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WITH YOU FROM BENCH TO BEDSIDE

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The Effect of Training Frequency on Hypertrophy

How many times per week should a muscle be trained to maximize muscle hypertrophy? A systematic review and meta analysis of studies examining the effects of resistance training frequency by Schaoenfeld et al. 2018

Key Points:

1. This is a meta-analysis of 25 studies including 800 subjects that looked at how training frequency altered muscle hypertrophy outcomes when volume was held constant.
2. In every analysis performed by the authors, no significant improvement was seen in higher frequency groups compared with lower frequency groups.
3. This paper serves as to update the previous meta-analysis published by the authors in 2016.

Introduction

This month's paper is meta-analysis published December of 2018 by Schoenfeld et al. looking at the effect of training frequency — i.e., the number of training sessions per week for a particular muscle group — on muscular hypertrophy outcomes.

Training frequency's effect on outcomes like strength performance and hypertrophy can be tricky to determine because its inherent relationship with weekly training volume, which is the product of sets, reps, and frequency.

For example, let's say we have a lifter using the following programming for their squat training:

- Day 1
  - Squat x 5 x 5 @ RPE 8
  - Volume= 25 reps
- Day 2
  - 2ct paused squat x 4 x 3 @ RPE 9
  - Volume= 12 reps
- Day 3
  - Tempo squat x 8 x 3 @ RPE 8
  - Volume=24 reps

The weekly volume here is 61 reps and the squat training frequency is three per week. If we increase the training frequency by adding another day of squats for 3 reps x 3 sets @ RPE 9, we increased **both** the frequency (now four times per week) and the training volume (now 70 reps).

Alternatively, we could add another day of squats but keep the volume the same:

- Day 1
  - Squat x 5 x 4 @ RPE 8
  - Volume= 20 reps
- Day 2
  - 2ct paused squat x 4 x 2 @ RPE 9
  - Volume= 8 reps
- Day 3
  - Tempo squat x 8 x 3 @ RPE 8
  - Volume=24 reps
- Day 4
  - Squat, no belt x 3 x3
  - Volume= 9 reps

In this example the volume is the **same** at 61 reps, but the frequency is **different** at four times per week. From a hypertrophy perspective, we'd expect that increasing the training frequency of a muscle group would tend to produce more episodes of muscle protein synthesis, thus producing more muscle mass gain. Consider the following two points:

Untrained individuals typically have a higher-than-normal muscle protein synthesis rate for ~48 hours after training. However, the duration of this muscle protein synthetic response decreases the more trained someone is.

Thus, we might hypothesize that by increasing training frequency of a muscle group, we could combat the shortened muscle protein synthesis elevation that is seen in trained individuals. This idea gains some support we look at a meta-analysis published two years prior in 2016 (link), which suggested that increasing frequency from once to twice per week while keeping total weekly volume the same improved hypertrophy outcomes.

There were a few problems with this initial review however, as only seven studies with 200 total subjects met the inclusion criteria and additionally, none of the included studies were able to show whether even higher training frequencies, such as three or four times per week, were better than two times per week.

Subjects and Methods

In the two years between the initial meta-analysis and this most recent paper, an additional 18 studies with 600 more subjects were published. Upon subsequent review, we now have a grand total of 25 studies (800 total subjects) that met inclusion criteria for the current meta-analysis.

Impressively, the new studies that were included measured hypertrophy outcomes directly by using MRI, CT, or ultrasound imaging, which all measure muscle mass more accurately compared to indirect measurements like bioelectrical impedance (BIA), DEXA, or other methods.

Findings

When analyzing the studies, the authors calculated effect sizes - a measure of the *magnitude* of change - in this case training frequency differences. Typically small, medium, and large effect sizes have values of 0.1, 0.3, and 0.5+, respectively.

In the previous meta-analysis the authors found the effect size difference between low and high frequency training to be 0.19 (indicating a modest effect),which translated to 3% greater average muscle growth in the higher frequency training groups. So, what did they find this time around?

