One on One

The One-On-One Column provides scientifically supported, practical information for personal trainers who work with apparently healthy individuals or medically-cleared special populations.

COLUMN EDITOR: Paul Sorace, MS, RCEP, CSCS*D

The Muscle Pump: Potential Mechanisms and Applications for Enhancing Hypertrophic Adaptations

Brad J. Schoenfeld, PhD, CSCS, CSPS, NSCA-CPT[1] and Bret Contreras, MA[2]
[1]Department of Health Sciences, Program of Exercise Science, City University of New York, Lehman College, New York, New York; and
[2]Department of Sport Performance, Auckland University of Technology, Auckland, New Zealand

ABSTRACT

CELLULAR SWELLING, OFTEN REFERRED TO AS "THE PUMP," HAS BEEN SHOWN TO MEDIATE INCREASES IN MUSCLE PROTEIN SYNTHESIS AND DECREASED PROTEIN DEGRADATION. THIS PAPER WILL EXPLORE THE POTENTIAL HYPERTROPHIC BENEFITS ASSOCIATED WITH THE PUMP AND DISCUSS PRACTICAL IMPLICATIONS FOR RESISTANCE TRAINING PROGRAM DESIGN.

INTRODUCTION

Resistance exercise has been shown to induce acute alterations of intra- and extracellular water balance (36), the extent of which is dependent on the type of exercise and intensity of training. The model for these changes in fluid balance has been described as such: During intense muscular contractions, the veins taking blood out of working muscles are compressed, whereas arteries continue to deliver blood into the working muscles, thereby creating an increased concentration of intramuscular blood plasma. This causes plasma to seep out of the capillaries and into the interstitial spaces. The buildup of fluid in the interstitial spaces brings about an extracellular pressure gradient, which triggers a flow of plasma back into the muscle (i.e., reactive hyperemia) (34). This enhanced reperfusion results in a phenomenon commonly referred to by sports scientists as "cellular swelling" and by bodybuilders as "the pump," whereby muscles become engorged with blood. The pump is magnified by resistance exercise that relies heavily on anaerobic glycolysis, particularly "bodybuilding-style training" that involves moderate to higher repetitions with limited rest intervals (35). Such exercise results in a substantial accumulation of metabolic byproducts including lactate and inorganic phosphate, which in turn function as osmolytes and thereby draw additional fluid into the cell (8,37).

The pump is generally thought to be a temporary phenomenon. Bodybuilders "pump up" by performing high repetition sets immediately before a competition in an effort to make their muscles appear full and dense while on stage (24). Moreover, there is a heightened sensation of

pleasure associated with the pump, which has been popularly described by Arnold Schwarzenegger as a "tight feeling…like somebody is blowing air into your muscles…it feels fantastic." (27). Lifters therefore often will "chase the pump" in their training regimens, structuring workouts to maximize intracellular fluid accumulation. Although these short-term effects of the pump are well documented, recent research suggests that the pump may, in fact, mediate long-term adaptive responses. This paper will explore the potential hypertrophic benefits associated with the pump and discuss practical implications for resistance training program design.

THE ROLE OF HEAVY LOADING IN MUSCLE HYPERTROPHY

Muscle hypertrophy represents the dynamic balance between protein synthesis and breakdown. Three primary factors have been postulated to mediate hypertrophic adaptations pursuant to resistance training: mechanical tension, metabolic stress, and muscle damage (34). There is compelling evidence that mechanical tension is the primary impetus for this adaptive response. Goldberg et al. (10) was the first to report that heightened force development is the critical factor governing increases in muscle hypertrophy. This finding has since been corroborated in numerous studies (17,26,38,43,46).

Tension on muscles initiates a phenomenon called mechanotransduction whereby sarcolemmal-bound mechanosensors, such as integrins and focal adhesions, convert mechanical energy into chemical signals that mediate various intracellular anabolic and catabolic pathways in a manner that shifts muscle protein balance to favor synthesis over degradation (48). Studies show that mechanical tension directly stimulates mammalian target of rapamycin (mTOR) (16), possibly through activation of the extracellular regulated kinase/tuberous sclerosis complex 2 pathway (26). These actions are believed to be carried out via synthesis of the lipid second messenger phosphatidic acid (PA) by phospholipase D (16,30). Research also indicates that PA can phosphorylate the downstream anabolic translational regulator p70S6 kinase in an mTOR-independent fashion (22), presenting yet another path whereby mechanical stimuli may directly drive anabolic processes.

