrhea	Self-report	Absence of menstrual cycle longer than 3 months
Estradiol	Blood test	Low for phase of menstrual cycle
Luteinizing hormone	Blood test	Low for phase of menstrual cycle
Follicle stimulating hormone	Blood test	Low for phase of menstrual cycle
Secondary	**Method**	**Description**
Iron status	Blood test	Low serum Fe
Fasting glucose	Blood test	Low fasting glucose levels
Fasting insulin	Blood test	Low fasting insulin levels
LDL cholesterol	Blood test	High LDL levels
Free T3	Blood test	Low thyroid levels
IGF-1	Blood test	Low IGF-1 levels
Systolic blood pressure	Automated cuff	Low systolic blood pressure
Diastolic blood pressure	Automated cuff	Low diastolic blood pressure

Findings

In Table 3, the mean changes from pre- to post-testing are displayed. At the group level, most variables remained unchanged, although there was a slight decrease in blood pressure and increases in IGF-1 and cholesterol.

Individual outcomes for the primary and secondary signs and symptoms of low energy availability are shown in all 13 athletes in Figure 1.

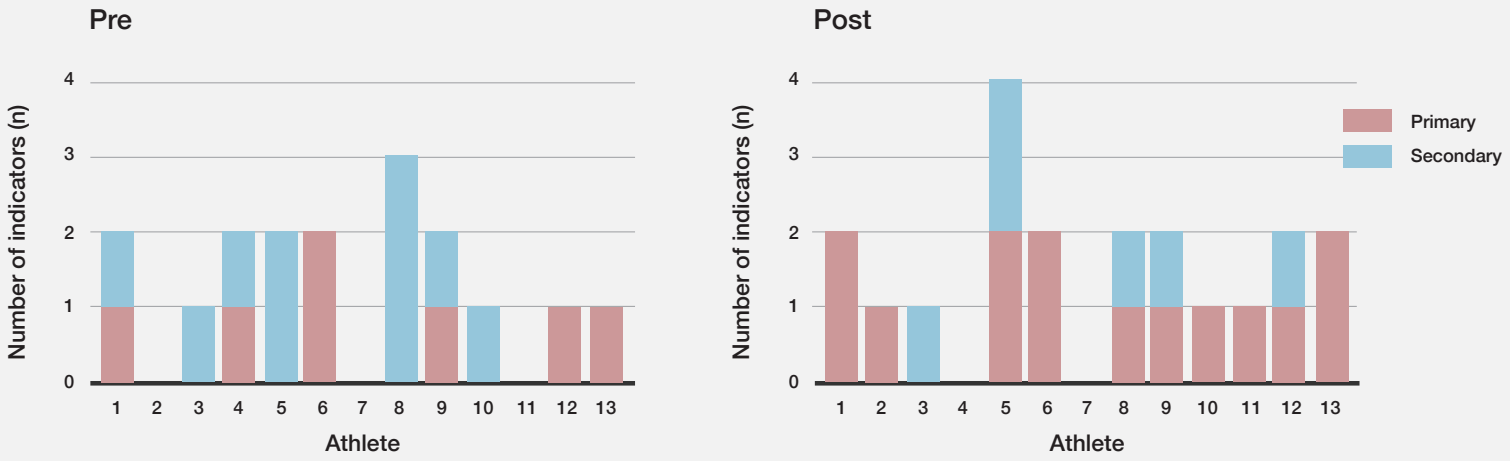
Researchers considered low energy availability present when an athlete showed primary and secondary signs of low energy availability. Specifically, four (31%) and seven (54%) of 13 athletes showed at least one primary and one secondary sign of low energy availability at pre- and post-testing, respectively.

Table 3 Changes in elite sprinters from pre- to post-testing

Body composition	PRE	POST
BMI (kg/m^2)	21.6 ± 1.4	21.8 ± 1.7
Body fat (%)	20 ± 2.9	20 ± 3
Fat mass (kg)	11.9 ± 2	12.1 ± 2.2
Lean mass (kg)	45.4 ± 3.7	45.9 ± 3.9
Sum of 8 skinfolds (mm)	76.7 ± 18.7	73.3 ± 16.9
Lean mass index	31 ± 2.7	31.3 ± 2.8
Metabolism		
REE (kcal)	1566 ± 162	1568 ± 150
REE/kg FFM (kcal)	33 ± 4.2	32.1 ± 2.5
RMR (% predicted)	104.7 ± 11.8	104.7 ± 6.4
RER	0.8 ± 0.05	0.81 ± 0.1
LEAF-Q		
LEAF-Q (score)	5.2 ± 3.6	6.8 ± 3.6
Blood work		
Hb (g/L)	128 ± 9	131 ± 7
sFe (µg/L)	66.5 ± 55.3	54.8 ± 41.7
Fasting glucose (mmol/L)	4.5 ± 0.5	4.5 ± 0.4
Fasting insulin (pmol/L)	45.5 ± 19.3	65.9 ± 53.5
Cholesterol (mmol/L)	**4.05 ± 0.68**	**4.33 ± 0.68***
LDL (mmol/L)	1.94 ± 0.44	2.12 ± 0.42
HDL (mmol/L)	1.78 ± 0.45	1.88 ± 0.44
Free T3 (pmol/L)	4.44 ± 0.39	4.47 ± 0.36
IGF-1 (µg/L)	**294 ± 61**	**328 ± 67***
Bone mineral density		
BMD (g/cm^2)	1.17 ± 0.08	1.16 ± 0.08
Z-score#	0.23 ± 1.3	0.18 ± 1.2
Blood pressure		
Systolic (mm Hg)	**105 ± 9***	**110 ± 7***
Diastolic (mm Hg)	**63 ± 6***	**73 ± 10***

Data are mean ± SD
** = $p \leq 0.05$; # = relative to normative data on female sprinters (Mudd et al., 2004)*

Figure 1 Prevalence of primary and secondary low energy availability indicators in female sprint athletes (n=13) at pre-season (Pre), and after five months of indoor training (Post)



Three athletes (23%) displayed these signs at both pre and post. Athletes who had at least one primary and one secondary indicator of low energy availability had a higher fat mass, assessed via DXA (13.0 ± 2.3kg vs. 11.2 ± 1.6kg, p = 0.03) compared to athletes who did not.

5 out of 13 (39%) participants reported previous stress fractures that were not explained by energy availability or ethnicity, but the number of previous stress fractures was related to BMI, lean mass, and RMR (r = 0.63-0.73, p = 0.005-0.02).

Finally, 6 out of 10 (60%) athletes not using hormonal contraception presented with low sex hormones (LH, FSH, and/or estradiol) at post-testing.

Interpretation

In the introduction, I defined energy availability mathematically as energy intake minus energy expended for exercise, divided by lean body mass. This concept of energy availability was created by Dr. Anne Loucks, a pioneer in Female Athlete Triad research (if you're not familiar with the term, I'll discuss it later in this review), approximately 25 years ago. A large body of her work addresses the concept of an energy availability threshold of 30kcal/kg. When energy availability drops below this point, signs and symptoms of metabolic and reproductive (in women) downregulation occur. However, the researchers in the present study did not calculate energy availability; rather, they just screened for the symptoms of low energy availability. While some might see this as a problem, I think it's more appropriate, as a hard-line cut off at 30kcal/kg is scientifically problematic. Conceptually, there is no reason the body "sees" energy expended from exercise any differently than non-exercise activity. The original research establishing the 30kcal/kg threshold was done in a homogeneous sedentary group, so the threshold likely

Table 4 Athletes displaying signs and symptoms at all time points

Primary indicators	Number of athletes at PRE n (%)	Number of athletes at POST n (%)	Athletes with repeated episodes (n)
LEAF-Q (≥ 8)	3 (23)	5 (39)	3
BMD (≤ 1.09 g/cm^2)	1 (8)	2 (15)	1
RMR (<29 kcal/kg FFM)	2 (15)	1 (8)	1
Amenorrhea (self-report)	0	0	0
Estradiol# (pmol/L)	0	4 (31)	0
LH# (IU/L)	0	3 (23)	0
FSH# (IU/L)	2 (15)	3 (23)	0
Secondary indicators			
sFe (< 25 µg/L)	2 (15)	2 (15)	1
Fasting glucose (< 4.0 mmol/L)	2 (15)	1 (8)	1
Fasting insulin (< 20pmol / L)	1 (8)	1 (8)	1
LDL cholesterol (> 3.0mmol/L)	1 (8)	0	0
Free T3 (< 3.5 pmol/L)	0	0	0
IGF-1 (< 114 µg/L)	0	0	0
Systolic BP (<90mm Hg)	0	0	0
Diastolic BP (<60mm Hg)	4 (31)	2 (15)	2

 # = Cut-off values for Estradiol, FSH, & LH according to follicular phase (F), mid-cycle peak (P), and luteal phase (L). Estradiol: (F<77, P<139, L<77 pmol/L). FSH: (F<3, P<3, L<1.5 IU/L), LH (F<2, P<8, L<1 IU/L).

applies in many cases, but in those with higher (or perhaps lower) non-exercise activity levels, it doesn't. For many, symptoms of low energy availability may or may not occur to various levels of severity within the range of 30-45kcal/kg (or perhaps lower). For all of these reasons, it might be more appropriate to assess not only the mathematical relationship, but more importantly, the signs and symptoms associated with low energy availability (2).

With that said, let's discuss what energy availability is all about. The original work on energy availability showed that when energy intake was maintained while exercise energy expenditure was increased, metabolic and reproductive function were negatively affected. Subsequently, when energy intake was raised, the negative effects were reversed (3). In reading this, it may just seem like energy availability is another term for energy balance. You might be thinking "I already know an energy deficit can cause downregulation of reproduc-

tive and metabolic hormones." That's correct; an energy deficit does result in decreased production of metabolic and sex hormones in both men and women, but what is critical to understand is this downregulation can occur at energy balance as well. For example, you might remember my review of the giant (relative to other bodybuilding studies) Finnish study of physique competitors where at 3-4 months post-competition, not all competitors had regained their menstrual cycle despite regaining body weight. A more extreme example is the case study of a 26-27 year old drug-free figure competitor whose menstrual cycle did not return until 71 weeks after competition, despite a regain of her body weight a year prior (4). Likewise, in the present study, the body mass and composition of the athletes didn't change much during the season, but the percentage of the cohort displaying signs of low energy availability increased. Furthermore, those displaying signs and symptoms of low energy availability were *higher* in body fat than those not showing signs and symptoms. So what is going on here?

What we are seeing in this study and others, and what I see anecdotally as a coach far more often than I'd like, is the result of maintaining or attempting to maintain a leaner body than is healthy. By systems we are still attempting to fully elucidate, we know that energy expenditure is decreased (often disproportionately) in response to both energy deficits and significant reductions in body mass (5). However, the magnitude of this decrease in response to an energy deficit and the degree of body mass loss that causes further decreases in energy expenditure is highly individual. Elite athletes need specific genetic traits in order to be as good as they are, and it is no accident that all elite sprinters are lean. Purely from a physics standpoint, elite performance in sprinting requires such a body composition. I suspect many elite sprinters naturally settle at a low body fat level and don't do long or hard diets or experience much metabolic adaptation in the process of reaching their condition. However, there are surely some who have traits that allow them to be incredibly fast, but don't have the same level of natural leanness. Thus, they have

A HARD-LINE CUT OFF AT 30KCAL/KG IS SCIENTIFICALLY PROBLEMATIC. CONCEPTUALLY, THERE IS NO REASON THE BODY "SEES" ENERGY EXPENDED FROM EXERCISE ANY DIFFERENTLY THAN NON-EXERCISE ACTIVITY.