In all sub-group analyses which included volume-equated studies on 1) hypertrophy in the upper body, 2) lower body, 3) total body hypertrophy for all subjects, and 4) total body hypertrophy in trained subjects, the authors found no difference between low and high frequency training. Most of the effect sizes where between 0.01 to 0.10. Among untrained subjects under volume-equated training regimens, there was insufficient data to draw any conclusions. The authors had this to say:

> *"Primary results showed that the number of times a muscle group is trained on a weekly basis has a negligible impact on hypertrophic outcomes on a volume-equated basis. In general, these results were constant even when studies were subanalyzed to account for the potential influence of different covariates. Alternatively, there was an effect of frequency when training volume was not equated between conditions, although the magnitude of the effect was modest."*

When looking at the raw data I find myself agreeing with the authors repeatedly, as it is repeatedly shown that more frequency in and of itself does not appear to drive greater hypertrophy responses when volume is kept constant. In studies where volume goes up along with frequency there does seem to be a dose-dependent response in hypertrophy, but we already knew that.

Why does this article matter?

Overall, it appears that increasing training frequency alone is unlikely to significantly change hypertrophy outcomes when volume is held constant, though strength outcomes may be improved with higher training frequencies. We also know that there are substantial inter-individual differences in responses to training, which helps explain why some may achieve better results with higher or lower frequencies (and volumes). We can use different training frequencies in order to change the weekly training volume to tailor the programming based on the athlete, their response to training, and their goals.

I think the strengths of this study compared to the previous meta-analysis include the increased size, the direct measure of hypertrophy as an inclusion criteria, and using consistent statistical analyses for comparative purposes that- in sum- suggest that increasing training frequency does not improve hypertrophy independent of training volume. The authors had this to say:

> *"The plethora of research that has been carried out on the topic since the publication of that meta-analysis now supplies data from 25 studies encompassing over 800 subjects for the present analysis, providing strong confidence in the veracity of our findings. The large number of studies meeting inclusion also allowed for subgroup analysis of covariates that provided novel insights into the nuances of the topic. This finding would seem to support the concept that frequency can be used as a tool to increase resistance training volume, which has been shown to increase muscle size in a dose-response manner."*

One thing that isn't addressed by this study is the effect of frequency on strength outcomes like a 1 rep-max (1RM) performance or similar. Previous research tends to suggest that increasing frequency up to 4x/wk for upper body exercises like the bench press and press tends to improve strength performance a few percent per week, but some of those studies aren't comparing volume-equated training and thus, the volume differences may be confounding the findings.

Still, I think there may be some benefit to increasing training frequency for improving strength outcomes. Briefly, strength improvements stem from improvements in muscle coordination, voluntary contraction force, changing tensile properties of soft tissue (stiffness of tendons, for example), etc. Hypertrophy improves the contractile force potential by increasing the muscular size, but the other aspects of improved strength performance are independent of hypertrophy and may potentially benefit from increased training frequencies, as reflected by previous studies. More research on this particular topic is needed.

In the meantime, however, I think it's reasonable to suggest that increasing training frequency is another tool to increase training volume, and that the increased training volume is likely the more important variable in training outcomes, especially hypertrophy. Individuals respond to training individually, thus it is no surprise that different people may respond to a given training frequency (and volume) differently. Ray Williams reportedly squats one time per week, whereas guys like Bryce Lewis squat three to four times a week, each choosing to solve the same problem (how to increase their squat) a bit differently.

Thanks for reading, everyone. See you next month!

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How Learning One's Genetic Risk Changes Physiology

Learning one's genetic risk changes physiology independent of actual genetic risk by Turnwald et al. 2018.

Key Points:

1. Being told information about genetic predispositions appears to influence physiology positively or negatively in an expectancy-based, self-fulfilling manner.
2. In some situations, perceptions of genetic risk -- even if inaccurate -- had greater impacts on physiological outcomes than actual genetic risk.
3. Given that results of modern laboratory testing can have such significant impacts on self-rated perceptions (and thus outcomes), we should consider the under-appreciated potential for harm before ordering such lab testing. This is especially true of the unvalidated or unreliable tests often promoted among alternative healthcare practitioners.

Introduction:

The allure of biomedical technology is near universal. We now commonly see people using activity trackers, sleep trackers, and heart rate monitors, as well as checking their own blood tests and even genetic testing through direct-to-consumer services. In fact, in 2017 more adults purchased direct-to-consumer DNA analyses than in *all prior years combined*. But as with any other intervention, we must weigh the potential benefits against the harms.

