Betaine, Hyperventilation, Hyperimmune Egg Protein and Other Novelties for Performance at the 2007 ACSM Annual Meeting

Will G Hopkins

This year’s annual meeting of the American College of Sports Medicine was held in New Orleans, May 30 through June 2. The conference consisted of about 2600 presentations on health, injury and performance aspects of physical activity. The total was about 10% down on last year, presumably because people had concerns about post-Katrina New Orleans. They needn’t have worried: everything in the vicinity of the conference and downtown was normal, albeit quiet. I got into a good little hotel, cheaper even than the official student-only hotel. It was only 5 min from the conference center, and I really enjoyed the daily walk through the warehouse district, which had lots of old architecturally interesting industrial buildings still in use.

Once again I consider it an obligation and privilege to write this report in recognition of the hard work of so many people. I have had to limit it to my own research interest of athletic performance. If your interest is health or injury, I strongly advise you to peruse the relevant abstracts. The book of abstracts was distributed only to attendees, but I explain below how you can access each section of the book as a PDF online. Note, however, that there are abstracts only for original-research presentations. Disappointingly, all you get for the symposium, president’s lectures, tutorial lectures and so on–the wisdom of the experts–is titles, speaker names and speaker affiliations.

There seemed to be more missing posters this year than ever before. I know of one PhD student who had a poster accepted then changed his plans and didn’t turn up. The penalty for a no-show is a ban on presenting at the next conference, and whether this applies to all authors on the presentation is not clear, so his supervisor got him to email something basic that could be printed off on sheets of paper and pinned up at the last moment. I was just a co-author…

Many people make the mistake of under-valuing the posters. They don’t realize that somewhere in those aisles you’ll find some of the most exciting new stuff and best-kept secrets. You also get a chance to have a great talk with the authors and anyone else standing in the vicinity. Sure, most of the posters have nothing new, and sometimes you wish the program committee would raise the bar for acceptance. But students and faculty don’t get paid to come unless they present something, and most won’t come if they have to pay for themselves, and it’s important for them to attend this biggest conference in our discipline, so we have to put up with a high proportion of seemingly pointless posters.
One of my colleagues who proofed this article suggested that the program committee could highlight some posters for special attention in the program and on the specific boards in the poster hall. Great idea! It would be wrong to put them into special sessions, because the other poster sessions would be treated even more like a ghetto. The committee already promotes apparently exceptional slide presentations in featured slide sessions, which work well. They also have thematic poster sessions, where the authors have to present their poster verbally. These sessions are less successful, in my experience: I’ve never managed to attend more than the informal start of any of them, because there is no specific scheduled presentation time for each poster and there are too many properly scheduled things on.

There was more genomics this year than ever—25 presentations with the word gene—but I’m losing the excitement I once had for its application to human performance. For me, it’s reached the point that biochemistry reached a few decades ago: instead of assaying enzymes, we’re now assaying mRNA (18 presentations) or its products (13 presentations with blot), and it has about the same marginal utility for those of us interested in performance enhancement. Yes, I know we have to understand how the body works, and I know that knowledge of the genes and gene products involved in the acute and chronic responses to exercise will lead to useful drug therapies for exercise pathologies or the age-related decline in performance, but athletes won’t be allowed to use any that enhance performance of healthy individuals. Talent identification based on performance-related genes might also become routine, but where’s the fun or challenge in that?

As in the past, I have omitted most reports of studies on untrained individuals and all studies on species other than Homo sapiens. Also, I have ignored the authors’ claims that a non-significant effect means no effect. Instead, I have calculated and shown the effect, where possible. I have also shown sample size to help you assess the outcome, be it trivial or substantial. A sample size of 10 in a crossover is usually reasonably definitive for performance, especially if the effect is large. The corresponding sample size for a parallel-groups controlled trial is 20+20 (yes, 4× as many: see Batterham and Hopkins, 2005).

The abstracts and presentations themselves had all the usual statistical errors and other presentation problems that I complain about perennially. These things aren’t improving quickly enough for me. See the introduction in last year’s report (Hopkins, 2006) for a summary. (You won’t, and even if you do, you won’t take any notice. Sigh…) A widespread howler that I haven’t noted previously is this: the authors report a statistically significant effect in the experimental group and a non-significant effect in the control group, therefore there is an effect! No, sorry, but you have to compare the groups properly, via the interaction term or the difference in the changes. See the end of this report for a good example of this bad practice.

