

## ***Lactate threshold: its significance and determination via field-test***

BY CHARLES HOWE

Imagine yourself on the starting line of an auto race. All cars have identical aerodynamic characteristics (i.e., the same body), so you're wondering what each one has "under the hood." Now, suppose your own engine is the most powerful in the race – it can reach 500 horsepower – but there's just one problem: when you try to sustain more than 80% of those 500 horses for a few minutes, a sort of governor kicks in that makes it cut back until various engine conditions reach a particular operating range. Your principal opponent, on the other hand, has an engine that can only reach 475 hp maximum – 5% less than yours – but *his* governor kicks in at 90% of that, or 428 hp – a full 7% more than you can maintain on a prolonged basis.

The horsepower-governor analogy helps illustrate the relationship between the central and peripheral determinants of endurance performance, i.e., maximal oxygen uptake ( $VO_{2max}$ ), and lactate threshold (LT), respectively:

1. the first sets the upper limit of ATP (adenosine triphosphate) production via aerobic metabolism, and is determined primarily by cardiac output (CO), the ability of the heart to pump oxygen-carrying blood to the working muscles; CO in turn depends on cardiac stroke volume (SV):  $CO = SV \times HR$  (the Fick equation); ATP is the sole and direct source of energy used by working muscles
2. the latter determines the percentage of  $VO_{2max}$  that can be utilized for an extended period (6 minutes or longer), and correlates with the density of mitochondria (the locus of aerobic ATP production) within the working muscles, as well as the extent of capillarization present, two adaptations that depend largely on years of specific endurance training at an adequate intensity

$VO_{2max}$  is determined in a lab by analyzing expired gasses during a "ramped" test on a calibrated ergometer cycle, wherein increasing work loads are imposed in a specific protocol until exhaustion is reached. LT is determined along the way, prior to exhaustion, through analysis of blood samples, with the ratio of expired  $CO_2$  to inspired  $O_2$  (known as respiratory exchange ratio, or RER) being used as a check. On a practical level, the importance of LT is primarily in setting training levels (such as [these](#)), as well as gauging pace during intervals and time trials, however, it need not be determined in a lab; average power over a 40-60 minute test TT, or "functional threshold power" (FTP), serves well as a "proxy" since it correlates very closely with  $VO_2$  at LT (although *power* at LT, as determined in the lab, will be somewhat lower than FTP). Such a test gives a "bottom line" measure that integrates *all* physiological determinants of endurance performance ( $VO_{2max}$ , LT, and efficiency), and so is an excellent practical alternative to a ramped lab test.

The drawback to functional field testing is that it is self-administered, rather than carried out under the watchful eye of a coach or exercise physiologist in a lab or other controlled setting, and can therefore be affected by environmental conditions, the motivation and concentration of the test subject, as well as (to a lesser extent) his or her judgment and skill in pacing correctly. For consistent and reliable test results, make sure you are adequately rested, with no illness or infection present, while avoiding extremes of temperature (especially heat) and wind. Flat terrain is recommended, but a steady, continuous uphill grade can also be used, and even a rolling to hilly course will do if the same one is used each time (average power on a rolling/hilly course, or in variably windy conditions, will be somewhat less than for a windless, constant-grade test of similar duration). It may take a few attempts to get the pacing just right and the wattage "dialed in" ([Figure 1](#)), but once it is, average power achieved in any carefully executed threshold test should be highly repeatable from day-to-day.

Wide and rapid variation in the energy demands of road cycling has led some, such as Dr. Arnie Baker, M.D., in *Smart Cycling*, to conceive of it as an anaerobic sport, but this is contradicted by what is already known:

1. most energy for a single *maximal* effort over 70 seconds, starting from a *rested* state, comes from *aerobic* sources ([Figure 2](#))
2. in four 30 second bouts of exercise, each separated by *complete* recovery, most of the energy utilized by the third bout comes from *aerobic* sources ([Figure 3](#)), and the predominance of aerobic metabolism becomes even more pronounced during longer exercise bouts ([Figure 4](#)), not to mention *continuous* exercise, such as any road race, where intensity is lower, and recovery is not nearly as complete

3. the extent to which anaerobic energy sources are taxed (and blood lactate is produced) for a given set of race demands will be determined by how much and often threshold power is exceeded, therefore, the higher it is, the less they will be called upon, while the more often it is exceeded, the more anaerobic sources are taxed. Furthermore, within the context of any road (i.e., endurance) event, how quickly one recovers from short, intense efforts is actually more reflective of aerobic, not anaerobic fitness, since 1) fatigue during intense exercise is related to changes in high energy phosphate (ATP) levels, 2) 100% of ATP resynthesis within working muscle occurs via aerobic metabolism, and 3) the rate of ATP resynthesis is correlated with mitochondrial respiratory capacity
4. racing categories and time trial performance both correlate much more highly with sustainable threshold power than with anaerobic capacity or sprinting power.