Wait … harms? It's just data. Knowledge is power, after all, right? What's the harm in knowing more about yourself? And *why won't my doctor order this test that I want*? A recent study by Turnwald et al. helps illuminate just a few of the issues associated with this approach [1].

Subjects & Methods:

Researchers performed two similarly-designed experiments as part of this paper.

Experiment 1: 116 healthy adults aged 18-50, 42% male, 51% white, mean age 24.7, mean BMI 23.3.

Experiment 2: 107 healthy adults aged 18-50, 32% male, 59% white, mean age 26, mean BMI 23.8.

In experiment 1, subjects had their DNA analyzed for specific variants of a particular gene (**CREB1**). The "high-risk" variant of this gene is associated with **poorer aerobic exercise capacity, higher body temperature during aerobic exercise, and a decreased adaptive response to aerobic exercise**. In contrast, the "protective" variant of this gene is associated with better aerobic capacity, lower body temperature during exercise, and a better adaptive response to aerobic exercise.

In experiment 2, subjects were similarly tested for variants of **FTO**, which is one of the most highly associated genetic risk factors for obesity. The "high-risk" variant is associated with l**ower reported satiety ("fullness") after a meal, stronger appetite and reward responses to images of food, and lower "physiologic" satiety** (i.e., hormones like GLP-1), whereas the "protective" variant is associated with *greater* reported satiety, decreased appetite/reward responses, and higher satiety hormone responses after a meal.

Both groups then underwent baseline testing *without* any knowledge of their genetic status. The first group underwent a maximal treadmill exercise test, with measurements of several **physiologic parameters** (e.g., oxygen/CO2 exchange and ventilation rate), **running endurance, and their subjective assessment of difficulty** (RPE). The second group consumed a standard 8-ounce, 480 kcal nutrition shake, followed by measurements of **subjective satiety** as well as **blood measurements of acyl-ghrelin and GLP-1** (hormones associated with hunger and satiety, respectively).

One week later, subjects were brought back to the lab to undergo repeat testing, performed by experimenters who were blinded to the subjects' baseline test performance as well as their actual genotypes. However, **before repeat testing, they were randomized to be told that they either had the high-risk or the protective genotype, regardless of their actual genotype.** This means that they were essentially given information about their genotype at random, and were subsequently confirmed to understand the significance of their result. The authors write:

"To convey this information, each participant received a genetic test report detailing his/her risk level and a pamphlet (constructed from published scientific and popular press articles about the CREB1 or FTO gene) explaining the gene's effects on subjective experience, behaviour and physiology, and the scientific evidence for its link to obesity through exercise capacity (CREB1) or satiety (FTO) ... The genetic test reports and pamphlets emphasized that the CREB1 and FTO genes were predictive of exercise- and satiety-related outcomes, respectively."

This is material that would be analogous to what an individual might search out and read on the internet about the results of their direct-to-consumer test results, in the absence of clinician interpretation.

(Results section on next page)

Results:

	Actual Genotype Effect size (*d*)	Perceived Genotype Effect Size (*d*)
Experiment 1		
Max CO2:O2 exchange rate	**−0.14**	**0.50**
Max ventilatory flow rate	0.12	0.08
Running endurance	0.41	0.16
Perceived exertion	0.40	0.29
Perceived heat	**0.14**	**0.34**
Experiment 2		
GLP-1 (physiological satiety)	**0.09**	**0.66**
Acyl-ghrelin (physiological hunger)	−0.21	0.25
Perceived satiety (fullness)	**−0.07**	**0.46**

The table lists experimental results in terms of *effect size,* which is a method reporting the *magnitude* of difference, either between groups or within a group across the study. This helps to give us a more useful idea of "significance" than the standard p- value calculation for statistical significance (to learn more about effect sizes, see here). In the table, "effect sizes with positive values indicate that the effects were in the hypothesized direction, except for acyl-ghrelin (for which negative values represent the hypothesized direction)." Bolded values reflect where the perceived *genotype effect* was greater than the *actual genotype* effect.

Discussion&Takeaways

Notably, the individuals informed they had the "high risk" genotype reached a significantly *lower capacity for oxygen and carbon dioxide exchange compared to their own baseline test,* as well as a *decrease in maximum ventilatory flow rate and endurance*. In contrast, the group told they had the "protective" genotype ran longer before reporting the test felt "hard". In the second experiment, individuals told they had the "protective" genotype had a *2.5-times greater GLP-1 (satiety hormone) response compared to their own baseline tests, as well as a 1.4-times greater subjective satiety (sense of "fullness") compared to their baseline, regardless of their actual genotype.*

This paper shows how simply learning of one's genetic predisposition -- in some cases *regardless of actual underlying genetics* -- can have substantial consequences on outcomes.