THE REASON THE SPRINTERS HIGHER IN BODY FAT SHOW MORE SYMPTOMS OF LOW ENERGY AVAILABILITY IS BECAUSE THEY ARE SHOWING THE SIGNS OF DIETING OR HAVING DIETED.

to do more dieting and regulate their body fat more assertively, spending more of the season in an energetically downregulated state. They chronically eat less, and to reach energy balance, their bodies expend fewer calories. However, despite being at energy balance while maintaining a reduced bodyweight, they exhibit symptoms of low energy availability, because part of this reduction in energy expenditure occurs via downregulating metabolic and endocrine function. Therefore, the reason the sprinters higher in body fat show more symptoms of low energy availability is because they are showing the signs of dieting or having *dieted*. A lean individual will often be in a state of low energy availability when in a caloric deficit, but the state of being at energy balance while exhibiting these signs and symptoms is known as "relative energy deficiency" (6).

Now, this wouldn't be a big deal if there weren't potential long-term health consequences to being in this state. Athletes who remain in a state of low energy availability can experience: negative effects on performance; negative effects on the endocrine, cardiovascular, immune, metabolic, reproductive, and gastrointestinal systems; the loss of menstrual function; and a reduction in bone health in women specifically. While these detrimental effects don't always coincide with disordered eating or a negative body image, they often do. The behaviors and self-regulation necessary to maintain low body fat and energy intake can lead to psychological stress due to the push and pull of the opposing desires to both maintain and relinquish control, as personal and athletic goals come into conflict with biologically driven desires. Low energy availability, menstrual dysfunction, and bone loss are known as The Female Athlete Triad, and the broader dysfunctions that can occur as a result of low energy availability (in men or women in sport) are collectively termed Relative Energy Deficiency in Sport or RED-S (6).

In power and strength athletes, there is traditionally less concern regarding The Female Athlete Triad or RED-S. For mechanistic reasons, high force activities are thought to be protective of bone health, and in support of that notion, fractures were not related to symptoms of energy availability in the present

APPLICATION AND TAKEAWAYS

If maintaining a certain level of leanness or body mass – and subsequently a restricted energy intake – results in the loss of menses or an irregular menstrual cycle, persistent food focus, more frequent illness, poorer mood state, an inability to increase performance, loss of libido, or metabolic or reproductive hormone panels outside of the reference ranges, increase your calories and consider that it may prove more optimal in the long run to maintain a higher body mass.

study. Rather, they were related to BMI, RMR, and lean mass (which really just means they were related to lean mass; more lean mass means a higher RMR and BMI). Thus, athletes who produce higher ground contact forces are more likely to get stress fractures, which is not at all surprising. Another reason strength and power athletes are not as well-researched in this area is their low energy expenditure compared to endurance sport. Additionally, clinicians unfamiliar with the nuances of strength sport often have a stereotyped perception of strength and power athletes as being big, bulky, and unafraid of consuming plenty of food. However, for weight class-restricted strength athletes and track and field athletes who have to propel their own bodies (rather than an implement), nutritional manipulation and restriction are incredibly common. The takeaway from this study is that even if you don't expend a ton of calories in training, if you maintain a leaner physique than your body "wants," there can be health and performance consequences. Sometimes, as was shown by the sprinters who started with symptoms, these consequences can even bleed over after an off-season recovery period.

Next Steps

I would love to see more research looking at the same symptomology in strength athletes who hold a potentially leaner than "normal" physique to stay in their weight class, and also bodybuilders who stay on the leaner side in the off-season. I think there are clearly potential performance benefits to both: a higher strength to mass ratio in the former, and a potentially less stressful diet in the latter. However, the level of leanness that provides the optimal balance between minimizing the potential negative effects and maximizing the potential positive effects is likely individual. It might also differ by the type of desired performance – strength, speed strength, or aesthetic – which would be clarified by similar studies in powerlifters, weightlifters, and physique competitors. Finally, I'd like to see more research

in the area of hormonal contraceptive use in female athletes who restrict their body fat, considering 6 in 10 athletes in this study who weren't taking them had reduced sex hormone concentrations. Are hormonal contraceptives helpful at keeping some of the negative effects of energy deficiency at bay, or are they covering up a bigger problem (or both)?

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Study Reviewed: Hormonal Contraceptive Use Does Not Affect Strength, Endurance, or Body Composition Adaptations to Combined Strength and Endurance Training in Women. Myllyaho et al. (2018)

Hormonal Contraceptives Don't Mitigate Strength Gains

BY GREG NUCKOLS

Many female athletes are still concerned that using birth control may hinder their strength gains or performance. Thankfully, that concern is probably misplaced.

KEY POINTS

1. Taking hormonal contraceptives does not seem to negatively impact performance or body composition changes after 10 weeks of strength training and high intensity aerobic training.

Many female athletes use hormonal contraceptives, but many athletes have concerns about them. Based on conversations with female lifters, there seems to be a fairly common belief that using hormonal contraceptives will make it harder to lose fat, gain muscle, or increase strength. Unfortunately, there's been a paucity of data on the subject, which helps contribute to the confusion.

In the present study, nine hormonal contraceptive users and nine nonusers trained for 10 weeks, doing a combination of strength training and high intensity aerobic training. Changes in performance and body composition – leg press 1RM, isometric leg press force, counter-movement jump height, 3000m run time, lean body mass, and body fat percentage – were very similar between groups. Thus, female athletes probably don't have to worry about their gains if they choose to use hormonal contraceptives, though the specific type of contraceptive may matter.

Purpose and Research Questions

Purpose

The purpose of this study was to investigate the effects of monophasic hormonal contraceptives on strength, endurance, and body composition outcomes after 10 weeks of combined resistance and high intensity aerobic training.

Hypotheses

The authors hypothesized that hormonal contraceptive use would lead to smaller improvements in strength, endurance, and body composition.

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Table 1 Pre-Training Characteristics

	Age	Height (cm)	Body mass (kg)	Body fat (%)	1RM leg press (kg)	Countermovement jump (cm)	3000m time trials (s)
Hormonal contraceptive users	28.2 ± 3.1	166.4 ± 5.0	59.3 ± 5.3	23.2 ± 7.1	114 ± 15	25.8 ± 3.0	829 ± 75
Non-users	31.3 ± 5.4	168.3 ± 5.0	60.6 ± 5.8	23.9 ± 6.7	118 ± 18	26.2 ± 4.9	806 ± 91

Subjects and Methods

Subjects

The subjects were 18 physically active women, aged 24-41. Nine women had been using monophasic hormonal contraceptives for at least one year, and nine had never used hormonal contraceptives. In addition to general health screening, the participants had to be reasonably fit; they had to be able to run at least 2300m (about 1.4 miles) in 12 minutes. They also needed to have regular menstrual cycles. All subjects had prior endurance training experience, but did not have prior resistance training experience. Further subject details are in Table 1.

Experimental Design

The participants trained for 10 weeks, with two lifting sessions and two high intensity cardio sessions per week. The lifting followed a linearly periodized approach, with loads increasing from 50% of 1RM to 85% of 1RM over the course of the study, using exercises targeting the lower body exclusively (squats, leg press, leg curls, and calf raises). They also performed a variety of jumping exercises. One day of the high intensity cardio consisted of four four-minute runs at intensities that increased from 70% of max heart rate to 90% of max heart rate. The second day of cardio consisted of three sets of three all-out 100m sprints, with two minutes of rest between sprints in each set, and five minutes between sets. Subjects were also encouraged to complete one low intensity cardio session per week, but that session wasn't monitored by the research staff.

All outcome measures were assessed before and after the 10-week training period and were scheduled to take place during the first five days of the women's monthly cycle. Performance measures included isometric leg press force, leg press 1RM, counter-movement jump height, and a 3000m timed run. Body composition measures consisted of lean mass and body fat percentage, assessed by DXA. Hormonal measures were also taken, but they were not primary outcomes of the study.

Findings

Both groups improved in measures of isometric leg press force and leg press 1RM, with no significant differences between groups. However, improvements in isometric leg press force tended to

Table 2 Summary of Results

	Hormonal contraceptives		Non-users			
Measure	Percent change	Within-group effect size	Percent change	Within-group effect size	Between group effect size	Significant difference between groups?
Isometric leg press force	15.1%	0.81	4.9%	0.25	0.57	no
1RM leg press	8.8%	0.60	8.5%	0.60	0.00	no
Countermovement jump height	5.0%	0.32	10.7%	0.69	-0.37	no
3000m time trial	-3.5%	0.35	-2.1%	0.20	0.14	no
Lean body mass	0.9%	0.11	2.1%	0.25	-0.14	no
Body fat percentage	-5.2%	0.17	-6.3%	0.22	-0.04	no

Effect sizes are color coded. Trivial effects ($d \leq 0.2$) are the lightest pink, small effect sizes ($0.2 < d \leq 0.5$) are the next lightest shade, medium effects ($0.5 < d \leq 0.8$) are the second darkest shade, and the large effect ($d > 0.8$) is the darkest red. A positive between-group effect size indicates a larger beneficial effect in favor of the hormonal contraceptive group.

be a bit larger for the group using hormonal contraceptives (~15% vs. ~5%; between-group effect size = 0.57). Similarly, counter-movement jump height increased significantly in both groups, with no significant differences between groups, and a small effect size difference favoring the nonusers. Improvements in 3000m run time didn't differ significantly between groups, though the improvement was only significant in the group using hormonal contraceptives (don't read too much into that, though; the between-group effect size was a trivial 0.14).

Body fat percentage decreased to a similar amount in both groups; the decrease was only significant in the women not using hormonal contraceptives, but the between-group effect size was trivial. Increases in lean mass tell a similar story; there was a significant increase in the nonusers but not the hormonal contraceptive users, but the between-group effect size was, again, trivial.

Interpretation

I almost thought I was going to be able to make it through an entire issue of MASS without having to complain about data reporting, but alas, this article broke the streak. However, my potent mixture of OCD and cynicism helps ensure you guys get an accurate representation of the results, so bear with me.

If you read this paper, two things should jump out to you: all of the effect sizes and some of the percent changes are implausibly large or just (seemingly) completely wrong. For an extreme example, this study also reported some hormonal data, and luteinizing hormone levels increased from 2.8 to 3.6mIU/L in the group taking hormonal contraceptives. When last I checked, an increase from 2.8 to 3.6 is a ~30% increase, but in this study, that's reported as a 616 ± 1697% increase. For an even more extreme example, testosterone *decreased* from 0.72 to 0.50 nmo-

TWO NULL RESULTS FROM STUDIES WITH DIFFERENT HYPOTHESES STRIKES ME AS PRETTY STRONG EVIDENCE THAT HORMONAL CONTRACEPTIVES PROBABLY DON'T NEGATIVELY IMPACT RESPONSES TO TRAINING.

l/L in the group taking hormonal contraceptives, but that's reported as an 88 ± 291% increase. I recognize that the mean percentage change computed from individual percentage changes may not perfectly match the percentage change in group means, but both of those numbers seem way off. There may be some really extreme distribution of percent changes where those numbers work out, but for the life of me, I can't figure out what it would be. And on the topic of effect sizes, for an extreme example, leg press 1RM increased by 10kg in both groups. That should be an effect size of zero (you calculate a Cohen's d, which is what is used in this study, by dividing the difference in mean change between groups by their pooled standard deviation). But, somehow, that's reported to be an effect size of 0.71 in the study, which would be classified as a medium effect, bordering on a large effect. I think they may have used the percentage change means and standard deviations to calculate their effect sizes, but the flaws of that approach should be obvious when you can wind up with an effect size of 0.71 in a situation where the change in group mean was identical in both groups.

So, when you look at the results table in this article, just know that the percent changes and effect sizes don't match what you'd see if you pulled up the full text of the presently reviewed study. I didn't have a brain fart doing data entry; I computed percent changes based on changes in group means, and computed effects sizes using the correct formula. This illustrates why it's important to read past the abstract and to actually think about the data reported in a study, instead of just accepting everything at face value.