Given the importance of mechanical tension in promoting anabolism, it is logical to conclude that training with heavy loads is an effective means for increasing muscle growth. The use of higher intensities places greater tension on muscles, thus stimulating greater mechanotransduction. As noted, however, other factors are purported to play a role in postexercise muscle protein accretion. In particular, there is compelling evidence that exercise-induced metabolic stress can mediate a hypertrophic response, and cell swelling is believed to be an important component to this process (35).

POTENTIAL HYPERTROPHIC MECHANISMS OF CELL SWELLING

In simple terms, the pump represents an increase in intracellular hydration that causes the muscle fiber to swell. Research shows that cell swelling acts as a physiological regulator of cell function (14,15), stimulating protein accretion by both increasing protein synthesis and decreasing protein breakdown (13,25,40). These effects have been demonstrated in a variety of different cell types including hepatocytes, osteocytes, breast cells, and muscle fibers (21). In muscle, fast-twitch (FT) fibers have been found to be particularly sensitive to osmotic changes, presumably related to their high concentration of water transport channels called aquaporin-4 (AQP4). AQP4 is strongly expressed in the sarcolemma of mammalian FT glycolytic and FT oxidative-glycolytic fibers, facilitating the entry of plasma into the cell (8). Numerous studies show that FT fibers display a superior potential for growth as compared with slow-twitch fibers (1,2,18,39), suggesting that cell swelling may promote hypertrophy by favorably impacting net protein balance in these fibers. Indeed, ablation of AQP4 was found to correlate with muscular atrophy in mice (3), although it is not clear whether this finding is related to an inhibition of cell swelling or simply a reduction in spontaneous physical activity.

Although the underlying mechanisms remain to be fully elucidated, it has been hypothesized that cell swelling-induced anabolism is a means of cell survival (Figure). According to theory, an increased pressure against the cytoskeleton and/or cell membrane is perceived as a threat to cellular integrity, thereby initiating an intracellular signaling response that promotes reinforcement of its ultrastructure (20,34). The signaling response is believed to be facilitated by integrin-associated volume osmosensors within muscle fibers (23). When the membrane is subjected to swelling-induced stretch, these sensors initiate activation of anabolic protein-kinase transduction pathways, potentially regulated at least in part by growth factors that exert their influence in an autocrine/paracrine fashion

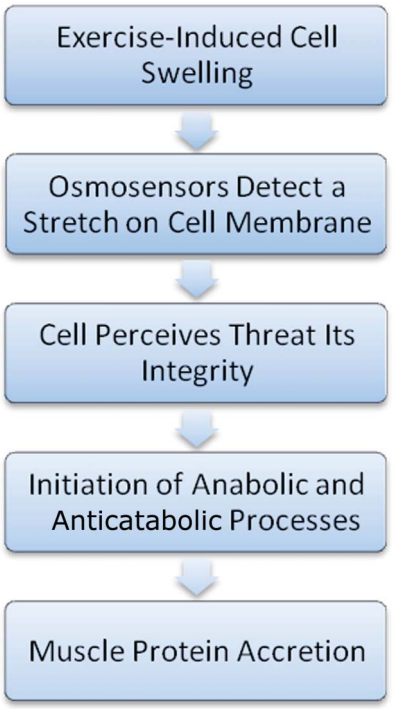

Figure. Theoretical schematic for cellular swelling mechanisms of action on muscle hypertrophy.

(4,19). Research suggests that these functions are carried out in an mTOR-dependent (9) and/or independent (32) manner, and there is evidence that mitogen-activated protein kinase pathways may play a role in associated anabolic signaling (7,33). Hyperhydration also may have a direct effect on amino acid transport systems. Phosphatidylinositide 3-kinase appears to be an important signaling component in modulating glutamine and methylaminoisobutyric acid transport in muscle because of increased cellular hydration (23).