The mechanisms by which these effects occur are unclear, but similar findings have been reported in other contexts as well.

For example, older adults who received information indicating a high risk for developing Alzheimer's disease subsequently performed worse on memory testing and had worse self-reported memory compared to individuals at also high risk but who were unaware of their risk status [2]. We have also discussed this concept with respect to sleep trackers. Additionally, many lifters approach us interested in measuring blood testosterone (or other hormone) levels, despite no significant clinical findings or symptoms suggestive of hypogonadism or other pathology.

In light of these research findings, consider the potential consequences of receiving information that your blood testosterone level is on the "low end of the normal range," or is otherwise l*ower than you expect / want* it to be. Anecdotally, we have observed these individuals to often subsequently report worse training performance, worse subjective perception of recovery, and increasing dissatisfaction with training outcomes, even though there is no clinical indication of hypogonadism (and indeed, there is evidence that such levels have relatively little impact on training adaptations [3]). Similarly, consider the potential implications of receiving information that you are genetically predisposed to obesity, specifically the potential for unnecessarily removing the locus of control from the patient.

In the same way that more expensive, high-tech interventions (like surgery) often provide greater placebo effects in the context of treating pain, it appears that high-tech tools (like genetic testing) can similarly provide large placebo and ***nocebo effects*** as well. And given that clinical testing is an imperfect science with risk for false positives and false negatives, individuals should be very careful in pushing for, or ordering for themselves, tests which may not be clinically necessary in order to guide treatment decisions.

Simplyput:sometimesyoudon't *"justwanttoknow"*.

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The Clinician's Narrative - Why Words Matter

Individuals' explanations for their persistent or recurrent low back pain: a cross-sectional survey by Setchell et al. 2017.

Key Points:

1. This is a cross-sectional qualitative analysis study that assessed patterns of thinking ("discourses") patients stated caused their recurrent low back pain and the origin of their narrative (healthcare provider, internet, family, friends, or other).
2. Of the 130 participants, 116 (89%) stated their narrative came from a healthcare provider.
3. The predominant discourse given was 'body as machine" and was found in almost all responses.

Introduction:

A primary outcome measurement clinicians are tasked with tracking and influencing is the patient's subjective report of pain. Persistent or chronic pain has become a major global health issue. In the United States alone, it has been estimated between the healthcare costs attributable to pain and the annual costs of pain associated with lower work productivity totals $560-635 billion.[Gaskin 2012] Globally, low back pain is the leading cause of years lived with disability (YLLD) according to the Global Burden of Disease 2016.

Historically, persistent pain has been viewed through a biomedical lens, which takes a reductionist approach to the problem. This approach assumes the presence of biomechanical / structural "abnormalities" as correlates to patient symptoms. When treatment is anchored to this biomedical approach, therapeutic modalities are -- seemingly logically -- selected based on the ability to correct these perceived abnormalities.[Durnez 2017] Unfortunately, this approach has fallen drastically short when it comes to improving outcomes for patients with pain.

The recent paradigm shift towards a multi-factorial approach to pain treatment has involved the application of the biopsychosocoial (BPS) model, which postulates that pain perception can be influenced by biological, psychological, and sociological factors [Gatchel 2007]. Mounting research evidence is demonstrating the influence of clinician language on patient perceptions and understanding of pain, and thus their outcomes.[Nickel 2017], [Barsky 2017]. In order to effectively change patient beliefs and thus behavior, we require a basic understanding of the person's thoughts on the matter and the origins of these beliefs.

Subjects & Methods:

Purpose:

The primary purpose of the article reviewed was to analyze the discourse of patients dealing with recurrent or persistent low back pain. Discourse, in this sense, equated to the participants' understanding, beliefs, and written communication of their low back pain. A secondary focus of the article was to assess the origin of participants' discourse(s).

Subjects:

This article is a cross-sectional study analyzing qualitative data collected from an online survey of 130 participants. The majority of participants were females from Australia who reported daily pain (see table 1). The survey assessed participants understanding of their persistent low back pain via 2 questions.

