



2

Train low - compete high!

Keith Baar

Division of Molecular Physiology, University of Dundee, Dow Street, Dundee DD1 5EH, United Kingdom

Introduction

Glycogen loading has been known to increase endurance performance for many years (Bergstrom & Hultman, 1967a). As a result, most athletes and coaches believe that training in a glycogen-loaded state is essential to optimal conditioning and performance. However, the validity of this philosophy is now being challenged. It is becoming clear that there are benefits to training in a glycogen-depleted state. The potential benefits of training in the glycogen-depleted state have recently led many coaches and scientists to espouse a new training philosophy: "Train low-compete high". Here we will discuss the evidence in support of this philosophy as well as the potential mechanism underlying the benefits of training in a low glycogen state.



Importance of glycogen as a fuel for endurance exercise

Glycogen is the principal storage form of carbohydrate in mammals. In 1858 (Bernard, 1858) Claude Bernard isolated carbohydrate from liver and muscle (Young, 1957). Bernard's landmark discovery provided direct evidence that muscle and liver had an accessible form of energy for meeting energy demands during exercise. Almost a century later, Bergstrom and Hultman began to investigate the role of glycogen in exercise (Bergstrom & Hultman, 1966); discovering a relationship between glycogen and exercise performance (Bergstrom et al., 1967). These early studies demonstrated that the glycogen content of a muscle is a major determinant of the capacity to sustain endurance exercise (Bergstrom & Hultman, 1967a). Importantly, they also demonstrated that diet and exercise could greatly vary the glycogen content in skeletal muscle (Bergstrom et al., 1967). This final observation, that eating a high carbohydrate diet following exercise increased the recovery of muscle glycogen stores compared to a fat or protein diet, provided direct evidence that dietary glucose was the precursor for muscle glycogen (Bergstrom & Hultman, 1967b; Hultman & Bergstrom, 1967) and suggested for the first time that a high muscle glycogen was beneficial for endurance performance.

Glycogen and whole body substrate utilization

In the low glycogen state, whole body metabolism shifts drastically. In humans, glycogen depletion results in increased systemic release of amino acids from muscle protein breakdown, increased fat metabolism (calculated from arterio-venous differences), reduced pyruvate oxidation, and increased stress hormones such as cortisol and epinephrine (Blomstrand & Saltin, 1999; Steensberg et al., 2002). As a result of these changes, it is not surprising that performance is negatively affected by low muscle glycogen. However, some have postulated that lower glycogen during training alters whole body substrate metabolism in a manner that stimulates the activation of cellular signaling pathways that might be involved in the muscular adaptation to training (Steensberg et al., 2002).

Glycogen depletion training and endurance training adaptation

In support of the beneficial effects of training in a glycogen-depleted state, Hansen et al. (Hansen et al., 2005) have shown that 10 weeks of training in a glycogen-depleted state resulted in an 85% greater increase in time to exhaustion compared with training with high glycogen. The reason for this greater increase in endurance was a larger increase in citrate synthase (CS) and 3-hydroxyacyl-CoA dehydrogenase (HAD) and other important enzymes of fat metabolism. These results have now been confirmed in highly trained cyclists suggesting that, regardless of the athlete's training state, training in a glycogen-depleted state results in an increased capacity to use fat as a fuel during exercise.

Glycogen depletion training and endurance performance

Since training in the glycogen-depleted state improves the capacity for fat oxidation, this type of training might be expected to have a glycogen sparing effect during competition leading to improved performance. While this might be true at low intensities (<70% whole body VO_2max) exercise, it does not appear to have a positive effect on performance at higher intensities (>70% whole body VO_2max) where CHO are the primary fuel source. What this means is that in long duration endurance competition (triathlon, marathon, road cycling), training in a glycogen-depleted state will have a positive effect on performance. However, in shorter, higher intensity events (10K run, time trial cycling, rowing), training in

TOPICS



a glycogen-depleted state will have less of a performance benefit. One caveat is that for competitions such as world championships and Olympics, where heats are run prior to the finals, low glycogen training, and the resulting increase in the capacity to use fat as a fuel, may improve recovery and therefore have beneficial effects on subsequent performances.