It has been hypothesized that cellular swelling may enhance hypertrophic adaptations through increased satellite cell activity (6). Satellite cells are muscle stem cells that reside between the basal lamina and sarcolemma. While resting, these precursor cells remain quiescent. When muscle is subjected to mechanical overload, however, satellite cells enter the cell cycle and initiate muscular repair by first undergoing proliferation and then differentiating into myoblast-like cells (31). Once differentiated, myoblasts are then able to fuse to traumatized myofibers and donate their nuclei to increase the cell's ability to synthesize new contractile proteins (47). Studies investigating the myogenic properties of creatine monohydrate (CM), an osmolyte, show a positive impact on satellite cell accretion (29) and differentiation (44), as well as myogenic regulatory factor expression (45). Dangott et al. (6) proposed that the osmolytic properties of CM may instigate proliferation of satellite cells and facilitate their fusion to hypertrophying myofibers. At this time, the satellite cell hypothesis remains speculative, however, because it is not clear whether myogenic effects are, in fact, mediated by cell swelling or simply resultant to external overload.

PRACTICAL APPLICATIONS

To date, there is a paucity of resistance training studies directly investigating the effects of acute cell swelling (i.e., the pump) on muscle hypertrophy. However, basic research provides a compelling reason to believe that exercise-induced cell swelling enhances hypertrophic gains. To achieve a pump, local muscle activation must be high enough to occlude venous output; however, the contractions must be repeated for sufficient repetitions to allow for the pooling of blood. Furthermore, muscle tension must remain persistent to prevent blood from escaping the musculature. For these reasons, exercise selection and manner of execution must be chosen wisely to provide a maximal cell swelling stimulus.

Bodybuilders seeking the pump generally employ 2 different sets, repetitions, and timing schemes. The first is the use of several high repetition sets combined with short rest periods. An example would be 2–3 sets of ~20 repetitions with 60 seconds of rest in between sets. The second is the use of repeated medium repetition sets combined with short rest periods. An example would be 5–10 sets of 8–12 repetitions with 30 seconds of rest in between sets. Both of these strategies are viable approaches and conceivably can be used interchangeably to maximize the pump.

Another option for enhancing the pump is to perform a drop set, whereby a high intensity set is immediately followed by a lower intensity bout with the load decreased by ~25–50%. This training strategy results in significant metabolite accumulation (12), thereby enhancing cellular hydration. Goto et al. (11) showed that a drop set protocol resulted in a significant increase in muscle cross sectional area, opposed to a traditional high-intensity strength training protocol alone. However, the study did not control for total training volume, leaving open the possibility that the increased muscle protein accretion was the result of an increased volume rather than from the effects of cell swelling.

Exercise selection is an important aspect of pump training. Some exercises place more constant loading on the musculature because of their torque-angle curves, whereas others load up a particular range of motion but diminish drastically in other ranges. Because cellular swelling is predicated on a prolonged venous occlusion, those exercises that maintain constant tension would necessarily maximize the pump. For example, the good morning requires the greatest muscle force in the hip extensors at long muscle lengths; the 45° hyperextension at medium muscle lengths and the horizontal back extension at short muscle lengths (5). Although the good morning, therefore, would have the greatest impact on inducing muscle damage (28), the lack of tension in the upper range of movement would diminish cellular swelling. On the other hand, the constant muscular tension (i.e., mean torque loading throughout the repetition) associated with the 45° hyperextension heightens vascular occlusion, thereby resulting in a greater pump. Traditional single-joint machine exercises such as the "pec deck," "reverse pec deck," leg extension, and seated leg curl exercises are generally good choices for pump training because of the constant tension they place on the musculature.

Exercises can also be modified for a greater pump effect. Exercises that have diminished loading on a particular muscle throughout the range of motion can be altered so that performance focuses only on the portion of the movement that maximally stresses the muscle is performed. For example, bottom-half push-ups or dips are a better strategy for achieving a pump in the pectorals than full range push-ups or dips. Resistance bands and chains can also be used in concert with the barbell to accommodate the strength curve and place more tension that is constant on the muscle.