Methods:

Participants were asked via open-ended question:

1. What is your understanding of why your low back pain is persisting or recurring?

Followed by a multiple-choice question:

2. Where does this understanding come from?

Participants received the following answer options:

- Health care provider
- Internet
- Family
- Friends
- Other (able to type answer)

A multidisciplinary (physiotherapy, psychology, medical, and social work) six-member team was utilized to assess and categorize the responses of the participants.

Findings

Origin of beliefs

The majority of participants (n = 116, 89%) identified healthcare professionals as the source of their beliefs regarding persistent and recurrent low back pain. Additionally, participants identified the internet as the second source of their understanding about low back pain (n = 31, 24%). See table 3.

Discourses Identified

The authors identified four discourses from the answers to question 1 (Table 2):

1. **Body as machine** (structuralist) = *"Like a machine, the body is considered to be able to break and can sometimes be repaired. LBP persists because something is physically defective."*

2. **Low Back Pain as permanent/immutable** (structuralist) = *"Related to the first discourse, LBP is conceptualised as a static or fixed entity that once 'broken', it cannot be 'fixed'. LBP is not dynamic or fluid but unchangeable and permanent."*

3. **Low Back Pain is complex** (multifactorial) = *"This is a counter discourse to the first two. Multiple factors can contribute to the persistence of LBP – not only biomechanical or anatomical but also possibly psychosocial or cultural factors. There is no simple explanation for ongoing LBP."*

4. **Low Back Pain is very negative** (catastrophizing) = *"LBP is conceptualised as abnormal, catastrophic, or very negative experience. LBP should be avoided and/or has a large effect on life."*

The first discourse, "B*ody as machine,*" was present in almost all participant responses. These participants viewed their body as having something mechanically wrong leading to their persistent/recurrent low back pain.

Here are two participant responses:

Participant 3:

> *"Degeneration of the integrity of my tendons and ligaments from faulty collagen due to Ehlers-Danlos Syndrome causing instability in my spine (and other joints) resulting in herniation of spinal discs (currently 3 cervical, 1 thoracic and 2 lumbar) and degenerative disc disease at L5/S1. Also sacroiliac joint dysfunction, hip dysplasia and instability has a correlating impact to my back issues."*

Participant 59:

> *"My motor control has suffered due to chronic low back pain initially caused by an injury and then perpetuated by degeneration in the joints. Even though there is no acute injury any more (arthritis is still there), my motor patterns are inefficient and I recruit larger muscles to stabilise my back due to pain inhibition. This means sometimes I do movements that are actually more forceful that needed and increase joint loading at the degenerating level, which is what causes a flare up."*

The authors found many participant responses included biomedical lexicon such as joint/muscle/nerve injury and disease, postural issues, and inflammatory conditions. Some examples included: "*fusion surgery leading to sacroiliac joint problems*", "*my L4 and L5 are rubbing together*", "*spinal damage caused by arthritis*", and "*spondylolisthesis L5S1 with pars defect*".

In conjunction with the "*body as machine*" narrative, many participant responses supported the second discourse that "*low back pain is permanent/immutable*".

Participants who cited a structuralist issue as the origin of their low back pain also believed the issue to be permanent. Participant responses included: "*Damage done earlier in life", and "Injury from high school...*"

The word degeneration was also frequently cited by participants to demonstrate an ongoing damaging process: "*Now, it has become a matter of degeneration to the structure due to age and injury*". "*arthritic changes in the bones*" "s*evere multi-level stenosis*" "*My understanding is that because of my scoliosis I may always have lower back pain – and this could increase as I get older.*"

Much less prevalent was discourse 3, which opposes narratives to discourses 1 and 2. The authors stated participants categorized into this discourse believed "*factors other than biomechanics and disease processes can contribute to LBP's recurrence or persistence.*"

Examples of participant responses:

"*…in part my dependence on medication*", "*Pain patterns in brain as well as muscles that engage to 'protect' me when they don't need to.*"

Participant 50:

