Experience Memory/Muscle Memory

Article one of a two article series

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Introduction

The concept of your body remembering how to do and actually doing almost anything is a lot easier than relearning how to do it from scratch is something many of us have experienced. Take riding a bike. Once you've learned how to ride a bike it's there for life as long as you haven't become disabled in one way or another. It's the same for almost any activity or process.

A personal example is a friend of mine who in his early years got pretty good at playing pool. He didn't play for over three decades, but it didn't take long before he was on his game again. His body kept the memory and thus his ability of his earlier pool expertise.

It's interesting to explore and also be able to utilize what I call experience memory (EM). The ability to relatively quickly to start again where we left off no matter what the activity as long as there aren't any significant mitigating factors, such as a decline in physical and/or mental health.

The obvious answer, at least to me, involves epigenetics, the influence of the environment on the raw DNA. Think of it as the old nature versus nurture controversy as to what's more important in forming who and what we are.

I never thought of this as an either/or concept as I've always believed that both are important to one degree or another. This belief was reinforced over five decades ago when I majored in genetics, and molecular biochemistry for my research-oriented Honors B.Sc. and later on when I went into medicine for my M.D. It was also a concept that I used in my philosophy dissertation.

Although the epigenetic model, where the environment influences the expression of DNA, was my first obvious explanation, the mechanism of how EM works its magic took a while to figure out and is something that is still a work in progress.

Muscle Memory

Rather than go in detail on the neuroanatomy of EM, a discussion that would take a book rather than an article, I'm going to examine the concept of muscle memory as a subset of neuromuscular learning. More specifically I'm going to concentrate on the hypertrophy and atrophy aspects of the skeletal muscle itself, as well as touching on hyperplasia.

Because of my heavy involvement in exercise training and competition in various sports, especially Powerlifting, the idea of muscle memory has always intrigued me. Muscle memory as related to resistance training, involving muscle hypertrophy, and at times muscle hyperplasia..

The Myonuclei Domain Hypothesis

The myonuclear domain theory is based on the limited amount of skeletal muscle cytoplasm that is under the control of a single myonucleus thus the need for multiple myonuclei in single muscle fibers. The greater the size of the muscle fiber, the more myonuclei are needed for the muscle fiber to grow in size.

A number of studies have shown that training increases the number of myonuclei in muscle fibers via the transformation of satellite/stem cells into myonuclei that are incorporated and become part of the muscle cell. The theory behind this belief is that skeletal muscle cells are extremely large and need multiple nuclei, each of which handles a certain circumscribed part of the muscle cell. So as the theory goes, with muscle hypertrophy extra myonuclei are needed as muscle cells get larger.

As muscles atrophy after a quiescent period of less to no exercise it's been discovered by some studies that while the muscle cells get smaller, the myonuclei don't decrease in number, thus maintaining the potential for an easier path to muscle hypertrophy when exercise is started up again. The theory thus provides a pathway for muscle memory. The theory is based on the fact that newly added myonuclei stick around even after the muscles atrophy from decreased use but when you use them again the whole muscle machinery is ready and able to regain muscle mass much easier than if you never had that muscle mass (and increased myonuclei) in the first place.

Possible Dilemma for Doping Control

If this theory holds true then there may be a dilemma as far as drug testing. For example, using performance enhancing drugs (PEDs), and especially drugs that are or mimic anabolic steroids and GH/IGF-1 , leads to an increase in the hypertrophic response and thus to added myonuclei due to the increase in skeletal muscle mass.

If this memory is retained then the use of these compounds may offer an advantage beyond the time that they were used. Thus, the dilemma in drug testing – should any athlete testing positive for these PEDs be banned for life?

Summary:

Training leads to muscle damage. Satellite cells react to the damage by attaching themselves to muscle fibers thus leading to increased muscle myonuclei and subsequent hypertrophy during recovery. With less or no exercise muscle fibers decrease in size but the number of myonuclei remain the same thus allowing hypertrophy to develop faster upon resuming exercising.

Is the Theory behind the Myonuclear Domain Valid?

The concept that increasing myonuclei are necessary for muscle hyperplasia, with the corollary that since the myonuclei remain in muscle and thus facilitate muscle hypertrophy after muscle atrophy seems to explain muscle memory.

However, in reasons I will give in part two of this article, the myonuclear domain theory is likely only a small part of the answer as to the processes underlying skeletal muscle hypertrophy and hyperplasia, and the concept of muscle memory.

Schwartz LM. Skeletal Muscles Do Not Undergo Apoptosis During Either Atrophy or Programmed Cell Death-Revisiting the Myonuclear Domain Hypothesis. Front Physiol. 2019 Jan 25;9:1887. doi: 10.3389/fphys.2018.01887.

