Are Multiple Reps Required for Hypertrophy?

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CONSIDERABLE CONFUSION exists as to how best to develop muscle hypertrophy. In adult mammals, most (95–100%) of total muscle hypertrophy can be attributed to cellular hypertrophy. Cellular hypertrophy is an increase in the cross-sectional area of a muscle cell. This increase in cell size is a direct muscle cell adaptation to progressive resistive training and can be attributed to (a) an increase in protein synthesis, particularly in the contractile proteins actin and myosin, (b) an increase in the number of myofibrils within the muscle cell, and (c) an increase in the size of the myofibrils within the muscle cell. The majority of these hypertrophic changes occur over the course of training and can be explained in part by 2 basic principles: specificity and overload.

When an untrained, previously sedentary individual begins a resistive training program, initial changes, especially related to the noticeable increases in strength, are more neurological than cellular. After this initial period in a resistive training program, the hypertrophic response in tissue begins. During this hypertrophic phase of training, training intensity is usually low and varies between 50 and 75% of 1RM. The training volume however is reasonably high. This high volume is achieved by incorporating high repetitions (10–20) and multiple sets (3–6) into the training program. This high training volume appears to be the critical factor when attempting to stimulate hypertrophic muscle cell adaptation. Optimal muscle cellular and organ growth appears to be associated with higher training volume. After long-term adaptation has occurred, a muscle fiber will approach its genetic hypertrophic limitation and high training volume will produce no further hypertrophic adaptation.

Consider the following 3 training scenarios designed to develop hypertrophy, strength, and power: (a) 60% of 1RM: 15 repetitions, 5 sets; (b) 85% of 1RM: 6 repetitions, 4 sets; and (c) 95% of 1RM: 3 repetitions, 3 sets. The total volume loads for these 3 scenarios are 4,500, 2,040, and 855, respectively. Although all 3 scenarios will provide sufficient stimulus for cellular adaptation, scenarios (b) and (c) require a level of training so high that even the most well-trained individual would not be able to sustain this level of demand without risk of overtraining. As a result, more recovery is required, limiting total training volume and the stimulus for hypertrophic growth. Thus, I recommend a high training volume, including multiple repetitions, when attempting to maximize hypertrophic cellular adaptation. I also recommend a program similar to the one described as scenario (a), coupled with multiple exercises per muscle group designed to vary the training angle, to allow for maximal muscle fiber stimulation and adaptation. If this type of program is followed, maximal muscle cellular and organ hypertrophy can be obtained. ▲

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Many different repetition schemes can induce muscular hypertrophy. However, a moderate repetition scheme (approximately 8–10 repetitions per set) provides the greatest potential for muscular growth. Moderate repetitions allow for the use of reasonably heavy weights, a prerequisite for optimal stimulation of muscle fibers. If the weights used are too light to stimulate available fibers optimally, hypertrophy will be suboptimal. This approach excludes high-repetition schemes and leaves either moderate- or low-repetition schemes as the best alternatives for maximizing hypertrophy.

Moderate-repetition schemes have a major advantage over those that advocate low numbers of repetitions in that the glycolytic energy system is the primary source for fuel (low numbers of repetitions are fueled predominantly by the phosphocreatine system). The energy pathway is significant because of the way lactate is handled. Lactate is a by-product of glycolysis and produces several anabolic effects. It potentiates the release of growth hormone, which in turn acts as a mediator of insulin-like growth factor 1 (IGF-1). Local IGF-1 expression is one of the primary hypertrophic stimulators that act through a proliferation of satellite cells and increased muscle protein synthesis. Local IGF-1 acts in both an autocrine/paracrine and an endocrine mode. The autocrine/paracrine isoform is called mechano-growth factor and is activated only by mechanical stimuli. However, the endocrine isoform is similar to hepatic IGF-1 and therefore is mediated by growth hormone (gH). It is likely that gH significantly increases intramuscular IGF-1 levels and thereby increases muscle protein synthesis.

There is some evidence that gH has a more direct effect on muscle mass. In birds, gH elicits satellite cell proliferation in an IGF-independent manner. Because satellite cells are an essential component of the hypertrophic response, this proliferation is significant. In rats, gH induces substantial hypertrophy of type II lower limb fibers. Similar results have been obtained in dogs, with hypertrophy of both type I and type II fibers. Although a clear link between exercise-induced gH levels and hypertrophy has not been established, a link probably exists.

More importantly, training with moderate repetition helps to increase cellular hydration. The combination of a muscle pump and lactate formation causes a concentration gradient that pulls water into the muscle cell. Cell swelling may augment hypertrophy. Simply put, when a cell becomes hydrated, it perceives the increased volumization as a threat to its integrity and responds by growing larger. Cellular hydration may increase protein synthesis and decrease proteolysis; these processes combined are essential for muscular growth. Because fast-twitch fibers (those with the greatest potential for growth) contain a water transport protein called aquaporin 4, they appear to be the primary beneficiaries of this hydration effect.

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