

## Athletic Performance at the 2001 ACSM Meeting

Will G Hopkins

Department of Physiology and School of Physical Education, University of Otago, Dunedin 9001, New Zealand. Email: [will.hopkins@otago.ac.nz](mailto:will.hopkins@otago.ac.nz)

Sportscience 5(2), [sportsci.org/jour/0102/wgh.htm](http://sportsci.org/jour/0102/wgh.htm), 2001 (3919 words)

Reviewed by Stephen Seiler, Institute of Sport, Agder University College, Kristiansand, Norway

[Anthropometry](#): skinfolds are still OK. [Mechanisms](#): sites of fatigue, and non-lactic acidosis. [Muscle Damage](#): benefits of vitamin C, anti-inflammatories, proteases, L-carnitine, and massage. [Nutrition](#): ribose seems to enhance repeated sprints. [Overtraining](#): it's hard to monitor. [Performance Genes](#): we're still waiting. [Tests, Technology](#): something for everyone. [Training, Performance](#): simulated altitude, optimum altitude, better warm-ups, oxygenized water, effective strength training, and tight jumping shorts. [Reviewer's Comment](#): advances in EPO detection, and reflections on a fatigue symposium.

KEYWORDS: altitude, anthropometry, elite athletes, ergogenic aids, nutrition, overtraining, performance enhancement, tests, training

The annual meeting of the American College of Sports Medicine, this year in Baltimore May 30-June 2, was the venue for a total of nearly 2000 presentations of original research, case reports, and review-type lectures in exercise and sports medicine and science. Inevitable clashes in timetabling meant I had to miss many of the talks I was interested in. I even missed key posters, because there were so many and because the poster hall was so far from the talks. By Friday I was suffering from conference shock and nearly took the day off to visit the Smithsonian Institutes in Washington DC. I'm glad I didn't, because Friday turned out to be the best day for me. The other three days were also winners.

This article is a summary of presentations relevant to athlete assessment and sport performance. Mostly I've only summarized the abstracts. My apologies to the authors of good abstracts I've missed on my way through. My apologies also to the experts who gave lectures. Unfortunately the abstracts for these lectures are unusable, either because they are written in the results-will-be-presented manner, or because the presentations for up to five speakers are shoe-horned into one normal-length abstract, or because there is simply no abstract for some types of session. Hence a plea to the ACSM program committee: upgrade and extend the abstracts of these valuable talks by publishing **one information-rich full-length abstract for each speaker**, regardless of the session. Sure, we can buy the tapes, but who has the time, or--if you live outside the US--the money?

In my opinion, many abstracts had serious flaws. Here's my advice on how to improve them in future...

- **Show the magnitude of an effect**, even when the effect is not statistically significant. Our core business is estimating magnitudes of effects, not testing for null effects.
- **Show the precision of your estimate as likely or confidence limits**, which represent the range within which the real (population) value is 95% likely to fall. If instead you use a P value or statistical non/significance, you will mystify most readers and probably misinterpret your own findings.

- **Interpret the magnitude of the effect and its likely limits in plain language.** Is the observed effect trivial, small, moderate or large? How big or small is it likely to be in reality?
- **Conclude "no effect" only when both confidence limits are trivial.** Lack of statistical significance by itself is not a sufficient reason to conclude little or no effect. To put it another way, failure to reject the null hypothesis is not a sufficient reason to accept the null hypothesis. Generations have misunderstood this point.
- **Show standard deviations.** These convey the useful impression of variation within groups. The reader can also gauge magnitude of a difference or change in the mean as a fraction or multiple of the within-group standard deviation(s).
- **Do not show standard errors of the mean.** These convey the less useful impression of statistical significance, and only in the special case of non-repeated measures with equal-sized groups. Standard errors do NOT convey statistical significance in controlled trials.
- **For athletes, show performance effects as percent changes or differences,** because the smallest effects that matter to athletes are best expressed as percents (~0.5-2%, depending on the sport and measure of performance).
- **Avoid abbreviations** for everything except units of measurement. Abbreviations almost always make anything hard to read. A keystroke, word, or space limit is no excuse.
- **Don't submit two abstracts when one is appropriate.** This advice is ACSM policy. The meeting would benefit from fewer abstracts.

In what follows, the # sign indicates the number of the Abstract in Medicine and Science in Sports and Exercise, Volume 31, Supplement 5. You may have trouble tracking down the supplement, which this year was distributed only to conference attendees. At the time of writing, you can still view most abstracts at the [FASEB-ACSM](#) site.