Skeletal Muscles Do Not Undergo Apoptosis During Either Atrophy or Programmed Cell Death-Revisiting the Myonuclear Domain Hypothesis

Abstract

Skeletal muscles are the largest cells in the body and are one of the few syncytial ones. There is a longstanding belief that a given nucleus controls a defined volume of cytoplasm, so when a muscle grows (hypertrophy) or shrinks (atrophy), the number of myonuclei change accordingly. This phenomenon is known as the "myonuclear domain hypothesis." There is a general agreement that hypertrophy is accompanied by the addition of new nuclei from stem cells to help the muscles meet the enhanced synthetic demands of a larger cell. However, there is a considerable controversy regarding the fate of pre-existing nuclei during atrophy. Many researchers have reported that atrophy is accompanied by the dramatic loss of myonuclei *via* apoptosis. However, since there are many different non-muscle cell populations that reside within the tissue, these experiments cannot easily distinguish true myonuclei from those of neighboring mononuclear cells. Recently, two independent models, one from rodents and the other from insects, have demonstrated that nuclei are not lost from skeletal muscle fibers when they undergo either atrophy or programmed cell death. These and other data argue against the current interpretation of the myonuclear domain hypothesis and suggest that once a nucleus has been acquired by a muscle fiber it persists.

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The concept of skeletal muscle memory: Evidence from animal and human studies.

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Abstract

Within the current paradigm of the myonuclear domain theory, it is postulated that a linear relationship exists between muscle fibre size and myonuclear content. The myonuclear domain is kept (relatively) constant by adding additional nuclei (supplied by muscle satellite cells) during muscle fibre hypertrophy and nuclear loss (by apoptosis) during muscle fibre atrophy. However, data from recent animal studies suggest that myonuclei that are added to support muscle fibre hypertrophy are not lost within various muscle atrophy models. Such myonuclear permanence has been suggested to constitute a mechanism allowing the muscle fibre to (re)grow more efficiently during retraining, a phenomenon referred to as "muscle memory." The concept of "muscle memory by myonuclear permanence" has mainly been based on data attained from rodent experimental models. Whether the postulated mechanism also holds true in humans remains largely ambiguous. Nevertheless, there are several studies in humans that provide evidence to potentially support or contradict (parts of) the muscle memory hypothesis. The goal of the present review was to discuss the evidence for the existence of "muscle memory" in both animal and human models of muscle fibre hypertrophy as well as atrophy. Furthermore, to provide additional insight

in the potential presence of muscle memory by myonuclear permanence in humans, we present new data on previously performed exercise training studies. Finally, suggestions for future research are provided to establish whether muscle memory really exists in humans.

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The effect of resistance training, detraining and retraining on muscle strength and power, myofibre size, satellite cells and myonuclei in older men.

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Erratum in

- Corrigendum to "The effect of resistance training, detraining and retraining on muscle strength and power, myofibre size, satellite cells and myonuclei in older men" [Exp. Gerontol., 133, 2020, 110860]. [Exp Gerontol. 2020]

Abstract

INTRODUCTION:

Ageing is associated with an attenuated hypertrophic response to resistance training and periods of training interruptions. Hence, elderly would benefit from the 'muscle memory' effects of resistance training on muscle strength and mass during detraining and retraining. As the underlying mechanisms are not yet clear, this study investigated the role of myonuclei during training, detraining and retraining by using PCM1 labelling in muscle cross-sections of six older men.

METHODS:

Knee extension strength and power were measured in 30 older men and 10 controls before and after 12 weeks resistance training and after detraining and retraining of similar length. In a subset, muscle biopsies from the vastus lateralis were taken for analysis of fibre size, fibre type distribution, Pax7+ satellite cell number and myonuclear domain size.

RESULTS:

Resistance training increased knee extension strength and power parameters (+10 to +36%, $p < .001$) and decreased the frequency of type IIax fibres by half (from 20 to 10%, $p = .034$). Detraining resulted in a modest loss of strength and power (-5 to -15%, $p \leq .004$) and a trend towards a fibre-type specific decrease in type II fibre cross-sectional area (-17%, $p = .087$), type II satellite cell number (-30%, $p = .054$) and type II myonuclear number (-12%, $p = .084$). Less than eight weeks of retraining were needed to reach the post-training level of one-repetition maximum strength. Twelve weeks of retraining were associated with type II fibre hypertrophy (+29%, $p = .050$), which also promoted an increase in the number of satellite cells (+72%, $p = .036$) and myonuclei (+13%, $p = .048$) in type II fibres. Changes in the type II fibre cross-sectional area were positively correlated with changes in the myonuclear number (Pearson's r between 0.40 and 0.73), resulting in a stable myonuclear domain.

CONCLUSION:
Gained strength and power and fibre type changes were partially preserved following 12 weeks of detraining, allowing for a fast recovery of the 1RM performance following retraining. Myonuclear number tended to follow individual changes in type II fibre size, which is in support of the myonuclear domain theory.

Full text in PDF format is available at: https://onlinelibrary.wiley.com/doi/epdf/10.1111/apha.13465