Skip this paragraph if you don’t want to access the abstracts on line. You have to be a member of ACSM, or you or your institution must have a subscription to ACSM’s journal, Medicine and Science in Sports and Exercise. ACSM members, log in via this link. Enter your username (default is first 3 letters of your family name followed by your member number) and password (your member number). Click on the MEMBER SERVICES tab, then on the link for Member Journals, then the link for MSSE. Otherwise get to this point at the MSSE site via your institution and/or log in with your own subscription info. Now, click on the main Search tab (not the one in the Quick Search box). In the Title field of the search form, type the presentation number shown [in brackets] in this article, select 2007 to 2007 for the date range, then click SEARCH. You should get one hit, the abstract you want. Some of the hits have a link to a large PDF containing the abstract. There are five PDFs, one each for the featured sessions, slides, clinical case slides, thematic posters, and posters. Strangely, only the first few presentations in each PDF show a link to the PDF. So, if you want the complete PDF for each type of session, put the following word in the Title field of the search form: osteoblasts (for featured sessions—too hard to explain why “osteoblasts” works), 509 (for slides), 975 (for clinical cases), 1174 (for thematic posters), and 1238 (for posters).

I am grateful to the Faculty of Health and Environmental Sciences and the Institute of Sport and Recreation Research NZ of AUT University for funding to attend the conference.
Acute Strategies

Hyperventilation before a swimming time trial improved performance time by an amazing 2.3% over 100 yd and 1.0% over 400 yd in 20 competitive swimmers [1981]. The effect seems to be due at least partly to a reduction in breathing during the swims.

An ankle-length “long-johns” swimsuit might increase 200-m freestyle swimming speed by ~1% [1431], but why did the researchers stop at only 6 competitive swimmers?

Warming up the respiratory muscles improved 200-m swimming time-trial time by 1.2% in 8 elite swimmers [1435]. No details of the usual and respiratory warm-ups are given in the abstract, and I missed the poster. Sorry!

Twelve trained female volleyball players who warmed up with PNF (proprioceptive neuromuscular facilitation) increased jump height by 2.6% relative to no warm-up, whereas two other warm-ups involving jumping produced gains of only 1.3% [1438].

Although the author claimed no significant difference, closer inspection revealed that warm-ups at moderate and high intensity produced substantial improvements of 1.0% and 1.9% in mean power in a 1-min test relative to a warm-up at low intensity in 11 trained male cyclists [2408].

Static stretching impaired the acceleration and maximum speed phases of sprinting in 20 elite female soccer players [1440]. Static or dynamic stretching tended to impair jump height in 12 female volleyball players [1441]. So don’t stretch for volleyball.

Whole-body vibration at various frequencies had little effect on sprint performance of 14 trained track-and-field athletes [1424].

Thirteen high-school baseball players didn’t save time by diving at first base, so take the safer option of running through it [1428].

Continuous ultrasound was more effective than pulsed ultrasound when used in combination with cold packs for the recovery of maximum isometric force in 6+6 healthy subjects [799 & 856; same data]. The authors attributed the effect to better repair of the extra-cellular matrix.

In an unusual investigation of short-term recovery, 22 competitive athletes did two sets of three 30-s cycling sprints (Wingates) morning and again in the afternoon either with a treatment consisting of a cocktail of antioxidant vitamins, ibuprofen, cold-water submersion, and whey protein or in crossover fashion with none of these, as a control [1835]. Performance in the afternoon session was 2-4% better on the cocktail and plasma creatine kinase (a marker of muscle damage) was 9% less the next morning. The author told me he is now investigating each component separately.

Biostatistics

A group that includes the venerable Andy Jackson has re-analyzed a published validity study (on prediction of VO2max) to demonstrate the artifactual bias that I discovered (Hopkins, 2004a) in Bland-Altman plots a few years ago [659]. Folks, well-meaning they may have been, and they have done plenty of other good stuff with stats, but Bland and Altman’s limits of agreement are a naked emperor. It’s time to admit it. Errors of measurement and correlations derived from linear or non-linear measurement models are far better tools to deal with validity and reliability.