In terms of the actual results, there didn't seem to be any major differences between groups for any of the performance outcomes. Surprisingly, this is only the second study (that I'm aware of) that tests whether hormonal contraceptives affect gains after strength training. The other study also found that hormonal contraceptive users gained strength just as effectively as nonusers (2). Interestingly, the authors of the presently reviewed study hypothesized

that hormonal contraceptives would decrease strength gains, while the authors of the older study hypothesized that they would increase strength gains. Two null results from studies with different hypotheses strikes me as pretty strong evidence that hormonal contraceptives probably don't negatively impact responses to training.

However, it should be noted that both of these studies used women taking contraceptives consisting of estrogen and progestins. There's some evidence (3) that birth control formulations that include antiandrogens (such as cyproterone acetate) may lead to smaller strength gains in response to training. As always, discussions about birth control are discussions you should have with your doctor; however, if you want to use hormonal contraceptives and you don't have a clear medical reason for using a formulation that includes antiandrogens, it would probably behoove you to seek out an option that only includes estrogen and progestins, or just progestins. It should also be noted that a conference abstract (7) reported smaller increases in lean mass in women taking oral contraceptives, though that study was never peer-reviewed and formally published, as far as I'm aware. It also found that women who used birth control formulations containing more androgenic progestins were more negatively affected than women who used formulations containing less androgenic progestins.

BIRTH CONTROL FORMULATIONS THAT INCLUDE ANTIANDROGENS MAY LEAD TO SMALLER STRENGTH GAINS IN RESPONSE TO TRAINING.

Some studies indicate that women who start hormonal contraceptives may gain some body fat within their first couple of months taking hormonal contraceptives (though the evidence is fairly mixed). In the present study, all women in the hormonal contraceptive group had been taking hormonal contraceptives for at least one year prior to the start of the study, so the results of this study wouldn't be able to account for those early changes in body composition (if they actually exist). Increases in body fat seem to be particularly common with progestin injections (4), but less common with other forms of hormonal contraceptives (5). In athletes, rates of perceived negative side effects are higher with progestin-only contraceptives as well (6). Overall, it seems that progestin-only contraceptives, or contraceptives containing more androgenic progestins (primarily found in first and second generation birth control pills) likely shouldn't be the top

APPLICATION AND TAKEAWAYS

If you're a female athlete and you want to use hormonal contraceptives, you can probably use most modern oral contraceptives without fear of sacrificing some of your potential gains.

choices for athletes. As always, however, these are subjects to discuss with a doctor.

Next Steps

Future research should look at the impact of different birth control options on muscle growth and strength gains now that we have evidence that, in aggregate, hormonal birth control consisting of estrogen and low-androgenic progestins doesn't seem to affect results. Since many female athletes use hormonal contraceptives, more granular information about the effects of different forms of birth control on muscle growth and strength gains would be helpful.

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Study Reviewed: Increased Rate of Force Development During Periodized Maximum Strength and Power Training is Highly Individual. Peltonen et al. (2018)

Power Training or Speed Work for Some, But Not All?

BY MICHAEL C. ZOURDOS

MASS has already covered individualization, but new information is emerging. In this study, rate of force development was maximized by some people through heavy training and by others through explosive training – but why? And what impact does that have for strength and hypertrophy?

KEY POINTS

1. This study examined how some people respond better to power-type training and some respond better to more traditional strength-type training.
2. To examine the responses to each type of training, 14 males trained for 10 weeks with strength-type training and then for 10 weeks with power-type training.
3. Six people improved rate of force development only in the strength block, while four people improved rate of force development only in the power block. Four people – "non-responders" – did not improve rate of force development at all. Importantly, all responders increased hypertrophy and strength to the same degree despite not improving rate of force development.

Maximizing one-repetition maximum (1RM) is, in part, a product of improving rate of force development. Rate of force development refers to how quickly muscle fibers can be activated, or the frequency at which muscle fibers can be stimulated within a motor unit (2). This ability to generate force quickly partly explains neuromuscular efficiency at a heavy load. For example, if two individuals squat 200kg at a velocity of 0.35 $m \cdot s^{-1}$, one might be able to squat 222.5kg at 0.22 $m \cdot s^{-1}$, while the other might top out at 215kg at 0.27 $m \cdot s^{-1}$. In this example, it's possible that the former individual has greater rate of force development, allowing this individual to work at a higher intensity, also resulting in a slower 1RM velocity. This study (1) aimed to examine if some individuals responded better to strength-type training, if some responded better to power-type training, and if some didn't respond to training at all. Twenty-four men participated, and 14 trained twice per week for 20 consecutive weeks. These 14 subjects performed a program focusing on maximal strength during weeks 1-10, and then performed a power-focused program during weeks 11-20. The other 10 subjects only completed pre-, mid-, and post-study measurements to serve as a control group. At pre-, mid-, and post-study, leg press 1RM, leg press power (at 50% of 1RM), isometric leg extension force and torque, hypertrophy of the quadriceps, and hormone levels were assessed. Additionally, to test the time course of responses and

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acute fatigue, isometric leg extension force and rate of force development were tested before and immediately after every seventh training session. After the 20 weeks, subjects were then categorized into groups for analysis dependent upon the phase in which they improved rate of force development. Specifically, subjects were categorized as maximum-strength responders (only improved during the strength period), power responders (only improved during the power period), or non-responders (no significant improvement over the entire 20 weeks). At baseline, those who ended up being classified as responders (of some kind) had higher 1RM strength and rate of force development than the non-responders. The maximum-strength responders increased rate of force development by 100% during the strength-focused block, but only added another 3% following the power-focused block. However, power responders increased rate of force development by only 11% after the strength-focused block, but improved by 53% following the power-focused training. Similarly, the power responders increased leg press power at 50% of 1RM during the power-training period and experienced decreased power during the strength-focused block, while the maximum-strength responders experienced the opposite. The other major findings include: both types of responders increased strength and muscle growth to the same degree over the 20 weeks, and the magnitude of increase in each did not differ between training type. Non-responders did not experience any significant change in muscle cross-sectional area despite training for 20 weeks; however, non-responders had similar strength gains to both responder groups. These results show that there are individual program design needs to maximize rate of force development. This article will discuss the relevance and application of these findings for lifters looking to maximize strength and will examine how programs may differ for non-responders based upon their difficulty to significantly hypertrophy muscle.

Purpose and Research Questions

Purpose

The purpose of this study was to examine if some people respond better to strength-focused training and if some people respond better to power-focused training.

Research Questions

1. Do some people respond better to strength-type training, and do some people respond better to power-type training?
2. If people respond differently to strength- and power-type stimuli, does the different response occur in all physiological and performance markers or only in some?

Table 1 Subject Characteristics

Subjects	Age (years)	BMI (kg·m^2)	Heigh (cm)
14 Men Training Group	28 ± 5	25 ± 4	179 ± 5
10 Men Control Group	30 ± 4	25 ± 2	178 ± 7

Data are Mean ± SD. BMI = Body mass index
Subject characteristics from Peltonen et al. 2018 (1)

Hypotheses

A formal hypothesis was not given. However, judging by the tone of the introduction, it seems as though the authors clearly expected there to be some who would classify as maximum-strength responders and some who would classify as power responders

Subjects and Methods

Subjects

Twenty-four men who were recreationally active (but with no resistance training experience) participated. Fourteen subjects performed the 20-week training program, while 10 subjects served as a control group and just did the pre-, mid-, and post-study measurements. More details about the subjects are available in Table 1.

Design and Training Program

This study spanned 20 weeks, with weeks 1-10 serving as a strength-focused training block, and weeks 11-20 being a power-focused block. The leg press, leg extension, and leg curl were performed twice per week for the 20 weeks; however, no details regarding the sets, reps, and intensities used were given. Fortunately, I inquired with the corresponding author, and he was very kind to offer me further insight. The training program was very similar to another recent study from the same group (3). In the strength-focused block (weeks 1-10), training was mainly 2-4 sets for 6-10 reps between 70-80% of 1RM. The power-focused block was mainly 3-5 sets for 4-10 between 30-50% of 1RM. In other words, the strength block was typical training, and the power training was explosive (with some moderate reps, but more on that later). During every seventh training session, rate of force development and force production were tested during a maximal isometric leg extension before

and after training to assess both the time course of progress and acute fatigue.

Outcome Measures

A plethora of outcome measures were assessed at pre- (Week 1), mid- (Week 10), and post-study (Week 20). Those measures are listed in Table 2.

Table 2 Outcome Measures at Pre-, Mid-, and Post-study

Outcome Measure
Leg press 1RM
Leg press power at 50% of 1RM
Leg extension isometric force
Leg extension torque
Rate of force development on the leg extension
Quadriceps cross-sectional area (via ultrasound)
Electromyography of the quadriceps during the leg extension isometric force test
Various hormones

1RM = One-repetition maximum

Findings

Group Categorization and Responder Magnitude

There were six maximum-strength responders, four power responders, and four non-responders. The response according to categorization was clear, as maximum-strength responders increased rate of force development by 100% in the strength-focused block, but only 3% in the power-focused block. Power responders improved rate of force development by 53% in the power-focused block and by only 11% in the strength-focused block. At pre-study, strength was greater in both responder groups compared to the non-responders; however, muscle cross-sectional area was not statistically different at pre-study ($p>0.05$) between responders and non-responders.

Hypertrophy and Strength

Cross-sectional area measurements showed hypertrophy (+12%) across all responders with no difference ($p>0.05$) between strength or power responders; however, non-responders did not experience any hypertrophy ($p>0.05$). Despite greater hypertrophy in responders, strength increased similarly in all groups (maximum strength responders: +12.38%, power responders: +15.17%, non-responders: +16.11%). The specific changes for all of these measurements are in Table 3.

Table 3 Hypertrophy, Strength, Power, and EMG Changes

	CSA			Leg press 1RM					Power 50% 1RM			
	VL muscle (CM^2)			Load (kg)			EMG (Δ%)		Peak Power (Δ%)		EMG (Δ%)	
	Pre	10wk	20wk	Pre	10wk	20wk	0-10wk	10-20wk	0-10wk	10-20wk	0-10wk	10-20wk
MS	29 ± 6	32 ± 6*	30 ± 2*	202 ± 20	218 ± 26*	227 ± 12*	15 ± 30	-1 ±19	35 ± 26	-7 ± 17	17 ± 38	6 ± 12
P	29 ± 5	32 ± 5*	31 ± 4*	178 ± 23	193 ± 25*	205 ± 18*	1 ± 40	28 ± 45	-7 ± 20	8 ± 8	-22 ± 12#	57 ± 10*
Non	23 ± 4	24 ± 9	23 ± 4	149 ± 21	168 ± 21*	173 ± 15*	15 ± 22	1 ± 28	3 ± 6	9 ± 16	-3 ± 27	8 ± 16

** = Significantly greater than pre; # = Significantly lower than pre; CSA = Cross-sectional area; 1RM = One-repetition maximum;* Δ% = Percent change

Power

Similar to strength, leg press power at 50% of 1RM increased in the strength responders during the strength block, but not in the power block, and vice versa for power responders (Table 3). Electromyography (muscle activity) of the quadriceps increased significantly only in the power responders group. Further, that increase occurred solely during the power-training block.

Hormone Responses

Testosterone, testosterone-to-cortisol ratio, and free androgen index all decreased ($p<0.05$) from pre- to post-study in both responder groups with no difference between groups ($p>0.05$) (Table 4). However, there was no change in sex hormone-binding globulin in either responder group. Both free androgen index and sex hormone-binding globulin concentrations were greater at pre-study in the responder groups versus the non-responders.

Acute Fatigue

Acute leg extension force following the strength-focused sessions decreased 12% and 11% more in the strength responders group than in the power and non-responder groups, respectively. In the power-focused sessions, acute force decreased 21% in the strength responders and 20% in the power responders, both of which were greater than a 13% decline in the non-responders.