### Anthropometry

Hydration status of **adult males and females** substantially affected percent fat determined by **bioelectric impedance** and by **near-infrared interactance** but not by **skinfolds** (#86).

A Tanita **bioelectric impedance** device trailed the field, but there was nothing to choose between **hydrostatic weighing**, **skinfolds**, and **dual X-ray absorptiometry** (DXA) for assessment of percent fat in **collegiate athletes**, relative to the criterion multi-compartment method (#1365).

There were many other anthropometric **methods** papers. See #77-88 and #1349-1370.

### Mechanisms

A team from Cape Town headed by Tim Noakes gave a mini-symposium on **fatigue** entitled "the ATP paradox, or why muscles do not develop rigor during exercise" (#537). Their idea is that depletion of energy (adenosine triphosphate, ATP) in muscle fibers during hard exercise would produce something akin to rigor mortis. Noakes didn't say why that would be bad, but I guess that having even a few stiff fibers scattered through a muscle would compromise muscle function and/or cause muscle damage during contraction. It follows that fatigue could be a protective mechanism to stop us pushing muscle fibers to the point of stiffness. The speakers made a case for the central nervous system as the site of this fatigue: they claimed that we can't voluntarily recruit all fibers in a muscle, even in all-out exercise. They also claimed that metabolic changes in active muscle modify recruitment of fibers via the central nervous system but do not produce performance-limiting fatigue in the muscle itself. These claims are debatable, depending on the type of exercise. In any case, there must also be a stiffness-limiting protective

fatigue mechanism within muscle fibers, because "no previous study has demonstrated... [that] muscle rigor occurs in human subjects... *even when their muscles are stimulated electrically*" (cited from the abstract, italics mine). The nature and relative importance of central-fatigue and muscle-fatigue mechanisms in various forms of exercise will be important future presentations from the Cape Town team.

Most **subjects** (#260) and most healthy, young, **active subjects** (#262) showed a **plateau in maximum oxygen consumption**, which is consistent with the idea that oxygen transport to the active muscles is the limiting factor in endurance for such subjects.

The **effect of caffeine** on performance of isometric contractions to fatigue appears to be mediated via cortical excitability in **male subjects** (#934). That's evidence for fatigue in the central nervous system.

**Speed skaters fatigue** faster than comparable **cyclists**, probably because the crouched position in speed skating reduces blood flow to active muscles (#1342).

Changes in pH in a test tube (#1565) appear to support the view that lactic acid does not contribute directly to the increase in **acidity** in muscle cells during **intense exercise**. Do the hydrogen ions come from breakdown of ATP or are they released early on in the pathway of the breakdown of glucose to lactate? Whatever, we don't produce lactic acid--we produce hydrogen ions and lactate ions. So, should we refer to metabolic acidosis rather than to lactic acidosis? Probably. For details, see the [article](#) in this issue of SportsScience by Rob Robergs.

### Muscle or Tissue Damage

Megadosing (3 g/d) with **vitamin C** before eccentric arm exercise in **untrained subjects** reduced the perception of soreness by an unstated amount and tended to reduce serum concentration of muscle creatine kinase, a marker for muscle damage (#694). Similarly, 9 d of 800 mg/d of vitamin C nearly halved serum myoglobin (another marker of muscle damage) after unaccustomed downhill running in **physically active males**, but claims for less loss of muscle function were not backed up with data (#695).

The anti-inflammatories (pain killers) **Ibuprofen** and **Bromelain** had "no effect" on pain or function following unaccustomed eccentric elbow flexion in an apparently high-powered study of untrained (?) **subjects** (20 males, 20 females), but the design (crossover?) and effects were not stated, and the analysis (MANOVA) was underpowered (#696). **Vicoprofen** may have done a bit better than **Ibuprofen** for several measures of damage and function in the first 2 d following eccentric exercise in healthy men (#1110; not enough data in the abstract). **Celecoxib** was a bit better than **Ibuprofen** and placebo for physician-assessed recovery of sprained ankles over 11 d in a cross-sectional study of 443 **adults** (#1109). The same researchers found similar effects for **Celecoxib** and **Naproxen** on ankle sprain (#1121).

Supplementation with pancreatic **proteases** four times a day over 4 d before and after downhill running reduced pain and loss of function in leg muscles of **males** (#697). The abstract featured detail of methods but no magnitudes of effects.

**L-carnitine** supplementation for 3 wk before "intense hypoxic exercise" (a series of squats) reduced pain after exercise by a worthwhile notch on a pain scale, and it reduced plasma myoglobin by an unstated amount compared with placebo in a 1-wk washout crossover study of healthy **active men** (#698). Possible mechanism: reduction in oxidative stress (#1233).