“Meta-analyses: appropriate growth or malignant tumor?” was the topic of a conversational forum chaired by Alan Batterham with contributions by Ian Shrier and me. In his introduction, Alan defined a meta-analysis as a review of studies in which there is a quantitative inference about an effect. Most meta-analyses are also systematic in their approach to inclusion of studies. About 1500 meta-analyses were published in 2006 and the rate of increase is currently ~10% per year. I was going to show an updated condensed version of my previous slideshow on meta-analysis (Hopkins, 2004b), but in the end I used a single slide with a working definition of a meta-analyzed estimate and then illustrated the process with data from a meta-analysis on altitude training I am doing with one of my PhDs, Darrell Bonetti.

Although I don’t like the way many meta-analyses are done and reported, I have been reasonably upbeat about them. But Ian made it clear in his slideshow that much of the growth is indeed malignant. He argued that the wide participation encouraged by the Cochrane Collaboration leads to loss of quality, that meta-analysts can easily miss or misinterpret studies, and that even experts can disagree about the clinical importance of meta-analyzed effects. Ian is attacking these issues experimentally by...
getting researchers and clinicians to interpret studies and meta-analyses. A meta-analysis of meta-analyses might also be valuable, if it doesn’t generate meta-misinterpretations and meta-disagreements.

**Nutrition**

The most exciting presentation of the conference for me was on the performance-enhancing effects of betaine from a group at Bill Kraemer’s lab. Few delegates saw the presentation, because it was mis-programmed into a session on hydration rather than ergogenic aids or supplements, and it came right at the end of the last slide session on the last day in the most remote lecture theater of the conference center. Betaine, which I had never heard of, turns out to be a derivative of the amino acid glycine. Plants make and use it apparently to resist dehydration, but in animals it is involved in the synthesis of creatine. The subjects in the double-blind crossover study were 12 men with at least 3 months experience of resistance training. They did one standardized resistance-training session a week to maintain fitness during the supplementation and washout periods, and performance testing was a high-intensity exercise challenge spread over 2 d to test the ability of betaine to maintain performance. After 14 d of supplementation (1.25 g, twice a day), bench-throw power increased by 16% and isometric bench-press force increased by 28% compared with placebo. Other measures of performance did not show “significant” changes. In question time I asked how they thought it might work, but all he could say was that it appears to be used up during exercise–plasma levels fell–which might explain why it didn’t work clearly for the strength-endurance measures that were assessed towards the end of the challenge.

Compared with a 6% carbohydrate drink alone, addition of 1.2% milk-protein hydrolysate produced a 35% increase in time to exhaustion at 80% VO\(_{2}\)max in 10 cyclists and runners, following an 8-km run and a 50-km cycle at 80% and 70% of VO\(_{2}\)max [904]. Doubling the protein reduced the benefit to only 9%. It’s too difficult to work out the effect in terms of mean power output in a time trial, but it’s probably something like 1-2% for the lower dose of protein and negligible for the higher dose.

In a blind crossover of 6 male and 6 female high-level swimmers, a 4:1 carbohydrate-protein supplement in gel form (80+20 g in total) consumed with 500 ml of water immediately before and during 24 100-yd sprints produced a gain in swimming speed of ~3% that was apparent by the fourth sprint and was sustained thereafter [2061]. To convert percent change in swimming speed or time to percent change in power output, I’ve figured out from data in a paper by Toussaint and Hollander (1994) that you have to multiply by a factor of 2. So the equivalent gain in power output is a phenomenal ~6%!! The same supplement in drink form produced a 2.3% enhancement in overall performance time in four 2-km sprints (with 30- to 60-min recoveries) on a cycle ergometer in a blind crossover with 17 aerobically trained subjects [2063]. Again, depending on the ergometer, the gain corresponds to ~5-6% in mean power. I chatted with the principal investigator, John Seifert, who told me that carbohydrate alone was less effective than carbohydrate plus protein in a subsequent study with the swimmers. He also said the carbohydrate-protein gel produced similar impressive enhancement of performance with downhill skiers. He was worried about getting these studies accepted, because they had measured little in the way of potential mechanism variables. I recommended Human Kinetics’ new *International Journal of Sports Physiology and Performance*.