Interpretation

The main take home from this study is that when it comes to improving rate of force development, some people benefit more from traditional strength-type training, while others will benefit from the inclusion of more power-type training. However, this study also demonstrates that even though non-responders didn't have measurable hypertrophy, they did increase strength. Let's use this interpretation to discuss the importance of individualizing training to maximize rate of force development, and how this study increases our knowledge of program design for non-responders.

First, it's wild that responses to different set, repetition, and intensity schemes can be so different across individuals.

Table 4 Hormonal Changes

		MS	P	Non
Cortisol [nmol/L]	Pre	515 ± 163	461 ± 148	622 ± 148
	10wk	539 ± 199	541 ± 115	610 ± 86
	20wk	490 ± 157	573 ± 84	562 ± 88
Total Testosterone [nmol/L]	Pre	26 ± 11	25 ± 12	25 ± 6
	10wk	21 ± 6	19 ± 6	22 ± 6
	20wk	20 ± 9	19 ± 7	19 ± 6
SHBG [nmol/L]	Pre	39 ± 11	27 ± 12	44 ± 5
	10wk	42 ± 13	29 ± 13	45 ±6
	20wk	40 ± 16	30 ±15	45 ± 1
Free Testosterone [µmol/L]	Pre	0,43 ± 0,20	0,44 ± 0,21	0,39 ± 0,10
	10wk	0,33 ± 0,11	0,33 ± 0,08	0,35 ± 0,11
	20wk	0,31 ± 0,16	0,32 ± 0,11	0,29 ± 0,11
T/C ratio	Pre	0,060 ± 0,043	0,057 ± 0,023	0,041 ± 0,013
	10wk	0,044 ± 0,022	0,037 ± 0,013	0,036 ± 0,006
	20wk	0,047 ± 0,032	0,033 ± 0,010	0,035 ± 0,010
FAI	Pre	69 ± 28	98 ± 19	56 ± 13
	10wk	51 ± 14	78 ± 34	50 ± 16
	20wk	53 ± 22	70 ± 20	43 ± 14

*Arrow and * indicates a significant decline in MS (Maximum Strength Responders) and P (Power Responders). The brackets and asterisks in the SHBG (Sex Hormone Binding Globulin) and FAI (Free Androgen Index) indicate that MS and P levels of SHBG were less than levels in Non (Non-Responders) at baseline, while for FAI, MS and P were greater than Non at baseline.*
T/C = Testosterone to Cortisol Ratio

As discussed in MASS before, we know that the rate of adaptation is highly individual, as Hubal et al 2005 reported a range in strength gains of 0-250% over 12 weeks (4), and the degree to which hypertrophy occurs might be dependent upon satellite cell and myonuclei number per fiber (5). However, just because rate of adaptation is individual doesn't necessarily mean that different styles of training – and not just different dosages of volume – are necessary to maximize individual adaptations. We must keep in mind that the disparity in the present findings was related to rate of force development and not strength and hypertrophy – those adaptations were similar between the responder groups; however, there is evidence that individual testosterone:cortisol responses to different training programs (6) and individual genetic variations (7) may affect muscle performance adaptations.

The introduction of this article illustrated how rate of force development could be a limiting factor for 1RM strength, so it is possible that some lifters might benefit from including more power-type training than others when looking to maximize strength. Importantly, the power training in this study was roughly 4 sets of 8 at 50% of 1RM, which is different from the two more prominent forms of power training. Specifically, dynamic effort training could be implemented with 40-60% of 1RM for 2-3 reps per set, while higher intensity power training would be single repetition sets with 80-90% of 1RM (8). Both of these training styles aim to stop a set before concentric velocity begins to slow. Even without velocity measures in the current study, we can be all but certain that velocity slowed, even if just a little bit, from the first to last rep in an eight-rep set; yet we can also be sure that all reps were still fast. So, a lot is left to be desired in terms of exact implementation; however, the principle of the present findings seems to be that some lifters maximize rate of force development

SOME LIFTERS MAXIMIZE RATE OF FORCE DEVELOPMENT THROUGH TYPICAL TRAINING WITHIN NORMAL REACH OF FAILURE, WHILE OTHERS MAXIMIZE RATE OF FORCE DEVELOPMENT WITH EXPLOSIVE REPS AND REMAINING FAR FROM FAILURE.

IN NON-RESPONDERS, STRENGTH GAINS STILL OCCUR AND ARE MUCH LESS AFFECTED THAN HYPERTROPHY.

through typical training within normal reach of failure, while others maximize rate of force development with explosive reps and remaining far from failure.

In terms of implementation, it seems that a "maximum-strength" responder may not need to implement power-type or speed-type training. A "power responder" also needs to perform mostly typical strength-type training to maximize strength; however, the power responder could be leaving kilos on the table by forgoing any power-type training due to not maximizing rate of force development. The remaining questions are: how much power training should a power responder do, and are some people power responders and some strength responders? I don't think we have a definitive answer to either question; however, for the former, I would currently recommend that power responders keep a 2:1 ratio of normal strength training (which I would define as ≥70% of 1RM for multiple repetitions and at ≥5 RPE) and power training. This means if training a muscle group or exercise three times per week and changing focus each session (i.e. a daily undulating periodization setup), then one of the sessions should be power-focused (preferably the one in the middle of the week). Or, if using a block periodization model, then one out of every three blocks should be power-focused. That is certainly not a magical ratio, but we can only be so specific with current evidence, so I think it's a good start.

As stated in the previous paragraph, I am not entirely sure why this phenomenon occurred, but let's speculate. Although the link between volume and hypertrophy (9) and strength (10) has been well-established, it's also clear that volume thresholds are individual; thus, for those that do better with lower relative volume, perhaps replacing a "normal" training day with a power day (similar to Table 5) is a practical way to keep volume in check. This would also avoid consistent overreaching while also attempting to maximize rate of force development, which would be beneficial at heavy loads. Therefore, if you are consistently keeping your training short of failure and performing a normal amount of sets (2-5) per exercise, and you continue to feel under-recovered with high frequencies, try replacing a typical training session with a power-type session. You

APPLICATION AND TAKEAWAYS

1. Some people improve rate of force development better through typical strength training, while others improve it better through power-type training. Further, some (non-responders) don't improve rate of force development at all.
2. Non-responders also struggle to improve hypertrophy, but in the short term, strength is not affected.
3. Those who are "maximum-strength responders" may not need to include power or speed training; however, those who are power responders should likely include power or speed training to maximize rate of force development, which can improve neural efficiency and, subsequently, 1RM.

won't lose your skill set because you have maintained frequency, and this might be a sign that you are a power responder. Besides, if you are doing exclusively strength-type training and it's not working, why not give some power training a shot? The goal is always progress, not "normal" training just for the sake of it. If the normal training isn't working, a different approach is warranted. Another way to look at it is that some power-focused training could serve as a taper or a deload week.

It's important to note that the non-responders in this study did not experience any measurable hypertrophy, yet they still improved strength to the same extent as both responder groups. In regards to hypertrophy, it still seems likely that hypertrophy would occur over the long term. Even in the short term, a different program (i.e. different dosage of volume or intensity) or other lifestyle changes (i.e. more sleep or altered nutrition) may have facilitated hypertrophy. However, with the premise that hypertrophy would at least be attenuated over the long term in this group, that factor would certainly lead to attenuated strength adaptations for non-responders over the long term.

Make no mistake: *In non-responders, strength gains still occur and are much less affected than hypertrophy*. As mentioned earlier, it may be the case that hypertrophy is limited by satellite cell and myonuclei number per myofiber (5); while this may affect strength over the long term, it is unlikely to be a major limiting factor for strength in the short term.

So, since non-responders will have minimal hypertrophic adaptations over the long term, how should this impact their training? Over the long term, I think non-responders would be well-advised to implement somewhat-frequent, heavy-ish singles on the main lifts (i.e. 7-8 RPE every few weeks, maybe even

every week). Because hypertrophy will be somewhat negligible, non-responders won't experience much strength benefit as a result of improvements in muscle cross-sectional area. Therefore, time is probably better spent working at high intensities for low volume instead of low-to-moderate intensities for high volume. In other words, non-responders should place more emphasis than responders on neural adaptations than on high-volume hypertrophy training. Lastly, the lower resting free androgen index (Table 4) in the non-responders may account in part for the non-responsive nature and could be a limiting factor for long-term strength.

Next Steps

There is a lot more to uncover in this area. It remains to be seen exactly what configuration of power training (a few configurations were mentioned earlier) is best in power responders. It also remains to be seen exactly what proportion of training should be comprised of power-type training in power responders. I'd like to see research tackle the latter question first; for example, implement a study design in which one group of power-responders performs half normal strength training and half power-type training, and another group performs power training one-third of the time.

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Study Reviewed: Homeostatic and Non-Homeostatic Appetite Control Along the Spectrum of Physical Activity Levels: An Updated Perspective. Beaulieu et al. (2017)

The Role of Physical Activity in Appetite and Weight Control

BY ERIC HELMS

Achieving weight loss is often viewed from the perspective of altering the energy-in or energy-out sides of the energy balance equation, but rarely is the effect of one on the other examined.

KEY POINTS

1. Physical activity does more than just increase total energy expenditure. When activity is low, appetite is dysregulated, resulting in excess food intake and weight gain. Higher levels of activity seem to increase appetite control.
2. The combination of being too high in body fat and also being physically inactive may further dysregulate appetite and satiety signaling, making weight loss efforts even more difficult.
3. Physical activity and exercise may only be effective to a point for the goal of weight loss. At very high levels of physical activity, additional increases may not result in an increase in total energy expenditure, but rather a downregulation of energy expended from other components of total energy expenditure and no change in net expenditure.

This narrative review on the impact of physical activity on appetite regulation covered a number of important concepts. Of principal importance, the authors point out that the view of physical activity purely as a tool to enhance the energy-out side of the energy balance equation misses the role of physical activity in appetite regulation, which can indirectly modify the energy-in side. Additionally, this review outlines the "J-shaped" relationship between physical activity and energy intake: At low levels of activity, energy intake is increased due to dysregulated appetite control (resulting in weight gain), and then only appropriately matches energy needs at higher levels of activity. This review helps to put the role of physical activity and exercise in context with the obesity epidemic, as well as addressing the emerging concept of a constrained model of energy expenditure and how this might impact the decision of how to use increases in physical activity as a tool to aid weight loss efforts.

Purpose and Research Questions

Purpose

The aim of this review was to clarify the not-well-explored or understood role of physical activity in appetite regulation and its subsequent effect on energy intake and weight gain.