**Massage** 2 h after eccentric exercise of the hamstrings probably reduced the perception of soreness and may have affected muscle function of **subjects**, but without data in the abstract it's hard to say (#699). In a similar study of mostly **athletes** and active adults,

massage increased range of motion moderately (effect size ~0.8); there was probably also a modest effect on strength (no data) and "muscle tissue energy absorption" (?), but not pain (#1103).

There was a trend towards a small sparing of muscle-protein breakdown in **endurance runners** on a **high-protein vs moderate-protein diet** (#919).

There weren't enough data for me to adequately assess an apparent trend towards less acute exercise-induced muscle damage in **male subjects** supplementing with **cracked pine pollen** (#923).

## Nutrition

Compared with placebo, **caffeine** enhanced performance lasting several hours on a cycle ergometer when the **competitive cyclists** took it as pills before the test, as pills during the test, and as Coca-Cola during the test (#249). The effect, obscured partly by abbreviations, was ~3% for mean power in a 30-min time trial following a 2-h pre-load. On the other hand, caffeine apparently (no data) had no effect on performance of sprints or on overall time in a 100-km performance test with **elite cyclists** (#944).

Supplementation for 11 d with **ribose** (the 5-carbon sugar that forms part of ATP and all other nucleic acids) may enhance performance of multiple sprints repeated over several days by as much as 5% (#251) in **healthy subjects**, but it's very expensive stuff. Possible mechanism: it partially reduced the fall in intramuscular adenine nucleotide concentration that occurred over 5 d of hard training (#943). Relative to placebo, ribose also produced a 22% increase in reps in 10 sets to failure following 4 wk of supplementation combined with heavy resistance training in **body builders** (#938); data on changes in body composition by DEXA were not reported, because--you guessed it--they weren't statistically significant.

It's reasonably clear that a **high-fat diet** followed by **carbohydrate loading** is better than a **high-carbohydrate diet** for **ultra-endurance**. In a crossover study (#291), seven **competitive cyclists** had either 6 d of high fat and 1 d of high carbohydrate or 7 d of high carbohydrate. On fat with carbo-loading they went 5% further (equivalent to an 11% increase in mean power) in a 1-h time trial on a cycle ergometer after 4 h at moderate intensity. The conditions (breakfast, supplements, exercise intensities) were more realistic than in previous studies, and a P value of 0.11 for the effect is equivalent to chances of 94.5% that the true effect was positive. The mechanism? Probably glycogen sparing resulting from fat adaptation, coupled with the extra glycogen from the day of carbo-loading, because oxidation of fat during the performance test was still high after fat with carbo-loading (#292).

The herbal-based supplement **Cordymax** increased fat oxidation in **endurance athletes** but apparently (no data!) had no clear-cut effect on maximum oxygen consumption (#928).

Supplementation with **sodium phosphate** (4 g/d for 4 d) apparently had "no effect" on a Wingate sprint or maximum oxygen consumption in a crossover with 12 **trained male cyclists**, but without data in the abstracts...? (#929, 936).

**Amino-acid** supplementation appeared to reduce effects of short-term resistance overloading on testosterone and hemoglobin indices in **resistance-trained men** (#1905, no data).

Highlights of a zillion abstracts (#1142-1166) on the short-term effects of **creatine** supplementation on performance: almost twice the effect in females as in males (#1152), and worthwhile effects in soccer (#1149) and squash (#1151) players. There were no

studies of effects of chronic supplementation on performance. I've ignored the substantial number of abstracts that did not include sufficient data.

In an abstract made virtually impenetrable with abbreviations, 60 d of supplementing with **bovine colostrum** vs a whey-protein control during upper-limb resistance training in **unspecified subjects** produced some hypertrophy, but it was in skin or other non-contractile tissue (#1913).

## Overtraining

**Sleeping heart rate** seemed to have little relationship to training load, but whether the four **runners** got near to overtraining is not clear (#750).

Serum **prolactin**, **catecholamine excretion**, and nightly and morning **heart rate** monitored 4 weeks before, 4 weeks during, and 8 weeks after a training camp aimed at overtraining 11 **cyclists and triathletes** did not give a consistent picture of the increased workload (#1621, 1623). But again, was anyone overtrained?

It's hard to make generalizations about the relationships between **blood tests**, **training loads**, and **performance** in 12 **highly trained swimmers** (#1920). It looks like you have to get to know your individual athletes when you monitor for overtraining.