Thus, it often goes unrecognized that greater aerobic ability (in particular, a high mitochondrial density within muscle) enhances the ability to sprint or attack in almost all race situations. The reason is that short-term power production is reduced when the effort is initiated from prior exercise (as opposed to from rest), and this reduction is in direct proportion to high-energy phosphate levels within the muscle. In other words, the really “strong” riders seem to be able to attack repeatedly, or when the pace is already very high, and then recover more quickly than others, largely because their muscles are more *aerobically* fit, **not** because they have markedly greater “lactate tolerance,” and despite the seeming importance of sprinting ability in determining race outcome, it is more the case that the sprinter with the highest threshold power wins. Adam Myerson, a pro/elite-level field sprinter, summed it up nicely by noting that sprinting ability may be what helps you win the game (race), but having a high threshold power is what allows you to play the game in the first place, and influences how well you can play at the end. Another insightful comment was recently offered by Andy Birko at [Wattage Forum](#):

*“When rested, I’ve got a pretty decent sprint (for a Cat. 4) at around 1100 Watts or so. When I hide, suck wheel, etc., in a long race, I can produce about 800 Watts or so in the final sprint. When I’m pulling, chasing etc., I’m lucky if I can hit 650 Watts by the end. There’s another guy in my club whose sprint speed is about the same as mine (I don’t know his power), and when we do sprint drills, the result is split about 50/50. When we do our monthly time trial, he goes about 10 seconds faster on his road bike than I do on my TT bike. Guess who beats whom more often when we do our training races.*”

*Anaerobic capacity is like a bank – every time you go over LT, you’re drawing from the bank, and again, the further and longer you go above LT, the quicker you’re withdrawing. You can only replenish the bank when below LT, so recovery from anaerobic efforts is directly related to how often and how much you go above LT, as well as long you stay there, so the higher your LT, the stronger your anaerobic efforts can be without draining the bank as much.”*

This interpretation is spot-on, and is essentially the same as what was presented in a [recent study](#) in the *Journal of Applied Physiology*. It explains why “genetic sprinters” need to be careful to “conserve their sprint” throughout most any road race; as Jim Martin (masters national match sprint champion, but only a Cat. 3 on the road) describes it: “I often to spend the whole race sitting in and suffering, waiting for the 1 km to go sign.” Further, world and Olympic match sprint champion Marty Nothstein is generally unable to contest for the win once the ‘smack’ really starts to go down in national-level points races and Madisons on the track, as well as road criteriums; despite his world-class sprint ability, and even though he has greatly improved his aerobic ability (threshold power), as evidenced by his win at the 2003 New York City Championship (a 100 km criterium), it still apparently is not enough to handle the repeated surges thrown at him by riders such as Colby Pearce, Jame Carney, etc. Even for a points racer, most training time should be spent working on threshold power, although the new rule awarding 20 points – but no other benefit – for lapping the field tends to tip the balance a bit more towards those who can sprint well.

Or in other words, it’s an **aerobic** sport, damn it! ☺

*Charles Howe is neither an exercise physiologist nor a USA Cycling<sup>TM</sup> licensed coach, but you can learn a fair amount by hanging around the right people. Special thanks to Andrew Coggan, Ph.D., for his contributions to this article.*