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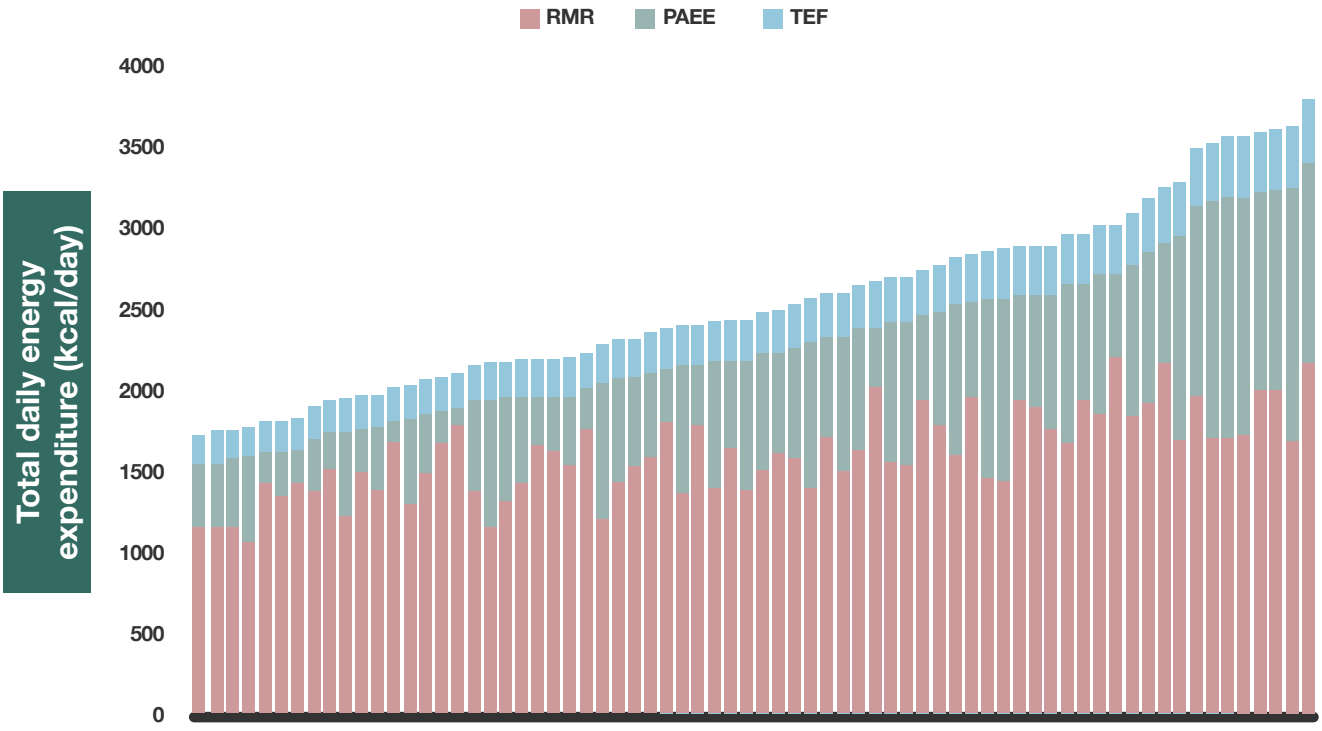
Figure 1 Individual profile of the components of TDEE



Individual profile (n = 70) of the components of TDEE including resting metabolic rate (RMR), physical activity expenditure (PAEE), and thermic effect of food (TEF). TDEE is composed primarily of RMR, followed by PAEE, which varies widely between individuals. From Beaulieu et al. [unpublished results].

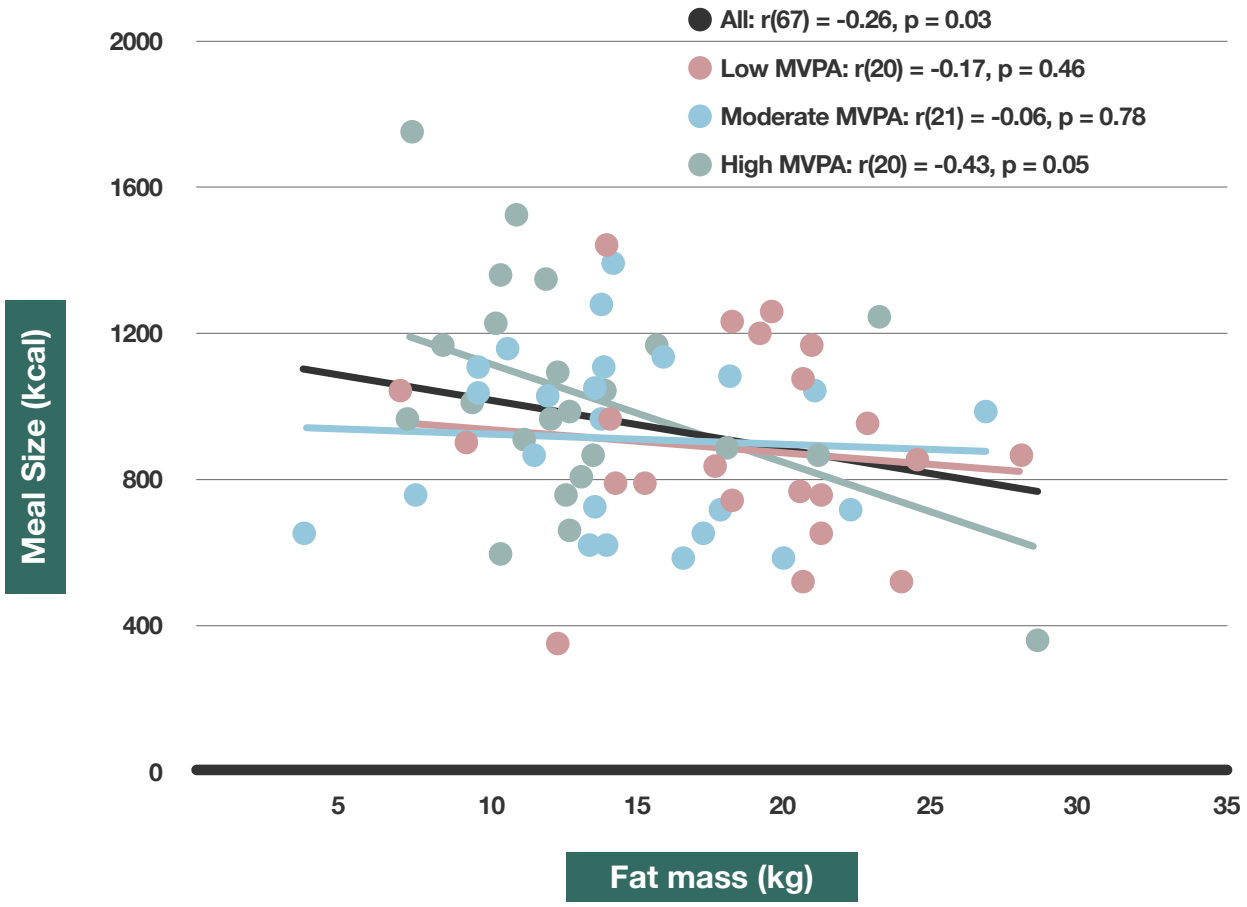
Subjects and Methods

As a narrative review with the purpose of presenting a model to better understand the role of physical activity in appetite regulation, this paper doesn't fit the typical mold of original research or systematic reviews or meta analyses. However, to give some context as to what the authors based their assertions on, the review did specifically focus on unpublished data from their own lab, investigating the relationship between physical activity and appetite regulation and synthesizing it with the existing data on this topic.

Findings

Physical activity plays a variable role in total daily energy expenditure (TDEE) depending on activity level. When defining the components of TDEE as the thermic effect of food (the energy cost of turning food into energy), physical activity, and resting metabolic rate (all the energetic requirements of your body at rest), physical activity can contribute anywhere from ~5-40% of TDEE (Figure 1). However, what is often overlooked is not simply the effect of physical activity directly on the energy-out side of the energy balance equation, but its indirect effects on energy intake

Figure 2 Relationship between fat mass and meal size

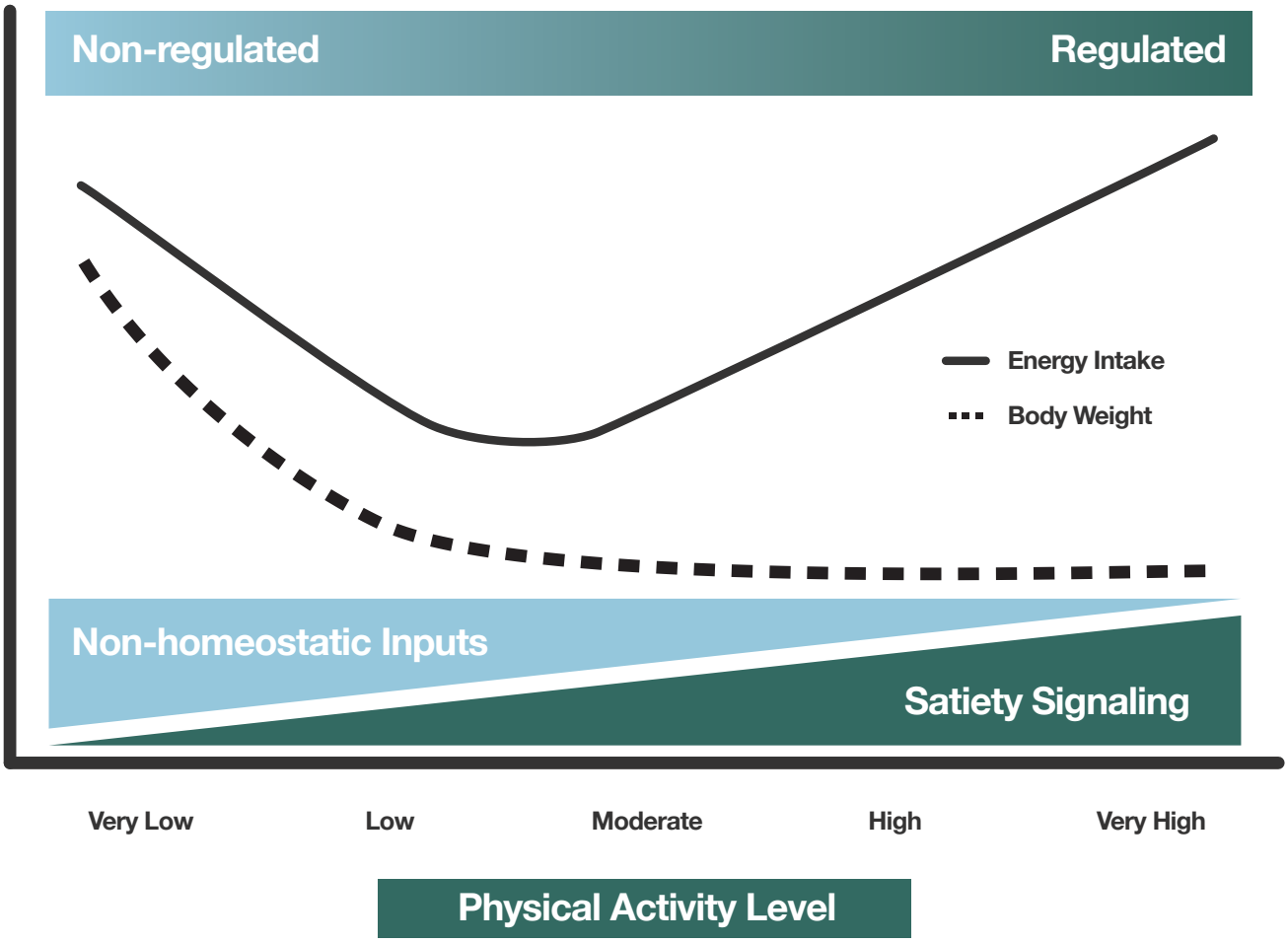


Relationship between fat mass and meal size within sex-stratified tertiles of moderate-to-vigorous PA (MVPA; n=70). The strength of the inverse association between fat mass and energy intake was found to be strongest in those with the highest levels of PA. From Beaulieu et al. [unpublished results].

through appetite and satiety regulation.