## Performance Genes

The gene for angiotensin converting enzyme--the **ACE gene**--has been a candidate for a performance gene, but now it's pretty-much down the gurgler. There were weak (?) associations of ACE-gene forms with muscularity in a comparison of **elite body builders** with controls (#1809, no usable data), and an apparent (weak?) association with **endurance** in Japanese athletes was different from that in other studies (#1810, no data). One of my students didn't find any obvious association between ACE genotype and response to altitude exposure in a small sample of **runners** (#11).

A session on genetic aspects of performance didn't include any breakthroughs (#1294-1298). There wasn't a consistent effect of the two forms of the **gene for ciliary neurotrophic factor** on **strength training** in arms and legs of healthy adult **males** (#1560). Forms of the gene for the most abundant **mitochondrial protein** were associated (weakly?) with **maximum oxygen consumption** in **blacks** but not **whites** (#1813, no data). A mutant form of the gene for **creatine kinase** was associated (weakly?) with lower **maximum oxygen consumption** training response in **blacks**, but the response in **whites** was, if anything, the opposite: reduced oxygen consumption at a submaximal workload following training (#1814, no data).

Conclusion: **no performance gene yet**.

## Tests, Technology

If your **cycle ergometer** has a flywheel, its inertia substantially attenuates peak power in Wingate tests (#1856). You can correct for it.

Profiling is a bit passé, but if you want to know how people are using or refining **tests on athletes**, see #133 (cycling), #883-904 (cycling, softball, soccer, tennis, football, swimming), #1372-1394 (volleyball, basketball, rugby, ice hockey, surfing, rowing, soccer, triathlon, running, football, rock climbing, BMX, cheerleading!), #1921-1927 (motor racing, cycling, canoeing, hockey, surfing, soccer).

See the poster session on **validity and reliability** for potentially useful stuff on sit-and-reach (#1686), pulmonary diffusion capacity (#1688), using a 3-L syringe to calibrate metabolic carts (#1689), cardiac output (#1690), the Cosmed portable metabolic system (#1691), jumps vs shuttle runs (#1692), 1RM vs 3RM (#1693), a lumbar extension

dynamometer (#1694), knee-extension test (#1696), swimming lactate threshold (#1698), a shuttle test (#1700), and an aerobic dance test (#1702).

There was no room to put it in the abstract, so here's the main point I made in my part of the mini-symposium on **reliability**: the typical (standard) error of measurement has to be similar in magnitude to the smallest clinically or practically important change in the measurement, if you want to track such changes in individuals or in studies with modest sample sizes (#983; [download](#) my PowerPoint presentation). Other points from the session: Andy Jackson talked about averaging several trials to improve reliability, and Greg Atkinson told us to watch out for differences in reliability between subjects.

The typical **variation in performance time** of **elite cyclists** from race to race is ~0.5% in the 1-km sprint and ~1.3% in road time trials of 46-75 km (#964). Divide these by ~2 to get the smallest worthwhile performance effect, then multiply by ~2 to convert time to mean power. So, you're chasing changes of 0.5-1.5% in mean power when you test top cyclists.

## Training, Performance

Eleven **runners** sleeping and resting in an **altitude tent** for an average of 8-11 h/d for 4 wk experienced on average an enhancement of performance in a ~5-min run to exhaustion that was the equivalent of ~1.5% in a time trial (#11). The effect wasn't exactly clear-cut, though.

In an unusual semi-crossover controlled study (#1634), 19 **cyclists** slept for 8-10 hours a night in an **altitude house** for 5, 10 or 15 d before performing a 4-min supramaximal cycling test at low altitude. Enhancement was similar for the three durations of exposure and averaged 2.3% in mean power relative to when cyclists in each group did their control training. (The 2.3% is probably contaminated partly by a practice effect.) There was also a whopping 13% relative increase in maximum accumulated oxygen deficit in the test, suggesting the performance enhancement was mediated primarily by a change in anaerobic capacity. So only 5 d in an altitude house or tent is enough? Or is it all just a placebo effect?

In a study of the effect of **different altitudes** on sea-level performance, 48 **runners** living high for 4 wk at 1780, 2085, 2454 or 2805 m while training high-low at 1250-3000 m experienced gains of 1.1, 2.8, 2.7, and 1.4% in 3000-m running time (#1642). Gains in maximum oxygen consumption were somewhat correlated. Conclusion: optimum altitude for live-high train-low is 2000-2500 m.

Four out of five male **high jumpers** who were undertwisted at the peak of the jump apparently needed to increase their "catting" (#572), whereas four out of five undertwisted females apparently needed a more vertical position at the takeoff. This practical application of **kinematics** needs following up with an intervention.