Illustrations

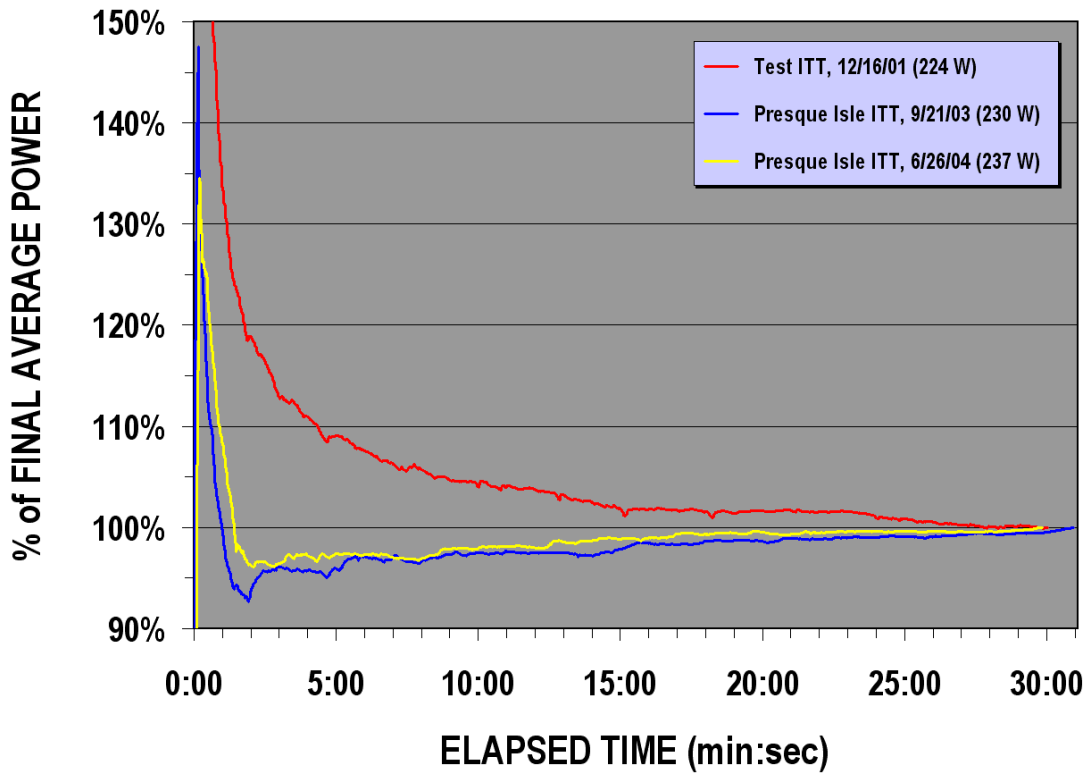


Figure 1. Running average power as a percentage of final average in three flat-terrain time trials.

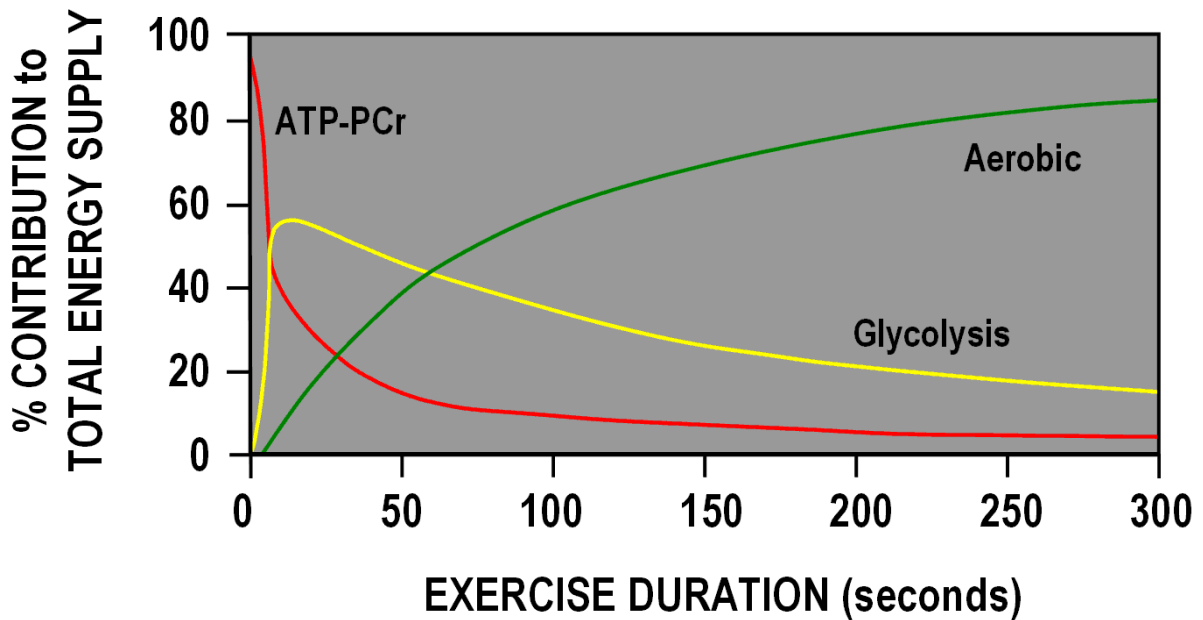


Figure 2. Relative contribution of three energy systems to exercise (Gastin, 2001).