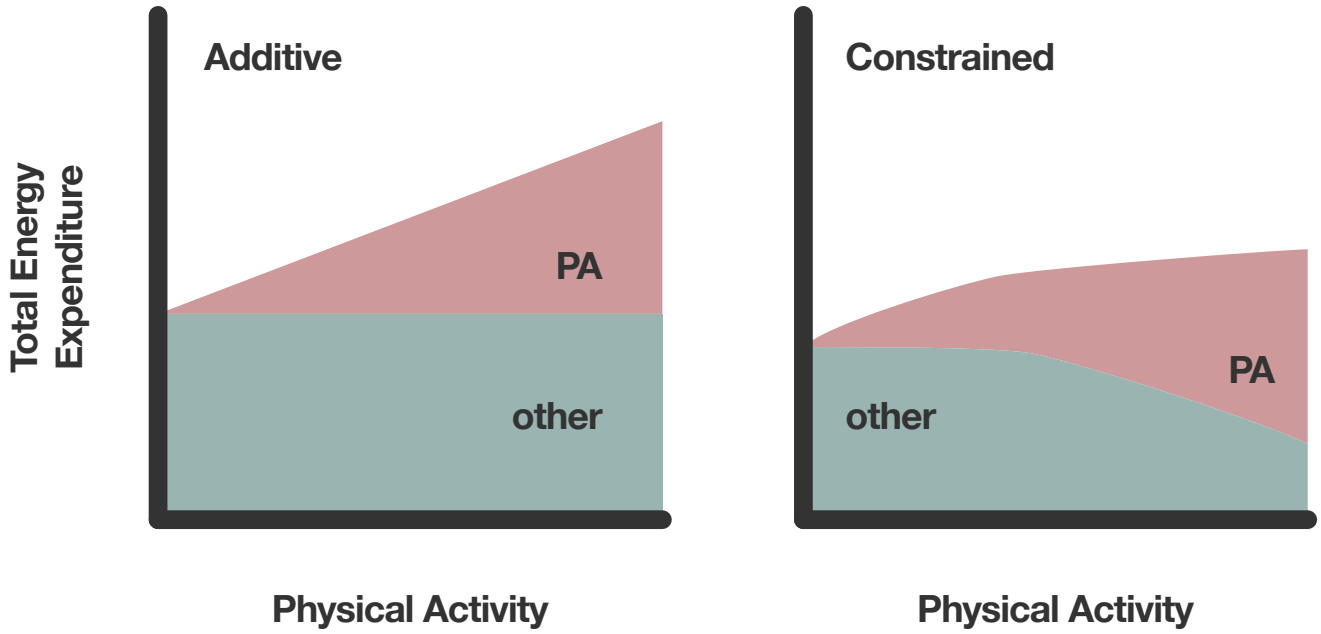
Humans have regulatory mechanisms in place to prevent under- or overconsumption of energy, thus preventing excess weight gain or loss. While these mechanisms should theoretically prevent both malnourishment and obesity, things may go awry when we stray far enough away from our ancestral levels of physical activity. Specifically, these mechanisms seem to work only when a high enough level of physical activity occurs and may be disrupted when activity levels are too low. Indeed, body fat levels share a significant, inverse association with meal size (in those with higher body fat, meal size is lower) only when physical activity levels are high. However, there is no significant inverse relationship between body fat and meal size at low or moderate levels of physical activity (Figure 2).

Figure 3 "J-shaped" Relationship of Appetite Control Along the Spectrum of Physical Activity



Increased physical activity does indeed increase the desire to eat, which is shown in studies where workers with higher physical activity levels were observed to eat more than those with lower levels (2). However, high physical activity levels also increase the satiety response to meals (3). Thus, when activity levels are higher, energy intake is more finely tuned and less likely to exceed requirements. When engaging in low levels of physical activity, homeostatic control of energy intake becomes dysregulated, and energy intake ceases to match energy expenditure due to a weaker satiety response to food, resulting in weight gain (4). Furthermore, as body fat accumulates, appetite regulation is weakened further. Indeed, in lean individuals, the insulin and leptin response to a meal acts to suppress appetite; however, body fat accumulation creates resistance to the negative feedback actions of insulin and leptin (5, 6). Unfortunately, a low

Figure 4 Constrained Versus Additive Model of Energy Expenditure



level of physical activity acts as a "triple whammy" for weight gain. It results not only in lower total energy expenditure, but also suppressed satiety after meals, and then a further suppression of satiety signals once body fat accumulates due to excess energy intake.

Effectively, there is a "J-shaped" relationship between physical activity and appetite regulation. At low levels of physical activity, energy intake is higher than requirements due to dysregulated appetite signaling. As physical activity increases, appetite regulation steadily improves, decreasing energy intake. Then, as physical activity moves to higher levels, appetite increases, resulting in an increase in energy intake. In this case, though, the coupling of energy intake and expenditure remains well-regulated, preventing excess consumption and subsequent weight gain (Figure 3).

This review provides a more hopeful outlook on the role of physical activity and exercise for weight loss than other headline-catching research outputs, which suggest exercise is ineffective for weight loss (7).

This review does come with caveats, though. For one, to get the appetite-regulating benefits of high physical activity, one truly needs to see activity as a lifestyle rather than a temporary solution to high body fat. The work used to form the conclusions of this review is based on individuals who maintain high activ-

APPLICATION AND TAKEAWAYS

1. Physical activity and exercise are useful for weight loss efforts not just because they increase energy output, but also because they may help you reduce energy intake through appetite regulation.
2. Caloric restriction alone may be difficult to adhere to for weight loss if your activity levels are low enough that you experience a dysregulated satiety response to meals.
3. Likewise, physical activity and exercise interventions on their own may only be effective to a point. Once you reach very high levels of activity, further increases may not reliably increase total energy expenditure; thus, a combination of energy restriction and physical activity may be the best approach.

ity levels, not those who simply increase activity levels for a short period, which likely doesn't exert the same effects (8).

The review also discusses some very interesting data regarding the constrained model of energy expenditure. Traditionally, the effect of physical activity on TDEE has been seen as additive: As one increases physical activity, TDEE increases in concert. However, this view has been challenged in recent years, as some hunter-gatherer societies that have much higher physical activity levels don't have the expected TDEE to match (9). This is not to say cardio or physical activity is not a useful tool for generating a caloric deficit. Rather, the traditional view that physical activity predictably increases total energy expenditure – and thus assists in weight loss efforts – may only hold true until a certain threshold of activity is reached. At that threshold, total energy expenditure is constrained, despite increases in physical activity (Figure 4).

Finally, it's important to point out that "activity" and "exercise" are not synonyms. For most people, solely hopping on the hamster wheel at the gym more often and for longer periods will not be a successful long-term strategy due to logistics, lack of enjoyment, and potentially overuse injuries (especially if the person in question is overweight). Rather, strategies should be implemented to increase activity in daily life. For example, an alarm on your phone to remind you to stand up and move around every hour at work if you have a desk job, riding a bike or walking to close locations rather than driving, using a stand-up desk, or replacing movies and TV with hikes, walks, mini-golf, bowling, or casual sports are all ways to "sneak in" physical activity while also enjoying the process.

Next Steps

To fully elucidate the relationship between physical activity and appetite regulation, further research is required to determine at what level of activity appetite becomes dysregulated, and at what point it becomes regulated again. Additionally, more research is required to understand the role of body composition and body fat status in the relationship between physical activity and appetite control. Finally, I would personally love to see the effects of a longer term, contest-prep-style study in which one group maintained a minimal amount of cardio while adjusting caloric intake as needed to achieve a very lean condition, while the other had a cap on how low they could drop their calories (say 10%), and then had to use cardio to achieve further fat loss. It would be interesting to see if the constrained model played a role such that the cardio group either had to do a disproportionate amount of cardio, or simply didn't get as lean despite a high level of activity.

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Study Reviewed: Human Skeletal Muscle Possesses an Epigenetic Memory of Hypertrophy. Seaborne et al. (2018)

The Science of Muscle Memory

BY GREG NUCKOLS

Many people have noted that, while it takes a long time to build muscle and strength initially, you can regain muscle and strength much faster after a period of detraining. A new study reveals a mechanistic reason: epigenetic memory.

KEY POINTS

1. Gene methylation patterns – which regulate gene expression – change in skeletal muscle after just one training bout, and after continued training.
2. Many of these changes are retained following a seven-week period of detraining, indicating an epigenetic memory of prior resistance training, in spite of losses in muscle mass and strength.
3. This epigenetic memory aids in subsequent retraining, which helps explain why it's hard to build muscle initially, but much easier to regain muscle after a period of detraining (i.e. "muscle memory").

At some point, I'm sure all of us have taken time away from training for some reason or another. Maybe you were rehabbing an injury, maybe your schedule got crazy, or maybe you just lost motivation. During your time away from the gym, you probably lost some muscle and strength. However, as you started retraining, you probably noticed that it didn't take very long to regain the ground you'd lost. Maybe you lost a year's worth of progress on the bench press in your time off, but you were back near your old PRs within a month or two of retraining.

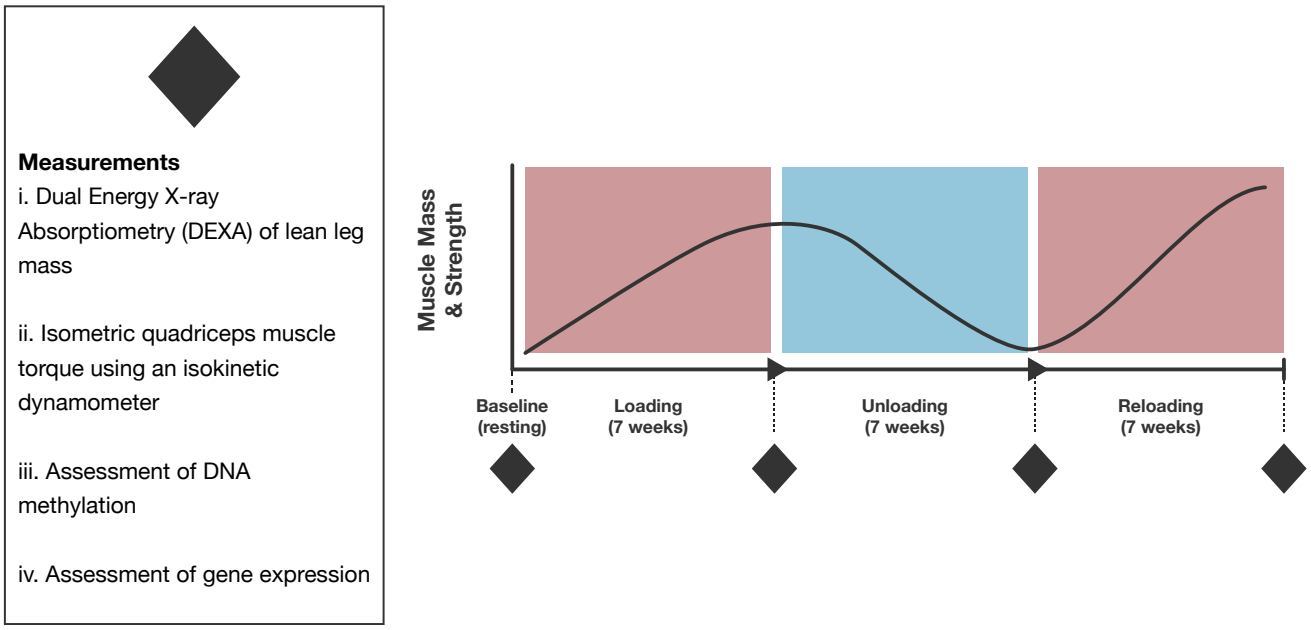
This process has been colloquially known as "muscle memory" for decades. The ability to rapidly regain strength has been at least partially understood for a long time. Namely, there's a large neural component of strength, and the motor patterns you build during your initial training get a little rusty with disuse but are re-mastered quickly with retraining. However, the mechanisms underlying the ability to rapidly regain muscle mass aren't as well-understood.

The study being reviewed is the first to directly study "muscle memory" of hypertrophy in humans. It did so by examining the epigenetic effects of training, detraining, and retraining. Epigenetics is the study of genome modifications. These modifications affect when and how genes are expressed, with alterations in gene expression ultimately affecting the proteins your body produces. Namely, this study examined gene methylation. An increase in methylation decreases gene expression, while

Listen to Greg, Eric, and Mike discuss this study and topic in the audio roundtable.

Go to playlist in Soundcloud

Figure 1 Schematic Representation of the Study Protocol



a decrease in methylation increases gene expression (I'll go into more detail in the interpretation as to what methylation is and why it's important).