Finally, when training for the pump, it is important to perform exercises in a continuous manner so that the target muscles are not allowed to relax. Tanimoto and Ishii (41) showed a significant decrease in local muscle oxygenation–consistent with vascular occlusion–in the performance of low-intensity knee extension exercise (50% 1RM) without

a relaxation phase as compared with high-intensity (80% 1RM) exercise performed with a 1-second relaxation between repetitions. The authors attributed this decrease in muscle oxygenation level to the continuous contractions of the knee extensor muscles in exercise without relaxation. Similar results were reported in follow-up during multijoint lower-body exercise (42), emphasizing the importance of maintaining continuous tension on the working muscles if the goal is to maximize cellular swelling.

CONCLUSION

In summary, progressive resistance training in low-to-medium repetition ranges has earned its keep in the training programs of bodybuilders and other athletes seeking to maximize hypertrophy, for good reason. Heavy loads maximize muscle activation, and progressive overload ensures that muscles receive increased mechanical tension over time. Therefore, increasing strength on heavy multijoint movements should be the foundation of long-term hypertrophy training. However, it is likely that exercise centered on achieving a "pump" through higher repetition sets combined with shorter rest periods also provides a potent hypertrophic stimulus that is synergistic to heavy compound lifting. Therefore, individuals seeking maximal hypertrophy should consider dedicating a component of their training sessions toward "pump" training, ideally after heavier strength work, to take advantage of the multiple pathways involved in muscle hypertrophy.

Future research should be undertaken to investigate whether cell swelling, in fact, leads to increased hypertrophy over that of heavy strength training alone (i.e., whether its inclusion is additive or redundant). Moreover, future research should determine the precise mechanisms through which "pump" training increases hypertrophy and determine which exercises and training methods are best suited for eliciting a pump in the various muscles of the body. Finally, future research should dictate the optimal manner in which heavier strength training and lighter pump training can be integrated together to maximize hypertrophic adaptations.

Conflicts of Interest and Source of Funding: The authors report no conflicts of interest and no source of funding.

Brad J. Schoenfeld *is a lecturer in the exercise science program at CUNY's Lehman College and director of their human performance laboratory.*

Bret Contreras *is currently pursuing his PhD in Sports Science at the Auckland University of Technology in Auckland, New Zealand.*

REFERENCES

1. Aagaard P, Andersen JL, Dyhre-Poulsen P, Leffers AM, Wagner A, Magnusson SP, Halkjaer-Kristensen J, and Simonsen EB. A mechanism for increased contractile strength of human pennate muscle in response to strength training: Ahanges in muscle architecture. *J Physiol* 534: 613–623, 2001.
2. Adams G and Bamman MM. Characterization and regulation of mechanical loading-induced compensatory muscle hypertrophy. *Compr Physiol* 2: 2829–2870, 2012.
3. Basco D, Blaauw B, Pisani F, Sparaneo A, Nicchia GP, Mola MG, Reggiani C, Svelto M, and Frigeri A. AQP4-dependent water transport plays a functional role in exercise-induced skeletal muscle adaptations. *PLoS One* 8: e58712, 2013.
4. Clarke MS and Feeback DL. Mechanical load induces sarcoplasmic wounding and FGF release in differentiated human skeletal muscle cultures. *FASEB J* 10: 502–509, 1996.
5. Contreras B, Cronin J, Schoenfeld BJ, Nates R, and Sonmez GT. Are all hip extension exercises created equal? *Strength Cond J* 35: 17–22, 2013.
6. Dangott B, Schultz E, and Mozdziak PE. Dietary creatine monohydrate supplementation increases satellite cell mitotic activity during compensatory hypertrophy. *Int J Sports Med* 21: 13–16, 2000.
7. Finkenzeller G, Newsome W, Lang F, and Haussinger D. Increase of c-jun mRNA upon hypo-osmotic cell swelling of rat hepatoma cells. *FEBS Lett* 340: 163–166, 1994.
8. Frigeri A, Nicchia GP, Verbavatz JM, Valenti G, and Svelto M. Expression of aquaporin-4 in fast-twitch fibers of mammalian skeletal muscle. *J Clin Invest* 102: 695–703, 1998.
9. Fry CS, Glynn EL, Drummond MJ, Timmerman KL, Fujita S, Abe T, Dhanani S, Volpi E, and Rasmussen BB. Blood flow restriction exercise stimulates mTORC1 signaling and muscle protein synthesis in older men. *J Appl Physiol (1985)* 108: 1199–1209, 2010.
10. Goldberg AL, Etlinger JD, Goldspink DF, and Jablecki C. Mechanism of work-induced hypertrophy of skeletal muscle. *Med Sci Sports* 7: 185–198, 1975.
11. Goto K, Ishii N, Kizuka T, and Takamatsu K. The impact of metabolic stress on hormonal responses and muscular adaptations. *Med Sci Sports Exerc* 37: 955–963, 2005.
12. Goto K, Sato K, and Takamatsu K. A single set of low intensity resistance exercise immediately following high intensity resistance exercise stimulates growth hormone secretion in men. *J Sports Med Phys Fitness* 43: 243–249, 2003.
13. Grant AC, Gow IF, Zammit VA, and Shennan DB. Regulation of protein synthesis in lactating rat mammary tissue by cell volume. *Biochim Biophys Acta* 1475: 39–46, 2000.
14. Haussinger D. The role of cellular hydration in the regulation of cell function. *Biochem J* 313: 697–710, 1996.
15. Haussinger D, Lang F, and Gerok W. Regulation of cell function by the cellular hydration state. *Am J Physiol* 267: E343–E355, 1994.
16. Hornberger TA, Chu WK, Mak YW, Hsiung JW, Huang SA, and Chien S. The role of phospholipase D and phosphatidic acid in the mechanical activation of mTOR signaling in skeletal muscle. *Proc Natl Acad Sci U S A* 103: 4741–4746, 2006.
17. Hornberger TA, Stuppard R, Conley KE, Fedele MJ, Fiorotto ML, Chin ER, and Esser KA. Mechanical stimuli regulate rapamycin-sensitive signalling by a phosphoinositide 3-kinase-, protein kinase B- and growth factor-independent mechanism. *Biochem J* 380: 795–804, 2004.
18. Kosek DJ, Kim JS, Petrella JK, Cross JM, and Bamman MM. Efficacy of 3 days/wk resistance training on myofiber hypertrophy