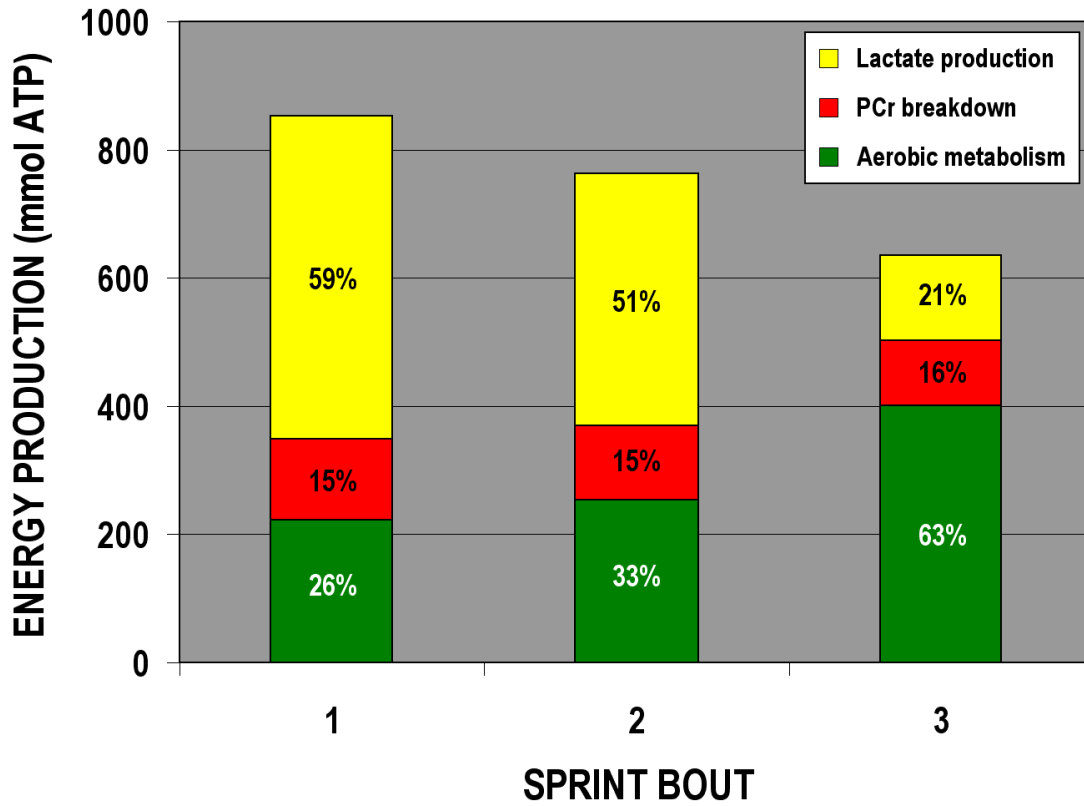


Figure 3. Contribution of three energy systems to three 30-second maximal exercise bouts separated by 4 minutes complete rest (Putman, 1995).