This study found that when untrained people begin resistance training, genome-wide methylation decreases in skeletal muscle, is relatively unchanged after detraining (in spite of a loss in muscle mass and strength), and decreases further following retraining. Importantly, methylation of key genes implicated in hypertrophy remains suppressed throughout a seven-week detraining period, indicating an epigenetic memory of the previous resistance training, which aids in subsequent retraining.

While the actual methodology of this study may go over most MASS readers' heads (and, frankly, I had to do an inordinate amount of extra reading to really understand this study), the implications are important and easy to understand.

Purpose and Research Questions

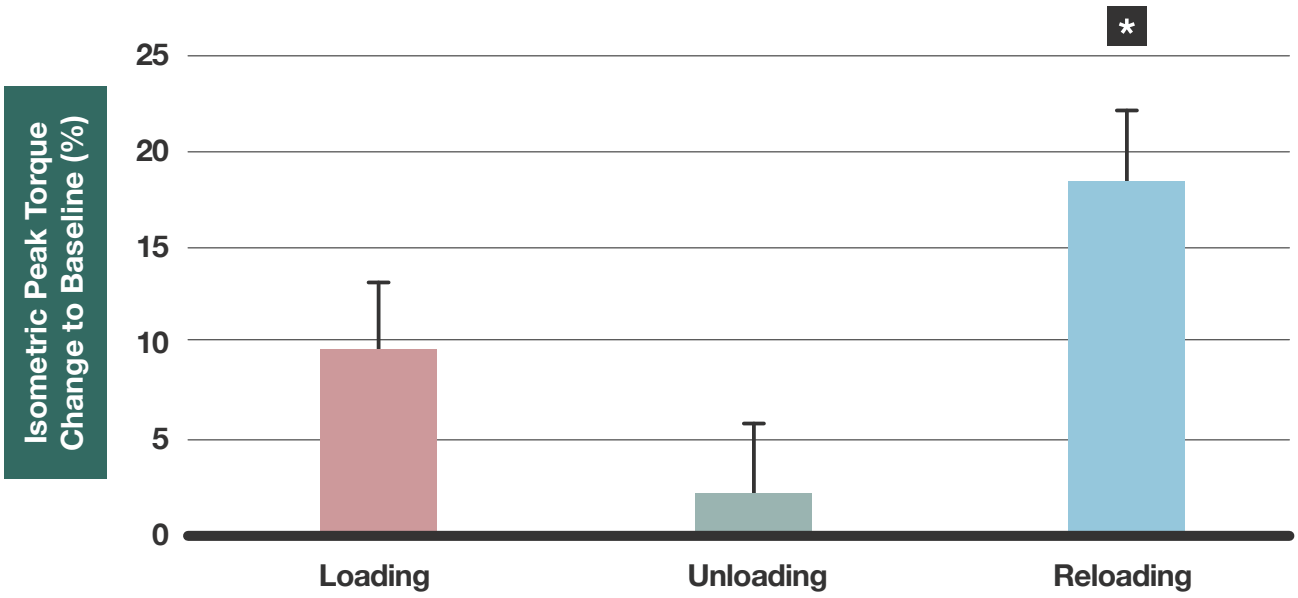
Purpose

The purpose of this study was to examine the effects of resistance training, detraining, and retraining on muscle mass, strength, and skeletal muscle gene methylation patterns.

Research Question

The primary research question was whether gene methylation patterns

Figure 2 Strength Changes



* = Significantly greater than baseline

would change with resistance training, and whether those changes would be maintained during a subsequent period of detraining in order to facilitate further increases in muscle mass and strength during retraining.

Hypotheses

It was hypothesized that methylation of genes implicated in muscle growth would decrease during an initial period of resistance training, and that those decreases in methylation would be maintained during a subsequent period of detraining.

Subjects and Methods

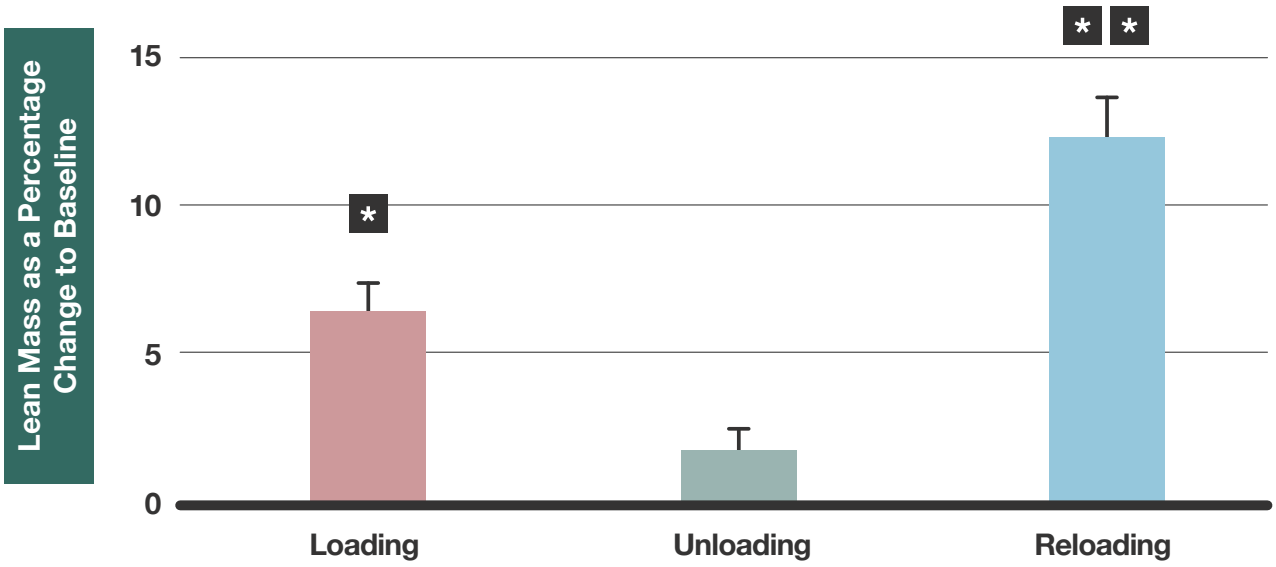
Subjects

The subjects were seven healthy, untrained males (27.6 ± 2.4 years old, 82.5 ± 6.0 kg).

Experimental Design

The study was split into three phases. The first phase involved seven weeks of progressive resistance training. The training program consisted of two days per week of lower body training (squats, leg press, knee extensions, leg curls, nordic curls, weighted lunges, and calf raises) and one day per week of upper body

Figure 3 Lean Mass Changes



** = Significantly greater than baseline; ** = Significantly greater than after the initial loading phase*

training (bench press, shoulder press, pull-downs, dumbbell rows, and cable triceps extensions). Each exercise was performed for 4 sets of 8-10 reps, and loads were increased when the participants completed 10 reps on their first 3 sets. This initial training phase was followed by seven weeks of detraining. After the detraining phase, the participants entered a seven-week retraining phase, using the same program they'd been on previously.

Measures

Strength was assessed via maximal isometric knee extension torque. Volume load was also calculated each week, and since increases in training loads were based on performance and the number of sets didn't change, volume load also serves as a proxy for whole-body strength increases. Leg lean mass was assessed via dual-energy X-ray absorptiometry (DEXA). Gene methylation and gene expression (RNA from key genes) were assessed from vastus lateralis (quadriceps) muscle biopsies.

Knee extension strength and leg lean mass were measured before the start of the initial training phase, at the end of the initial training phase, before the start of the retraining phase, and at the end of the retraining phase. Muscle biopsies were also performed at those four time

points, and after an acute lower body training bout before the start of the initial seven-week training period.

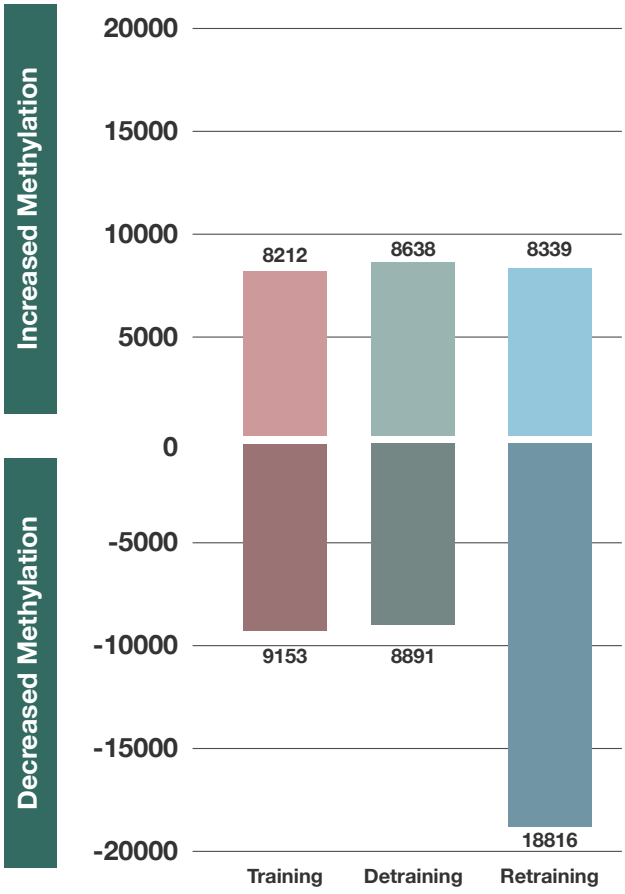
Findings

Leg lean mass increased by 6.5 ± 1.0% during the initial training phase, decreased by 4.6 ± 0.6% during detraining, and increased further (12.3 ± 1.3% above baseline, and 5.9 ± 1% above the initial training phase) during retraining. Knee extension torque followed a similar pattern, increasing by 9.3 ± 3.5% during the initial training phase, decreasing by 8.3 ± 2.8% following detraining, and increasing further (18.0 ± 3.6% above baseline) during retraining.

Genome-wide methylation patterns changed following the initial training phase, with decreased methylation at ~9,150 sites and increased methylation at ~8,200 sites. Following detraining, the same methylation pattern was largely preserved, with decreased methylation at ~8,900 sites and increased methylation at ~8,650 sites. Following retraining, the number of sites with increased methylation remained essentially unchanged (~8,350), while the number of sites with decreased methylation roughly doubled (~18,800).