and myogenic mechanisms in young vs. older adults. *J Appl Physiol* 101: 531–544, 2006.

19. Lambert IH, Hoffmann EK, and Pedersen SF. Cell volume regulation: Physiology and pathophysiology. *Acta Physiol (Oxf)* 194: 255–282, 2008.
20. Lang F. Mechanisms and significance of cell volume regulation. *J Am Coll Nutr* 26: 613S–623S, 2007.
21. Lang F, Busch GL, Ritter M, Volkl H, Waldegger S, Gulbins E, and Haussinger D. Functional significance of cell volume regulatory mechanisms. *Physiol Rev* 78: 247–306, 1998.
22. Lehman N, Ledford B, Di Fulvio M, Frondorf K, McPhail LC, and Gomez-Cambronero J. Phospholipase D2-derived phosphatidic acid binds to and activates ribosomal p70 S6 kinase independently of mTOR. *FASEB J* 21: 1075–1087, 2007.
23. Low SY, Rennie MJ, and Taylor PM. Signaling elements involved in amino acid transport responses to altered muscle cell volume. *FASEB J* 11: 1111–1117, 1997.
24. Meth S. Gender differences in muscle morphology. In: Swedan NG, ed. *Women's Sports Medicine and Rehabilitation*. Philadelphia, PA: Lippincott Williams & Wilkins, 2001. pp. 5.
25. Millar ID, Barber MC, Lomax MA, Travers MT, and Shennan DB. Mammary protein synthesis is acutely regulated by the cellular hydration state. *Biochem Biophys Res Commun* 230: 351–355, 1997.
26. Miyazaki M, McCarthy JJ, Fedele MJ, and Esser KA. Early activation of mTORC1 signalling in response to mechanical overload is independent of phosphoinositide 3-kinase/Akt signalling. *J Physiol* 589: 1831–1846, 2011.
27. Monaghan L. Looking good, feeling good: The embodied pleasures of vibrant physicality. *Sociol Health Illness* 23: 330–356, 2001.
28. Nosaka K and Sakamoto K. Effect of elbow joint angle on the magnitude of muscle damage to the elbow flexors. *Med Sci Sports Exerc* 33: 22–29, 2001.
29. Olsen S, Aagaard P, Kadi F, Tufekovic G, Verney J, Olesen JL, Suetta C, and Kjaer M. Creatine supplementation augments the increase in satellite cell and myonuclei number in human skeletal muscle induced by strength training. *J Physiol* 573: 525–534, 2006.
30. O'Neil TK, Duffy LR, Frey JW, and Hornberger TA. The role of phosphoinositide 3-kinase and phosphatidic acid in the regulation of mammalian target of rapamycin following eccentric contractions. *J Physiol* 587: 3691–3701, 2009.
31. Philippou A, Halapas A, Maridaki M, and Koutsilieris M. Type I insulin-like growth factor receptor signaling in skeletal muscle regeneration and hypertrophy. *J Musculoskelet Neuronal Interact* 7: 208–218, 2007.
32. Schliess F, Richter L, vom Dahl S, and Haussinger D. Cell hydration and mTOR-dependent signalling. *Acta Physiol (Oxf)* 187: 223–229, 2006.