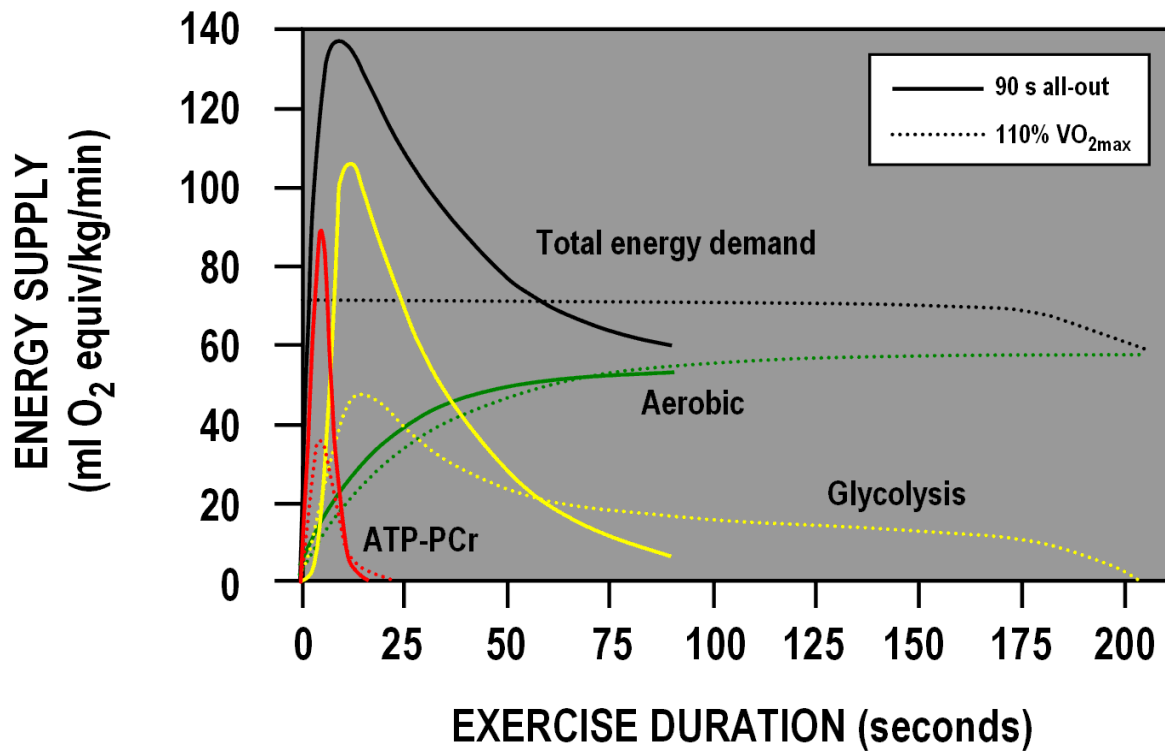


Figure 4. Contribution of three energy systems at two exercise intensities (Gastin, 2001.)