"*I have a severe burst dispersion fracture of L1 with up to 75% of the body of L1 crushed and dissolved. I have no neurological impairment and the fracture was stabilised without surgery. In 2013 I had a 20-year MRI and consulted a private pain specialist (also ortho surgeon) and he confirmed that the root cause is mechanical. My background pain was very high for approx 1 year (mid 2012-13) during a suicidal depression period. I have several month long bouts of depression every 3-5 years but the 2012 episode was worse than others. This fed the pain which fed the depression and I started hating my pain for the first time in 22 years. Although it can be tiring and exasperating at times, I had never hated the pain or wished it gone. Interestingly, during a few months of intense psychological treatment sessions, I had a week and a half long bad pain episode but it wasn't until the 4th day that I realised that my attitude to the pain and my 'automatic responses' to it had reverted back to my usual acceptance so I saw that as a step forward. The year highlighted again the direct correlation of mood to pain.*"

The final discourse, "*LBP is very negative*", was prevalent in many participant responses. Example of responses classified for discourse 4 include:

"*Severe spinal stenosis and an awful scoliosis*"

"*severe sciatica...pain never goes away*"

"*I have worn out, my L5/S1 to the point, it can't take anything else.*"

"*My understanding is that because of my scoliosis I may always have lower back pain – and this could increase as I get older.*"

Why does this article matter?

Based on this article, it appears that people's beliefs about their persistent low back pain primarily originated from healthcare providers and were anchored to a biomedical model. The most prevalent discourse was based on the idea our bodies are "machines", capable of being broken and requiring fixing. This biomechanical approach predisposes the belief that if treatment is not sought to correct the perceived issue, then pain and damage will persist or worsen.

Studies such as this one demonstrate the importance of disseminating the most evidentially-supported information we currently have available as clinicians. Additionally, this study sheds light on how our narratives influence the beliefs of our patients and may perpetuate an external locus of control affecting pain management. Locus of control can be defined as a person's viewpoint about the level of control they have over events in their lives. A person with an internal locus of control typically believes events in their life are related to their personal decisions, while an external locus of control attributes outside influences and factors other than the person as major contributors to life events.

A narrative rooted in biomechanical lexicon may instill the belief something is wrong with the patient which requires correcting. Although biology will remain a correlate, research continues to demonstrate psychological and sociological factors may play an equally important, if not greater, role in persistent pain. As clinicians, research like this will continue to challenge our beliefs about pathologies and biological issues necessitating care or perpetuating a therapeutic illusion.[Thomas KB 1978]

Our narratives are long-lasting with patients and have the ability to influence behavior. Patients unnecessarily worried about or catastrophizing over a biomedical issue may develop kinesiophobia, which will perpetuate disability and seeking of treatment. However, our narratives can also positively influence behavior. The framing of low back pain can be altered to one of acceptance of a typically normal occurrence, as demonstrated by the mounting evidence of asymptomatic imaging findings of the spine.[Romeo 2018] [Brinjikji 2015]

Patient interaction can be utilized to reassure and instill the belief the issue will likely improve while supporting patient self-care and building self-efficacy in their own ability to manage pain. The authors do caution they are not denying the potential biomedical correlates of low back pain. Rather, they intended to highlight such discourse is not applicable to everyone and may instill false beliefs, thereby perpetuating fear-avoidance behaviors.

The authors admit that initial causes of low back pain may be related to biomedical issues. However, little evidence exists supporting such a belief for persistent or recurrent low back pain, and instead demonstrates a biopsychosocial and multi-factorial approach.

In conclusion, this study furthers our understanding how people dealing with persistent and recurrent low back pain view the issue and where their beliefs originated. The study emphasizes our need to formulate our narratives around a biopsychosocial approach and cautions disseminating negative beliefs centered around a "*body as machine*" discourse.

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Is strength or Ballistic Training Better for Developing Athletic Performance in Untrained Individuals?

Adaptations in Athletic Performance after Ballistic Power versus Strength Training by Cormie et al (2010).

Key Points:

1. Strength and power training elicited similar improvements in jump and sprint performance while strength training improved maximal strength significantly more than power training.
2. The sample size is likely too small to definitively say the results of the study are transferable to the general population.
3. This study was conducted on untrained individuals which can serve as a starting point for exercise prescription in this cohort. The recommendations will likely change in more trained individuals.