One set of reps per session in a 10-wk **strength-training** program with previously **untrained subjects** produced a 22% increase in 1RM strength, whereas three sets produced a 31% increase. The authors misinterpreted statistical non-significance as equal effectiveness (#435). In subjects with up to 2 y weight-training experience, there was "no significant difference" in the effects of three sets of two reps at 90% 1RM vs a traditional 8-rep program of three sets at 65, 70 and 75% 1RM, but without data... (#1827).

Two bouts of **high-speed running** (apparently between 1500-m and 3000-m pace for a few minutes) on a treadmill twice a week for 4 wk improved 3000-m time-trial time by about 3% in **highly trained runners**. We don't know what happened in a control group or in a group who ran at the same intensity for slightly longer, because these groups had "no significant improvement" (#748). And there was no comparison of groups.

In **collegiate footballers**, a 10-wk program of **single sets of high-intensity resistance training** (unstated weekly frequency) produced a greater increase in time to exhaustion in upper- and lower-body isokinetic exercise than a traditional multi-set program (#756). There weren't enough data in the abstract for me to estimate the difference meaningfully.

**Active recovery** between high-intensity bouts had such a positive effect on performance of total work in a Wingate test that the difference (~7%) relative to the usual passive recovery was clear cut with the sample of only three **ice-hockey players** (#780).

The optimum **rest interval** for gains in bench-press strength in **recreational weight trainers** appears to be 4-6 min (#1828, 1829).

Doubling the usual duration of the morning **warm-up** slowed time-trial time of age-group **competitive swimmers** by 1.9%, whereas cutting the afternoon warm-up to one-third of normal had little effect (#893).

Replacing the last 5 min of the usual aerobic **warm-up** with high-intensity sprints resulted in 4.6% higher peak power and 2.0% higher mean power in a 2-min kayak ergometer sprint test with experienced male **paddlers** (#1916). Spectacular!

High-load low-rep maximal-effort **strength training** made an improvement in running economy in **soccer** players that should amount to a 4-5% enhancement in endurance performance (#1529). Wow! This paper is consistent with other recent publications on the benefits of high-intensity resistance training for endurance athletes.

Here's an unusual potential measure for **talent identification**: concentration of various intramuscular phosphates, as determined by magnetic resonance spectroscopy. In **junior ice speed skaters**, it predicted capacity for sprint performance 6 y later (#837).

The longer the **running** distance, the bigger the difference in world-record performance between **males and females**: 7% for the 100 m through 19% for 200 km (#902). Reviewer's note: these competitive performance data certainly do not support theories and limited laboratory tests suggesting greater fatigue resistance in females.

Does drinking **oxygenized water** (water saturated with oxygen gas) really enhance endurance performance (#945)? You can't get nearly enough oxygen into water to account directly for the higher arterial saturation (91% vs 87%) the authors observed at the end of the constant-load test to exhaustion on a cycle ergometer in this double-blind crossover study of 20 male and female **regular exercisers**. There was even a significant effect on performance in an incremental test for the fitter subjects (2.6% for performance time, which would convert to less for peak power), although that kind of post-hocery needs following up with another study. Again, the dissolved oxygen can't account directly for the effect. The findings mystified the authors.

And finally, tight **neoprene-butyl rubber** shorts enhanced jump performance in 10 female and 10 male **varsity sprinters and jumpers** (#1340; no data). Energy storage during the counter-movement phase of the jump?

### Reviewer's Comment

I was also in Baltimore for the ACSM meeting. A recurring theme during lectures and lively, lunch-time discussions--complete with laptop presentations of data!--was the increasing sophistication and effectiveness of **erythropoietin (EPO) testing**. International cooperation is paving the way. By the 2002 Winter Olympics, multi-factorial blood-profile testing will be in place. If you are using EPO, they will get you. More importantly, if you *were* using EPO, they'll *still* get you! New banned substances will continue to appear, but in many cases, a test will be developed even before they hit the market. Nice work, guys!

It was standing room only for the mini-symposium on **fatigue** (the ATP Paradox...) by Tim Noakes, Zig Gibson and Vicky Lambert (#537). The talks were entertaining and at times thought provoking. Questions raised about how the CNS determines and carries out accurate pacing strategies at the start of exercise were quite captivating. However, the apparent attempt to force a paradigm shift from peripheral contractile fatigue mechanisms to central mechanisms was weak. A lot more research needs to be done before we reinterpret a great deal of existing work supporting the importance of peripheral fatigue mechanisms. --Stephen Seiler

Webmastered by Will Hopkins.  
Published Sept 2001.  
[editor@sportsci.org](mailto:editor@sportsci.org)  
©2001