## Bibliography

- Coggan, A.R., and D.L. Costill. Biological and technological variability of three anaerobic ergometer tests. *International Journal of Sports Medicine* 5(3):142-5, June 1984.
- Coyle, E.F., A.R. Coggan, M.K. Hopper, and T.J. Walters. Determinants of endurance in well trained cyclists. *Journal of Applied Physiology* 64(56):2622-30, June 1988.
- Gastin, P.B. Energy system interaction and relative contribution during maximal exercise. *Sports Medicine* 31(10):725-41, 2001.
- Hickey, M.S., D.L. Costill, G.K. McConell, J.J. Widrick, and H. Tanaka. Day to day variation in time trial cycling performance. *International Journal of Sports Medicine* 13(6):467-70, August 1992.
- Mattern, C.O., R.W. Kenefick, R. Kertzer, and T.J. Quinn. Impact of starting strategy on cycling performance. *International Journal of Sports Medicine* 22(5):350-5, July 2001.
- Mujika, I., and S. Padilla. Physiological and performance characteristics of male professional road cyclists. *Sports Medicine* 31(7):479-87, 2001.
- Putman, C.T., et al. Skeletal muscle pyruvate dehydrogenase activity during maximal exercise in humans. *American Journal of Physiology* 269(3 Pt. 1):E458-68, September 1995.
- Shakeshaft, M. Developing a Pacing Strategy For The Ten Mile Time Trial. Internet article posted at <http://freespace.virgin.net/martin.shakeshaft/10tt.htm>
- Spriet, L.L., M.I. Lindinger, R.S. McKelvie, G.J. Heigenhauser, and N.L. Jones. Muscle glycogenolysis and H<sup>+</sup> concentration during maximal intermittent cycling. *Journal of Applied Physiology* 66(1):8-13, January 1989.
- Swain, D.P. A model for optimizing cycling performance by varying power on hills and in wind. *Medicine and Science in Sports and Exercise* 29(8):1104-8, August 1997.
- Swensen, T.C., C.R. Harnish, L. Beitman, and B.A. Keller. Noninvasive estimation of the maximal lactate steady state in trained cyclists. *Medicine and Science in Sports and Exercise* 31(5):742-6, May 1999.
- Thomas, C., P. Sirvent, S. Perrey, E. Raynaud, and J. Mercier. Relationships between maximal muscle oxidative capacity and blood lactate removal after supramaximal exercise and fatigue indexes in humans. *Journal of Applied Physiology* 97(6):2132-8, December 2004.
- Trump, M.E., G.J. Heigenhauser, C.T. Putman, and L.L. Spriet. Importance of muscle phosphocreatine during intermittent maximal cycling. *Journal of Applied Physiology* 80(15):1574-80, May 1996.
- Race pacing. Unsigned article at <http://www.pponline.co.uk/encyc/0972.htm>.
- Barstow, T.J., S. Buchthal, S. Zanconato, and D.M. Cooper. Muscle energetics and pulmonary oxygen uptake kinetics during moderate exercise. *Journal of Applied Physiology* 77(4):1742-9, October 1994.
- Barstow, T.J., S.D. Buchthal, S. Zanconato, and D.M. Cooper. Changes in potential controllers of human skeletal muscle respiration during incremental calf exercise. *Journal of Applied Physiology* 77(5):2169-76, November 1994.
- Bendahan, D., G.J. Kemp, M. Roussel, Y.L. Fur, and P.J. Cozzone. ATP synthesis and proton handling in muscle during short periods of exercise and subsequent recovery. *Journal of Applied Physiology* 94(6):2391-7, June 2003.
- Bogdanis, G.C., M.E. Nevill, L.H. Boobis, H.K. Lakomy, and A.M. Nevill. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *Journal of Physiology* 482.2:467-80, 15 January 1995.
- Bogdanis, G.C., M.E. Nevill, L.H. Boobis, and H.K. Lakomy. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *Journal of Applied Physiology* 80(3):876-84, March 1996.
- Cooke, S.R., S.R. Petersen, and H.A. Quinney. The influence of maximal aerobic power on recovery of skeletal muscle following anaerobic exercise. *European Journal of Applied and Occupational Physiology* 75(6):512-9, 1997.
- Guthrie, B.M., S.P. Frostick, J. Goodman, D.J. Mikulis, M.J. Plyley, and K.W. Marshall. Endurance-trained and untrained skeletal muscle bioenergetics observed with magnetic resonance spectroscopy. *Canadian Journal of Applied and Occupational Physiology* 21(4):251-63, August 1996.
- Hogan, M.C., R.S. Richardson, and L.J. Haseler. Human muscle performance and PCr hydrolysis with varied inspired oxygen fractions: a <sup>31</sup>P-MRS study. *Journal of Applied Physiology* 86(4):1367-73, April 1999.