Resistance training in a glycogen-depleted state

Unlike endurance training, resistance training in a glycogen-depleted state does not seem to have any beneficial effects. If anything, weight training in a glycogen-depleted state may decrease training adaptations. It is already clear that the transcriptional changes following resistance exercise are no different in a glycogen-depleted state and the greater metabolic stress of training with low glycogen will negatively affect the primary pathway leading to increased muscle protein synthesis. Therefore, for strength events, training in a glycogen-depleted state should be avoided.

Why is endurance training in a glycogen-depleted state beneficial?

Endurance training in a glycogen-depleted state results in an improved capacity to use fat to fuel exercise. One important question is why? Some recent work has given clues as to how training in a glycogen-depleted state results in this beneficial effect. Narkar et al (Narkar et al., 2008) recently showed that training rats on a treadmill while at the same time giving them a drug that activated a transcription factor called PPAR β resulted in the same changes that occur when training in the glycogen-depleted state: increased capacity to use fat as a fuel. Increasing the enzymes that are required for oxidizing fatty acids is what PPAR β does. The result in this study was the rats that both got the drug and trained on the treadmill increased their ability to run at $\sim 50\%$ VO_2max by 70% over those that just ran on the treadmill. These data suggest that exercising in the glycogen-depleted state activates PPAR β to a greater extent than training in the glycogen-loaded state. PPAR β seems to be activated by a byproduct of the breakdown of fat in muscle. As discussed above, exercising in the glycogen-depleted state increases circulating fatty acids and the oxidation of fat during exercise resulting in more of the byproduct and more PPAR β activation.

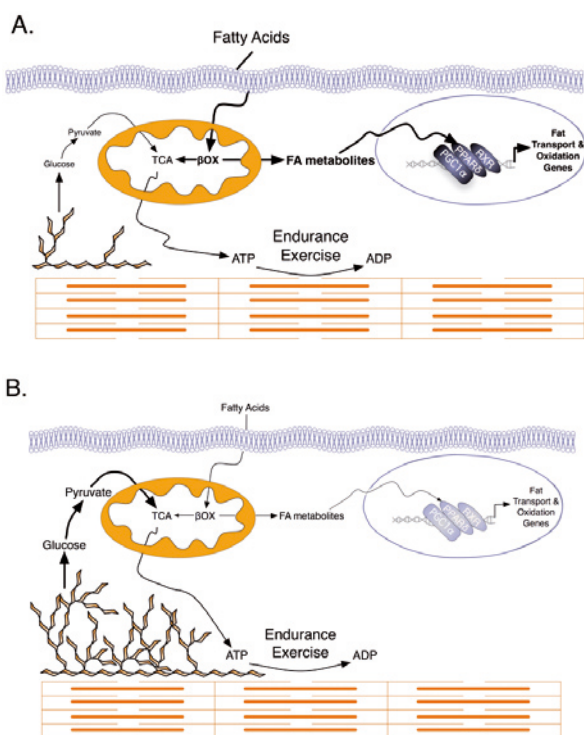


Figure 1

The potential effects of training in low muscle glycogen states on the PPAR transcription factor. A. In the low muscle glycogen state, more fatty acids are available resulting in the activation of PPAR β ; B. In the high muscle glycogen state, a greater proportion of carbohydrates are used resulting in lower PPAR β activation and less adaptation of the fatty acid oxidation enzymes.



How to train in a glycogen-depleted state

If you compete in long duration endurance events, or train athletes who do, a natural question is how do I implement these techniques in my own training? The positive effects of training with low glycogen require glycogen levels to be decreased by about one third that of the normal. This can be accomplished by performing steady state exercise at $\sim 70\%$ of



max for 30 minutes to 1 hour without consuming a CHO supplement. Following the depletion stage, a second session is performed. This session can be performed immediately, or following a fast of 1-3 hours. Ideally, the second session should include high intensity work as this type of training maximally activates the molecular targets that improve endurance performance (Table 1). As with all training techniques, each athlete will have to determine whether training with low glycogen affects their recovery and therefore the overall intensity of their training.