The sample of 5 male and 1 female trained athletes was a bit small, but wow, the gains from recovering on a carbohydrate-protein drink compared with carbohydrate or water placebo were clear. The athletes consumed the drinks in crossover fashion following a 2-h fatiguing ride, then 4 h later rode to exhaustion at 70% VO\(_{2}\)max. Performance time was 55 min on placebo, 78 min on carbohydrate, and 93 min on carbohydrate plus protein [2064]. The 20% difference between carbohydrate and carbohydrate plus protein would correspond to ~1.5% increase in mean power output. In a study with a comparable design, 10 male subjects ran for 8 km and cycled for 50 km consuming either carbohydrate, carbohydrate plus protein, or carbohydrate plus a double dose of protein, then ran to exhaustion [2066]. Data on performance time weren’t provided in the abstract, and I didn’t speak with the presenter to find out, but there was least muscle soreness
and least decline in force of a maximum voluntary contraction 24 h later with the double dose of protein. But drinks containing carbohydrate plus protein or carbohydrate alone had little effect on performance and markers of muscle damage following unaccustomed eccentric exercise (30 min of downhill running) in a randomized blind trial of 18 females [2065].

Here’s something unusual: immunize hens with 26 pathogens, extract hyperimmune egg protein from their eggs, then feed it to 12 recreationally active males while a placebo group receives protein from normal eggs. I don’t know how, but after 10 d the immunized eggs reduced submaximal heart rate by ~5 beats per min and increased 30-s (Wingate) peak power by ~10% [2075]. The analysis even included baseline performance as a covariate! This finding will either disappear without trace or shift a paradigm.

The herbal supplement echinacea continues to show promise as a legal way to raise erythropoietin (by about 70%) and thereby possibly enhance endurance performance via an increase in red-cell mass [908]. The authors must have had a pretty good metabolic cart to get statistical significance for their reported 1.5% increase in VO2max relative to placebo, although they did have a reasonable sample size (12-12 males). They also reported 1.5-2.0% significant improvement in economy, something you don’t expect EPO to affect. In the only other presentation on echinacea, the authors found that the *simulata* species was better than *pallida* at stimulating white cells harvested from the blood of elite wrestlers [2471]. I’m not sure what to conclude from that.

Giving a high-sodium drink to 13 trained female cyclists in the high-hormone phase of their monthly cycle an hour before a constant-load test in the heat resulted in a 26% increase in time to exhaustion relative to a low-sodium drink [729]. The equivalent effect on time-trial power output would be about 2%, because of the relationship between power output and duration (Hopkins et al., 2001).

Caffeine continued to be beneficial at the endurance end of the performance spectrum or for brief bouts affected by fatigue [698-701, 903, 954], as well as for tennis skill [953]. It helped 15 college-age males do more reps with less pain than placebo or aspirin in a resistance-training session, but not without side effects (restlessness, tremor, stomach distress) [1586]. Caffeine may work by reducing muscle pain during exercise, at least in 16 college females, and it reduced pain more in those with less anxiety sensitivity [1217]. Low doses of caffeine (2-3 mg per kg of body mass) with 13 male cyclists could be as effective as the more usual doses (~6 mg.kg-1) in their abstract [701], but the authors reached a different and wrong conclusion in the podium presentation after “protecting the p value”. Ever tried desecrating the p value? It’s liberating.

Gains in strength and power in 8 apparently untrained males training for 12 wk with a commercial protein supplement containing arginine and HMB (hydroxymethylbutyrate) were at least 1.5x greater (on the poster) than in 9 matched males training with a placebo [1587].

Beta-alanine taken as a supplement gets converted in muscle to carnosine, a dipeptide that acts probably as buffer for acidity in intense exercise. It’s best to take it many times a day for at least 4 wk [910]. In randomized blind controlled trials, 6+6 elite cyclists got enhancements in time to exhaustion and isokinetic work [2069—the data aren’t in the abstract and I forgot to get them off the poster], and 11+11 nondescript women got gains in endurance performance tests that I can’t convert to mean power [2072]. I chatted with the leading researcher in this field, Roger Harris, and we worked out that gains in mean power in time trials would be of the order of a few percent.

Quercetin is an anti-inflammatory antioxidant flavonoid found in various fruits and vegetables. It had little apparent effect on various markers of immune function of 20 cyclists taking 1 g.d-1 for 3 wk, but it was a different story with infections of the upper respiratory tract: only 1/20 of the quercetin group got infected, in comparison with 9/20 of a placebo group [784]. It looks like it also reduced or prevented the impairment of cognitive function that occurred in a double-blind controlled trial of 20+20 trained cyclists who did 3 h per day of reasonably intense exercise for 3 d [1247]. On the other hand, 18 runners who took quercetin and 21 who took placebo in the weeks before, during and after a 160-km race showed little difference in various markers of immune function, muscle damage and perceived effort [2476, 2563]. It also had little effect on per-
ceived effort during 3 d of cycling for 3 h.d\(^{-1}\) in a randomized blind trial of 20+20 cyclists [2562]. So quercetin might have some beneficial effects, but apparently not on performance.