33. Schliess F, Schreiber R, and Haussinger D. Activation of extracellular signal-regulated kinases Erk-1 and Erk-2 by cell swelling in H4IIE hepatoma cells. *Biochem J* 309: 13–17, 1995.
34. Schoenfeld BJ. The mechanisms of muscle hypertrophy and their application to resistance training. *J Strength Cond Res* 24: 2857–2872, 2010.
35. Schoenfeld BJ. Potential mechanisms for a role of metabolic stress in hypertrophic adaptations to resistance training. *Sports Med* 43: 179–194, 2013.
36. Sjogaard G. Water and electrolyte fluxes during exercise and their relation to muscle fatigue. *Acta Physiol Scand Suppl* 556: 129–136, 1986.
37. Sjogaard G, Adams RP, and Saltin B. Water and ion shifts in skeletal muscle of humans with intense dynamic knee extension. *Am J Physiol* 248: R190–R196, 1985.
38. Spangenburg EE, Le Roith D, Ward CW, and Bodine SC. A functional insulin-like growth factor receptor is not necessary for load-induced skeletal muscle hypertrophy. *J Physiol* 586: 283–291, 2008.
39. Staron RS, Malicky ES, Leonardi MJ, Falkel JE, Hagerman FC, and Dudley GA. Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. *Eur J Appl Physiol Occup Physiol* 60: 71–79, 1990.
40. Stoll BA and Secreto G. Prenatal influences and breast cancer. *Lancet* 340: 1478, 1992.
41. Tanimoto M and Ishii N. Effects of low-intensity resistance exercise with slow movement and tonic force generation on muscular function in young men. *J Appl Physiol (1985)* 100: 1150–1157, 2006.
42. Tanimoto M, Sanada K, Yamamoto K, Kawano H, Gando Y, Tabata I, Ishii N, and Miyachi M. Effects of whole-body low-intensity resistance training with slow movement and tonic force generation on muscular size and strength in young men. *J Strength Cond Res* 22: 1926–1938, 2008.
43. Vandenburgh H and Kaufman S. In vitro model for stretch-induced hypertrophy of skeletal muscle. *Science* 203: 265–268, 1979.
44. Vierck JL, Icenoggle DL, Bucci L, and Dodson MV. The effects of ergogenic compounds on myogenic satellite cells. *Med Sci Sports Exerc* 35: 769–776, 2003.
45. Willoughby DS and Rosene JM. Effects of oral creatine and resistance training on myogenic regulatory factor expression. *Med Sci Sports Exerc* 35: 923–929, 2003.
46. Witkowski S, Lovering RM, and Spangenburg EE. High-frequency electrically stimulated skeletal muscle contractions increase p70s6k phosphorylation independent of known IGF-I sensitive signaling pathways. *FEBS Lett* 584: 2891–2895, 2010.
47. Zammit PS. All muscle satellite cells are equal, but are some more equal than others? *J Cell Sci* 121: 2975–2982, 2008.
48. Zou K, Meador BM, Johnson B, Huntsman HD, Mahmassani Z, Valero MC, Huey KA, and Boppart MD. The alpha(7) beta(1)-integrin increases muscle hypertrophy following multiple bouts of eccentric exercise. *J Appl Physiol (1985)* 111: 1134–1141, 2011.