Table 1: Examples of glycogen-depletion training sessions for different sports

Sport	Depletion Session	Adaptive Session
Marathon	1h @ 75% HRmax	6 x 800m at 1 mile pace with 1.5min recovery, or 4 x 1200m at 3K race pace with 3min recovery, or 2 x 2 miles at 10K pace with 10min recovery 1h at 75% HRmax
Road Cycling	1h @ 70% HRmax	6 x 5min at 95% HRmax with 2min recovery 2 x 20min hills @ 80% Wmax
Swimming	20x 150m @ medium-high effort 15 sec rest 30 x 100m @ medium-high effort 15 sec rest	15 x 50m with 10sec recovery, or 10 x 200m with 20sec recovery, or 4 x 400m with 40sec recovery
		All with increasing intensity (1st med – last race pace)
TRIATHLON	4h bike with no supplementation Low CHO dinner	Morning - 3h ride with 3 x 10min @ 90% Wmax, or Morning – 1h run with 2 x 1 mile at 10K pace
Football/Soccer	30min run @75% HRmax	Regular training with team, skills sessions, repeated sprints, ball skills, etc.
Rugby/US Football, Sprinting, Rowing, Time trial cycling	This type of training is not recommended	

Conclusions

Training in a muscle glycogen-depleted state increases an athlete's ability to oxidize fat. In long duration endurance competition this increase in fat oxidation may spare muscle glycogen and improve performance. However, in strength events and endurance events lasting less than 1 hour, where stored ATP, phosphocreatine, and CHO are the primary sources of fuel, there is no performance benefit to training in a muscle glycogen-depleted state.

References

- Bergstrom J, Hermansen L, Hultman E & Saltin B. (1967).** Diet, muscle glycogen and physical performance. *Acta Physiol Scand* 71, 140-150.
- Bergstrom J & Hultman E. (1966).** The effect of exercise on muscle glycogen and electrolytes in normals. *Scand J Clin Lab Invest* 18, 16-20.
- Bergstrom J & Hultman E. (1967a).** A study of the glycogen metabolism during exercise in man. *Scand J Clin Lab Invest* 19, 218-228.
- Bergstrom J & Hultman E. (1967b).** Synthesis of muscle glycogen in man after glucose and fructose infusion. *Acta Med Scand* 182, 93-107.
- BERNARD C. (1858).** Nouvelles recherches expérimentales sur les phénomènes glycogéniques du foie. *Comptes rendus de la Société de biologie* 2, 1-7.
- Blomstrand E & Saltin B. (1999).** Effect of muscle glycogen on glucose, lactate and amino acid metabolism during exercise and recovery in human subjects. *J Physiol* 514 (Pt 1), 293-302.
- Hansen AK, Fischer CP, Plomgaard P, Andersen JL, Saltin B & Pedersen BK. (2005).** Skeletal muscle adaptation: training twice every second day vs. training once daily. *J Appl Physiol* 98, 93-99.
- Hultman E & Bergstrom J. (1967).** Muscle glycogen synthesis in relation to diet studied in normal subjects. *Acta Med Scand* 182, 109-117.
- Narkar VA, Downes M, Yu RT, Emblar E, Wang YX, Banayo E, Mihaylova MM, Nelson MC, Zou Y, Juguilon H, Kang H, Shaw RJ & Evans RM. (2008).** AMPK and PPARdelta agonists are exercise mimetics. *Cell* 134, 405-415.
- Steensberg A, van Hall G, Keller C, Osada T, Schjerling P, Pedersen BK, Saltin B & Febbraio MA. (2002).** Muscle glycogen content and glucose uptake during exercise in humans: influence of prior exercise and dietary manipulation. *J Physiol* 541, 273-281.
- Young FG. (1957).** Claude Bernard and the discovery of glycogen; a century of retrospect. *Br Med J* 1, 1431-1437.