Other anti-oxidants featured in several presentations. A 28-wk course of capsules containing powdered fruit and vegetable juice produced lower resting protein carbonyls (a measure of oxidative stress—presumably bad) and TNF-\(\alpha\) (a measure of immune activation—good or bad?) in the blood compared with a placebo in a crossover study of 40 trained men in a special-forces group [1224]. But there was apparently little effect of or effect on a ride to exhaustion at 70% of VO\(_{2}\)max. On the other hand, protein carbonyls did rise following exercise and the concentration was higher at rest and in exercise when the 12 trained males exercised and recovered with a drink containing carbohydrate, protein, vitamin C and vitamin E when the drink contained only carbohydrate [1225]. So, no evidence at this conference for benefits of anti-oxidants on performance.

What about trehalose, a glucose dimer that might transit the gut faster than other sugars? The 6 cyclists improved their time by 4.6% in a 10-km time trial following a 60-min preload when they had ingested a 6.4% drink of either trehalose or glucose compared with water placebo [905]. Again, the gain in power output in a time trial is hard to estimate, but we can assume trehalose is not the next big thing.

Not surprisingly, 9 elite swimmers improved their 200-m freestyle time by 1.6% when they consumed sodium bicarbonate instead of a placebo [1462].

It’s a banned substance in most sports, so it doesn’t do you much good to know that DHEA (dehydroepiandrosterone) reduced muscle pain and a blood marker of muscle damage (creatine kinase) and raised testosterone in badminton players during a 6-d training camp [2581].

Tests and Technology

A delegation from Germany presented six papers on hemoglobin mass: it’s a strong predictor of VO\(_{2}\)max and endurance performance, and it can be measured with sufficient reliability to catch athletes who boost their hemoglobin mass artificially with injections of erythropoietin [520-525].

Polar have devised a new running sensor that clips into your shoelaces and that appears to measure running speed accurately [1421]. It doesn’t account for the extra work on hills, though.

Two commercially available GPS (global positioning system) units are still not good enough for tracking athletes [1203]. Total distance and mean speed seemed to be tracked almost perfectly in another study with one of the devices [1427], but there were no data on tracking of short-term distances and speeds, which is what you would need for monitoring team-sport athletes. Someone used GPS to quantify training of four cross-country runners and related it to their performance over 4 months reasonably well with Banister’s fitness-fatigue exponential model [2225].

From the many posters on body composition [2088-2116] I’ve singled out one that rings alarm bells about the DEXA method: in comparison with the four-compartment model, it appeared to have more than twice the error for quantifying change in percent body fat and similar error but about half the sensitivity for quantifying change in fat-free mass in 8 physically active lean males who detrained over 3 wk [2096]. I put the effects and the \(p\) values from in the abstract into my spreadsheet for confidence limits to make these conclusions.

Here’s a novel use of training data from cycle power meters (mobile ergometers): put the highest mean power in 1 min, 5 min and 20 min into the critical-power model, calculate critical power, then see how well it predicts competitive endurance performance [2409]. It did well, and apparently better than 30-min maximal mean power. The author should try a log-log model, because the critical power model is not appropriate for the submaximal intensity represented by the 20-min maximal mean power (and the critical-power model may be no good anyway—see below). Another reasonably successful novel approach to power-meter training data is to convert the data to Andy Coggan’s normalized power (see last year’s report: Hopkins, 2006), then use that as the training impulse in Banister’s fitness-fatigue model [2415].

An assumption underlying the critical-power model—that the anaerobic work capacity is constant in supramaximal bouts of whatever duration—appears to be invalid, at least for self-paced time trials: it’s bigger for longer time trials, by 50% for a 5-min trial compared with a
1-min trial [2410]. This result is based on the assumption that efficiency is constant, but violation of that assumption would also sink the critical-power model.