The genes with methylation patterns that changed during the initial training phase and were maintained during the detraining phase ran the gamut from genes directly implicated in hypertrophy,

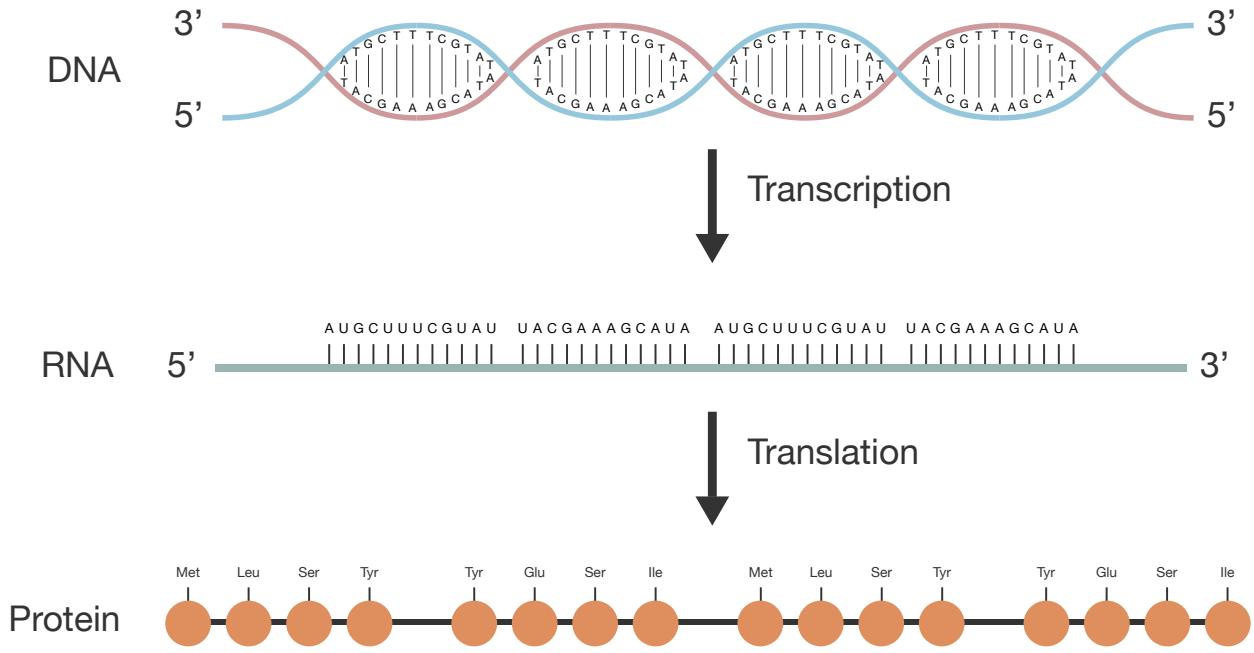
Figure 4 Number of Sites with Increased and Decreased Methylation Following to Initial Seven Weeks of Training, Detraining, and Retraining



to genes involved in cellular growth and proliferation, to genes involved in catabolism and inflammatory signaling. However, without getting too bogged down in the specifics, this nugget from the authors really sums up the epigenetics findings:

"This maintenance of hypomethylation (decreased methylation) during unloading, suggested that the muscle 'remembered' the epigenetic modifications that occurred after an earlier period of load induced muscle

Figure 5 Reading DNA to Produce RNA then Translated into Protein



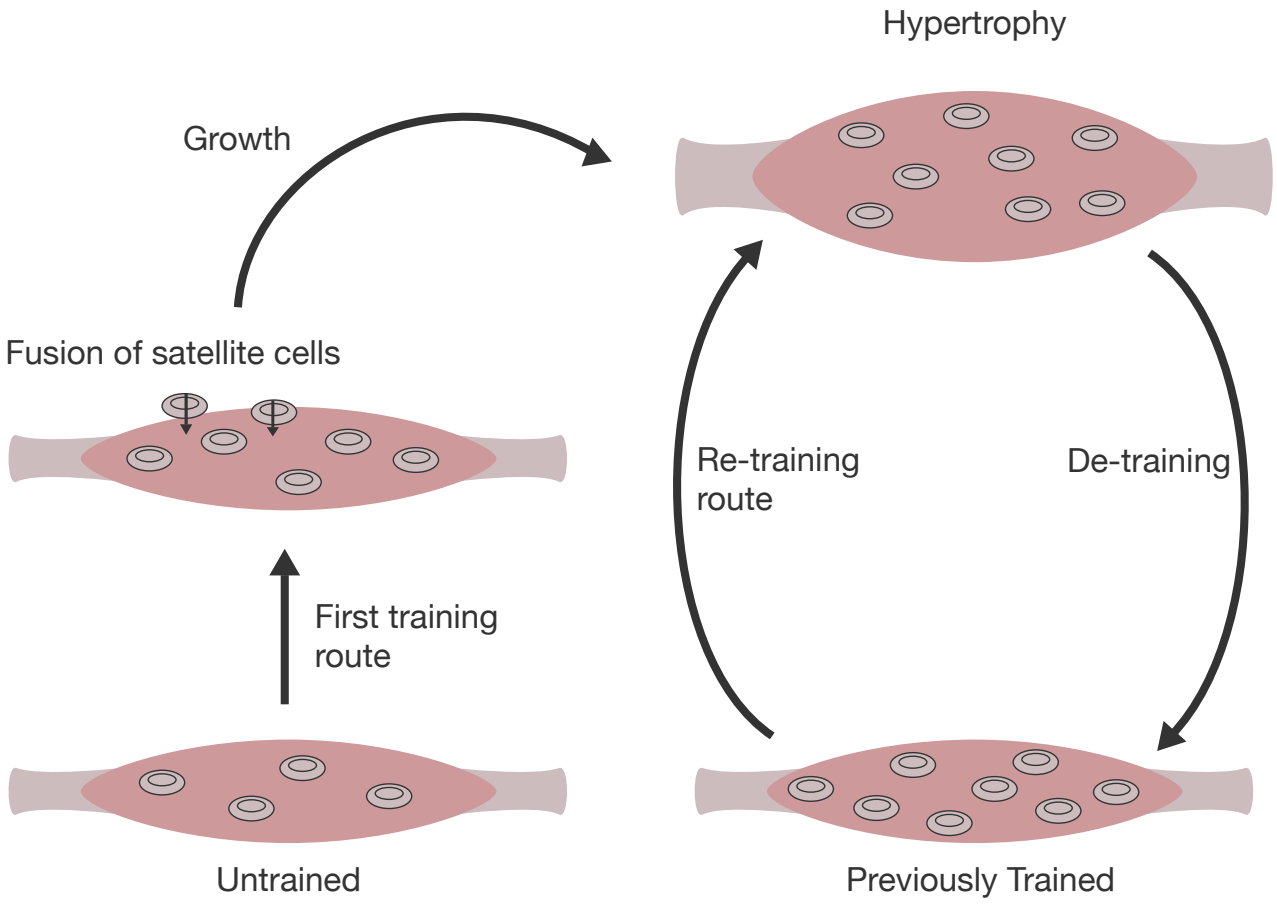
hypertrophy. As reduced DNA methylation of genes generally leads to enhanced gene expression due to the removal of methylation allowing improved access of the transcriptional machinery and RNA polymerase that enable transcription...this would be suggestive that the earlier period of hypertrophy leads to increased gene expression of this cluster of genes that is then retained during unloading to enable enhanced muscle growth in the later reloading period."

Interpretation

To understand this study, it's necessary to have at least a basic understanding of genetics. DNA contains genes, which are "read" (transcribed) to produce RNA. This RNA is then translated into proteins, and those proteins carry about essentially every interesting cellular function in your body. However, it gets a little more complicated than that, because you'd be in really bad shape if any gene in any cell could be "read" at any time.

It's very important that your DNA has ways to ensure that only the right genes get expressed. Most of your cells contain a complete copy of your DNA, so any cell could express any gene and produce any protein without mechanisms to ensure that only the correct genes were expressed. Many of these mechanisms fall under the umbrella of epigenetics: modifications to the genome that regulate which genes can be expressed in which

Figure 6 Muscle Memory Mechanism Based on Myonuclear Accumulation



cells. While some epigenetic modifications essentially function as on/off switches for genes (i.e. if they're present, a gene will simply not be expressed in a given cell) others also work by fine-tuning how much a gene is expressed. For example, in response to the exact same signal, a particular gene may be "read" 5 times to produce 5 RNA strands to code for a particular protein, or it may be "read" 50 times to produce 50 RNA strands to code for a particular protein. That's essentially what increased or decreased methylation (the epigenetic modification investigated in this study) does: An increase in methylation means a gene will be read fewer times and produce fewer RNA strands (leading to fewer proteins), while a decrease in methylation means a gene will be read more times and produce more RNA strands (leading to more proteins). The main finding of this study was that, not only do methylation patterns and gene expression change after just seven weeks of training, but that those changes are retained and "remembered" through

a period of detraining, which facilitates rapid hypertrophy and strength gains with retraining.

This study is groundbreaking because it's the first study providing a clear mechanism for "muscle memory" of hypertrophy in humans. We've had clear evidence of "muscle memory" in humans for a while now (2), but no prior studies really delved into the mechanisms. Furthermore, some rodent research (3) provides insight into another mechanism of muscle memory – myonuclei are gained with hypertrophy, retained with detraining, and then aid in subsequent muscle growth with retraining – but that research has only been partially replicated in humans.

It's worth noting that this study doesn't tell us how long these epigenetic changes last. The detraining period in this study was seven weeks, but it's likely that these epigenetic changes persist for much longer. This was the first study showing an epigenetic "muscle memory" of hypertrophy, but other research (4) has shown that epigenetic modifications in skeletal muscle during utero or early childhood can persist throughout an entire lifespan. Thus, it's possible that these epigenetic modifications can last for years to facilitate enhanced responsiveness to retraining after very long layoffs (i.e. if you used to lift in high school, and then started training again in your 40s). Similarly, it's possible that some of these modifications could occur during growth and development if you did a lot of physically strenuous activity during childhood, thus facilitating enhanced responsiveness to strength training later in life. However, these two possibilities are speculative.

WHEN EVALUATING WHAT STYLES OF TRAINING WORK BEST FOR YOU, PAY CLOSE ATTENTION TO THE PROGRAMS YOU USED WHEN YOU WERE REACHING PREVIOUSLY UNCHARTED TERRITORY.

There are two clear implications of this study. One relates to the way you should view your training looking forward, and one relates to the way you should evaluate your training looking backward. Looking forward, this study should give you more confidence in taking time off of hard training when dealing with injuries. A lot of lifters make the mistake of trying to train through injuries because they fear that dialing back their training or taking time off will be a major setback, costing them months of progress.

APPLICATION AND TAKEAWAYS

1. You retain an epigenetic muscle memory of prior training after at least seven weeks of detraining, which aids in regaining muscle and strength during the retraining process.
2. With this in mind, be careful that you don't falsely assume a particular training program is super effective for you just because it helped you quickly regain lost ground. You were epigenetically primed to regain lost ground in the first place, regardless of training program.
3. Similarly, don't freak out if you need to take it easy in the gym for a few weeks, or even take some time away from training to deal with injuries. Whatever gains you lose during this process will come back quickly once you can start training hard again.

This study gives us strong evidence that your muscles "remember" prior training for, at minimum, seven weeks, helping you regain any lost ground quite quickly.

Finally, when looking back at old training journals to evaluate the effectiveness of past programs, keep in mind that results during periods when you were regaining lost ground probably aren't representative of a "normal" training response. After a layoff, it's possible (likely, even) that just about any program could get you back near your old levels of strength and muscularity. Don't get married to a particular style of training just because it worked super well when you were coming back from a layoff. Rather, when evaluating what styles of training work best for you, pay closer attention to the programs you used when you were reaching previously uncharted territory.

Next Steps

In terms of elucidating mechanisms of "muscle memory" in humans, I'd love for someone to repeat the myonuclei study in humans. It would probably need to be modified to more closely resemble the design of the present study, rather than initially inducing hypertrophy with steroids (as was done in the prior rodent research), but that should be a feasible design. Furthermore, I'm intrigued by the additional decreases in methylation during the retraining phase in this study; since decreases in methylation promote gene expression, it seems that the participants in this study were more epigenetically "primed" to respond to training after seven weeks of retraining than they were after the initial seven weeks of training. I've always been curious whether time away from training resensitizes people to the anabolic stimuli of lifting,

and I'd be interested in seeing a study in well-trained lifters who report being at a plateau that's designed to answer that question. For example, one group could train for 12 weeks consecutively, while another group trained for 3 weeks, took 3 weeks off, and then trained for 6 weeks, with a comparison of strength gains and hypertrophy after the 12 weeks.

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VIDEO: Program Troubleshooting

BY MICHAEL C. ZOURDOS

It's great to program around scientific principles, and we always should. However, life gets in the way, and we need to troubleshoot for feasibility. This video examines how to anticipate things like time constraints and travel to make adjustments, yet still adhere to scientific guidelines.

Click to watch Michael's presentation.

PROGRAM TROUBLESHOOTING

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VIDEO: Sustainable Motivation for Sport and Fitness

BY ERIC HELMS

It's natural to focus on the next competition, PR, a specific body fat percentage, getting your pro card, or qualifying for worlds…but, what if I told you that might actually be harming your chances of achieving those goals and staying in your sport long-term?

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