Grandad, it ain’t what you eat, it depends when you eat it – that’s how muscles grow!

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The word ‘sarcopenia’ was coined to identify the condition of loss of skeletal muscle that occurs in wasting diseases and also during ageing. This loss of muscle constitutes a major problem for patients with a wide variety of chronic medical conditions including cancer, cardiovascular, respiratory and renal disease, and when it affects the respiratory muscles it prevents weaning from artificial respiration of patients in intensive care units. In the elderly, the maintenance of muscle strength is a major contributor to the ability to continue to carry out daily physical tasks successfully and safely. Naturally, many physiologists and rehabilitation experts have been drawn to the question of how to prevent the loss in muscle mass and possibly how to reverse it. There have now been over 50 studies carried out in older subjects, of ages up to 97 years! Most of these have demonstrated that resistance exercise can produce substantial increases in muscle strength and power and more limited, but nevertheless significant, increases in muscle mass and muscle fibre cross-sectional area (CSA) (Frontera et al. 1988; Skelton et al. 1995; Young, 1997). Nevertheless, it would make sense to find ways of increasing muscle mass in the elderly, not only because muscle strength and functionality would probably be improved more but also because of the attendant metabolic advantages in having a high lean to fat weight body ratio, including increases in glucose tolerance and the capacity to oxidize fat.

The paper published in this issue of The Journal of Physiology by Esmarck et al. (2001) provides us with some very interesting insights into the nutritional control of muscle mass. What Birgitte Esmarck and her colleagues have done is to demonstrate that, for the elderly, it matters considerably when a person eats a protein meal after having done some exercise. Delaying the consumption of a meal for 2 h after exercise limited the increase in muscle fibre growth after a programme of progressive resistance exercise in elderly men – of a mean age of 74 years. However, when the meal was taken immediately after exercise there was much greater efficacy in stimulating muscle growth, measured as increases in muscle fibre CSA (determined histochemically) and whole muscle CSA (determined by magnetic resonance imaging) (see Fig. 1).

It has been known from previous work that strenuous exercise alone stimulates human muscle protein synthesis for up to 48 h after the bout with the maximum response occurring somewhere in the period between the end of exercise and 12 h afterwards – and very probably at about 3 h (Phillips et al. 1997). This increase occurs even without amino acids being supplied exogenously, either orally or intravenously, but the stimulation is markedly increased when amino acids are given concurrently with or immediately after exercise (Tipton et al. 1999). However, the paper by Esmarck et al. (2001) is the first to demonstrate that the net result of stimulating the anabolic processes in muscle by strenuous exercise and timely consumption of a protein-containing meal causes a bigger increase in net muscle growth, i.e. fibre size and macroscopic CSA, than when the meal is delayed to 2 h after exercise.

There are, however, some puzzling aspects to the results. Blake Rasmussen and colleagues from Bob Wolfe’s lab in Galveston have shown in young subjects that there is little difference in the stimulation of muscle protein synthesis (and no difference in the inhibition of muscle protein breakdown) when a protein–carbohydrate meal is given either 1 or 3 h after strenuous exercise (Rasmussen et al. 2000). The net balances of amino acids across the previously working leg were identical, suggesting that the net accretion of protein was the same whether the meal was given at 1 or 3 h. This obviously is at odds with the results presented by Esmarck et al. (2001). Of course, the major difference may be in the relative sensitivity of the muscle of the elderly subjects to contractile activity and to exogenous amino acids. Work from the Galveston lab has also shown recently that elderly subjects appear to exhibit what might be called ‘nutrient resistance’ of protein synthesis, in as much as they show a diminished response to exogenous amino acids plus carbohydrate, compared to young subjects (Volpi et al. 2000). Maybe the elderly muscle is stimulated by contractile activity normally but the effect wears off faster than in the young and then ‘nutrient resistance’ might stop a full response to food.

This paper by Esmarck and colleagues (2001) not only provides important clues about the control of muscle mass by contractile activity and nutrition, but also provides practical insights for those wishing to help the elderly in maintaining size and functions of their muscle by a judicious combination of exercise and adequate and timely dietary intake.


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Figure 1
The relative efficacy of immediate rather than delayed post-training feeding on muscle hypertrophy (redrawn from data in Esmarck et al. 2001).