**Training**

Priscilla Clarkson gave a great opening president’s lecture on muscle soreness. Main points: we’re still not sure what causes it; acute and chronic stretching don’t help; massage has some effect, but there’s little evidence for benefit of cryotherapy (cooling); NSAIDs work to some extent, but there are side effects; megadoses of vitamin C but not vitamin E reduce it, and other sources of anti-oxidants in fruit and berries might help; but the reactive oxygen species in inflammation have a positive role in adaptation, so, for example, vitamin C delays recovery of strength. She concluded with a call for more attention to individual differences in the response to and treatments for muscle-damaging exercise.

Carl Foster, Romain Meeusen and Jack Raglin brought us up to date with a tutorial lecture on overtraining. We learned that it’s difficult to do original research on this topic, so researchers resort to writing reviews and defining new terms to describe old phenomena. Pushing athletes so hard that their performance starts to fail—defined this year as functional overreaching—is the usual way to prepare for important competitions, because a taper produces supercompensation. When the athlete doesn’t bounce back, it’s now known as non-functional over-reaching. Is that the same as staleness, under-recovery, prolonged maladaptation, and indeed overtraining? Probably, although Meeusen thinks there might be some subtle but as yet unclear differences (see Meeusen et al., 2006, for more). Raglin told us that the Holy Grail (not his words) is a predictor of staleness that is quickly and easily administered without an exhausting exercise test and that gives an immediate result. I would add that it has to be sensitive and specific. We don’t have one as yet, but the closest thing is a downturn in mood state, especially in the depression dimension. Various hormones of the hypothalamic-pituitary axis sometimes show dysfunction in overtraining. For example, the normal increases in growth hormone, adrenocorticotropic hormone (ACTH), and prolactin following exercise are suppressed, especially after a second VO₂max test on the same day. Overtrained athletes also show the same depletion of brain serotonin as patients with post-traumatic stress disorder—hard training takes on a whole new meaning! But it’s impractical to monitor these things, and they tell you whether an athlete is overtrained, not whether an athlete is on the verge to overtraining. Meeusen suggested storing blood samples of athletes when they are not overtrained to help diagnose when they are, but again, once an athlete has failed to recover from hard training, a blood test to demonstrate presence or absence of hormonal of other dysfunction is pointless, in my view. My conclusion: good coaches will know their athletes well enough to read the psychological symptoms of incipient overtraining and will cut back the training load before it’s too late. Training logs that include psychological state might help. But I know of one coach of Olympic medal-winners who effectively uses overtraining to filter out athletes who can’t adapt to the severe training program.

Now, back to the original research… There were several posters on acute effects of hypoxia [2454-2457], but nothing this year on altitude training or other forms of adaptation to hypoxia for sea-level performance.

Eight female rowers who suffered from severe exercise-induced arterial hypoxemia showed improvements of 5.6% in 2000-m time and 2.7% in 5-min distance following 6 wk of inspiratory muscle training, whereas 8 similar rowers who trained as usual showed little change (not apparent in the abstract—you had to see the poster) [1178]. Multiply these gains by 3 to convert them to mean power (Hopkins et al., 2001) and you’ll have to agree that the effect was amazing, if not too good to be entirely true. The study was done in the pre-competition phase (I had to ask the presenter), so there is probably less headroom for gains in competitions. Even so, inspiratory muscle training is bound to work well with any endurance athletes limited by their lungs. It’s simple to diagnose this limitation with a pulse oximeter in a maximal test.

A 14-wk controlled trial of 31 female basketball players randomized to isometric training with and without vibration produced, if anything, better gains in jump performance without vibration [1455].

**Forced repetitions** have now helped another nationally ranked junior powerlifter to
get past a plateau, this year for the squat rather than the bench press [1780; compare with last year’s 1837]. It would be nice to see a study with a sample next year.

Sixteen weeks of traditional weight training with 7+7 junior elite cyclists in what must have been a base training phase had an effect similar to that of usual endurance training on 5-min endurance power (~4%) [2416]. The effect on 45-min power was stated as a “significant” 8% for the weights group, whereas the effect in the control group was “not significant” and no value was stated. But the effect in the control group turned out to be ~7%, when I read it off the graph on the poster! These results fit with other research and reviews showing that traditional weight training has little benefit for endurance athletes (e.g., Paton and Hopkins, 2004), whereas high-resistance interval training produces spectacular gains, even in the competitive phase (Paton and Hopkins, 2005).

View commentary by Stephen Seiler.

References


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