- Kuno, S.Y., M. Akisada, and F. Mitsumori. Phosphorus-31 nuclear magnetic resonance study on the effects of endurance training in rat skeletal muscle. *European Journal of Applied and Occupational Physiology* 65(2):197-201, 1992.
- Johansen, L., and B. Quistorff. 31P-MRS characterization of sprint and endurance trained athletes. *International Journal of Sports Medicine* 24(3):183-9, April 2003.
- Jubrias, S.A., G.J. Crowther, E.G. Shankland, R.K. Gronka, and K.E. Conley. Acidosis inhibits oxidative phosphorylation in contracting human skeletal muscle in vivo. *Journal of Physiology* 553.2:589-99, 1 December 2003.
- Kemp, G.J., D.J. Taylor, P. Styles, and G.K. Radda. The production, buffering and efflux of protons in human skeletal muscle during exercise and recovery. *NMR in Biomedicine* 6(1):73-83, January-February 1993.
- Kemp, G.J., C.H. Thompson, P.R. Barnes, and G.K. Radda. Comparisons of ATP turnover in human muscle during ischemic and aerobic exercise using 31P magnetic resonance spectroscopy. *Magnetic Resonance in Medicine* 31(3):248-58, March 1994.
- Kemp, G.J., C.H. Thompson, D.J. Taylor, and G.K. Radda. Proton efflux in human skeletal muscle during recovery from exercise. *European Journal of Applied and Occupational Physiology* 76(5):462-71, 1997.
- Kent-Braun, J.A., R.G. Miller, and M.W. Weiner. Phases of metabolism during progressive exercise to fatigue in human skeletal muscle. *Journal of Applied Physiology* 75(2):573-80, August 1993.
- Marsh, G.D., D.H. Paterson, J.J. Potwarka, and R.T. Thompson. Transient changes in muscle high-energy phosphates during moderate exercise. *Journal of Applied Physiology* 75(2):648-56, August 1993.
- McCann, D.J., PA Mole, and J.R. Caton. Phosphocreatine kinetics in humans during exercise and recovery. *Medicine and Science in Sports and Exercise* 27(3):378-89, March 1995.
- McMahon S., and D. Jenkins. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. Review. *Sports Medicine* 32(12):761-84, 2002.
- Park, J.H., et al. Functional pools of oxidative and glycolytic fibers in human muscle observed by 31P magnetic resonance spectroscopy during exercise. *Proceedings of the National Academy of Sciences in the U.S.A.* 84(24):8976-80, December 1987.
- Price, T.B., D. Laurent, K.F. Petersen, D.L. Rothman, and G.I. Shulman. Glycogen loading alters muscle glycogen resynthesis after exercise. *Journal of Applied Physiology* 88(2):698-704, February 2000.
- Sahlin, K., J.B. Sorensen, L.B. Gladden, H.B. Rossiter, and P.K. Pedersen. Prior heavy exercise eliminates VO<sub>2</sub> slow component and reduces efficiency during submaximal exercise in humans. *Journal of Physiology* 564.3:765-73, 1 May 2005.
- Schocke, M.F., et al. High-energy phosphate metabolism during two bouts of progressive calf exercise in humans measured by phosphorus-31 magnetic resonance spectroscopy. *European Journal of Applied Physiology* 93(4):469-79, January 2005.
- Takahashi, H., et al. Control of the rate of phosphocreatine resynthesis after exercise in trained and untrained human quadriceps muscles. *European Journal of Applied and Occupational Physiology* 71(5):396-404, 1995.
- Tomlin, D.L., and H.A. Wenger. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. Review. *Sports Medicine* 31(1):1-11, 2001.
- Thompson, C.H., G.J. Kemp, A.L. Sanderson, and G.K. Radda. Skeletal muscle mitochondrial function studied by kinetic analysis of postexercise phosphocreatine resynthesis. *Journal of Applied Physiology* 78(6):2131-9, June 1995.
- Walter, G., K. Vandenborne, K.K. McCully, and J.S. Leigh. Noninvasive measurement of phosphocreatine recovery kinetics in single human muscles. *American Journal of Physiology* 272(2 Pt 1):C525-34, February 1997.
- Walter, G., K. Vandenborne, M. Elliott, and J.S. Leigh. In vivo ATP synthesis rates in single human muscles during high intensity exercise. *Journal of Physiology* 519.3:901-10, 15 September 1999.
- Whipp, B.J., S.A. Ward, and H.B. Rossiter. Pulmonary O<sub>2</sub> uptake during exercise: conflating muscular and cardiovascular responses. Review. *Medicine and Science in Sports and Exercise* 37(9):1574-85, September 2005.
- Yoshida, T., and H. Watari. Muscle metabolism during repeated exercise studied by 31P-MRS. *Annals of Physiological Anthropology* 11(3):241-50, May 1992.

- Yoshida, T., and H. Watari. <sup>31</sup>P-nuclear magnetic resonance spectroscopy study of the time course of energy metabolism during exercise and recovery. *European Journal of Applied and Occupational Physiology* 66(6):494-9, 1993.
- Yoshida, T., and H. Watari. Metabolic consequences of repeated exercise in long distance runners. *European Journal of Applied and Occupational Physiology* 67(3):261-5, 1993.
- Yoshida, T., H. Watari, and K. Tagawa. Effects of active and passive recoveries on splitting of the inorganic phosphate peak determined by <sup>31</sup>P-nuclear magnetic resonance spectroscopy. *NMR in Biomedicine* 9(1):13-9, February 1996. Erratum in: *NMR Biomedicine* 9(6):277, September 1996.
- Yoshida, T. The rate of phosphocreatine hydrolysis and resynthesis in exercising muscle in humans using <sup>31</sup>P-MRS. *Journal of Physiological Anthropology and Applied Human Science* 21(5):247-55, September 2002.