Introduction:

The topic of what training modality is best to maximize gains in athletic performance is constantly up for debate. Some advocate for only sport-specific training, while others prefer a more general approach to athletic development. These preferences are often rooted in anecdotal experience and conjecture. If we are to develop the best training programs with which to facilitate athletic development, we need good science to justify our recommendations. This is likely contingent upon the individual athlete's goals or, within the general population, a means with which to begin athletic development. Among untrained subjects, different modalities have all shown efficacy for athletic development, but there is no consensus on which modality is best when initiating training. This randomized controlled trial set out to study the effects of strength training or power training versus a control group on outcomes pertaining to athletic development.

Subjects & Methods:

Subjects

The subjects in this study were 24 males who could perform a back squat with "*proficient technique*." Their baseline demographics were: age 23.9 +/- 4.8 years, height 180.0 +/- 6.4 cm, mass 79.8 +/- 12.0 kg.

Methods

The subjects were randomized into three groups; strength training (ST), power training (PT), and control. The ST and PT groups trained three days/week for 10 weeks while the control maintained their prior level of activity. The ST group performed only the back squat each session with training loads all greater than 75% 1RM. The weekly variance in training went as follows:

- Session 1-3 sets of 3 at 90% 1RM
- Session 2-3 sets of 6 at 75% 1RM
- Session 3-3 sets of 4 at 80% 1RM

Rest between sets was 5 minutes. One rep max was determined at the first session with load reduced by 5% for the remainder of a session if subjects were unable to complete the sets and repetitions.

The power training session consisted of a warm-up with a session split of:

- Session 1 and 3- 7 sets of 6 maximal effort squat jumps
- Session 2- 5 sets of 5 maximal effort squat jumps with 30% 1 RM

Rest between sets was 3 minutes.

Subjects were tested on muscle architecture of the vastus lateralis, maximal dynamic strength via a 1RM back squat, body composition via dual energy x-ray absorptiometry (DEXA), maximal isometric strength, jump squat performance at 0%, 20%, 40%, 60%, and 80% of back squat 1RM and squat jump at 0% 1RM. On a separate day they were tested on a 40-m sprint. The ST group completed an additional testing session 7 days after the first with tests identical to those of session 1.

Results:

At baseline there were no differences between groups for 1RM, 1RM/BM or maximal isometric force. Both the ST and PT groups improved their overall athletic performance in jump (peak power ST=17.7%+/-9.3%, PT=17.6% +/-4.5%) and sprint (40m time ST=2.2% +/- 1.9%, PT=3.6% +/-2.3%). Both groups also improved their maximal strength, but the ST group significantly more so (squat 1RM ST=31.2% +/-11.3%, PT=4.5% +/-7.1%).

The ST group also increased average leans mass of the legs while the power training group experienced a slight decrease in mass. Muscle thickness of the vastus lateralis also increased greater than 10% in the ST group while it was less than 5% in the power training group.

Take Home Message:

While both groups did improve on the outcomes studied, overall the ST group improved more on the variables related to strength, with no differences seen in outcomes related to power. While these were untrained individuals, this is not far from where many people begin when looking to start training. A simple program of back squatting 3x/week was able to increase both strength and power. We often look to develop complex programs for novice athletes as a means of addressing the many deficits with which they present. This study demonstrates that a "*less is more*" approach can be used with which to improve a myriad of athletic components in untrained individuals.

This has been demonstrated in the youth population by a meta-analysis by Behm et al in which consistently large effect sizes were shown for strength training in athletic development.

If the goal is to increase overall athletic performance, we are likely best served with a strength training foundation before the addition of power components. What is more, strength training alone was able to improve variables typically associated with power, such as jump power and sprint speed. There does seem to be a correlation with foundational strength levels and the ability to improve power as well as demonstrated by James et al. While this study was underpowered to make definitive claims, it would lend credence for the integral role of strength training in the development of athletic skills in an untrained population.

We have recommendations for dosage in resistance training for both the youth and older populations by Lesinski and Borde respectively. Gains in many variables can be made from a simple resistance training program including back squatting, therefore following current recommendations for programming should be able to improve outcomes. Both of those reviews recommended up to 5 exercises with intensity greater than 75%. If an individual is untrained, it is safe to start with resistance training as a means of developing overall athletic development, and likely more efficacious than an early focus on power/ballistic training. This study used a dose below the current recommended threshold for long term athletic development and still showed improvements in strength and power. Further research should look to implement current recommendations into a program to track changes in untrained individuals.

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