

3) PUTTING THE PIECES TOGETHER

a) *The Lactate Threshold*

In exercise physiology, there have been few topics more frequently investigated, or more vigorously debated than the lactate threshold. It is the details, not the basics that create the big research problems. However, it is the basics that have great application to training and performance. So, we'll stick to those.

What is Lactic Acid and Where Does it Come From?

When you consume carbohydrate, it consists of several different sugar molecules; sucrose, fructose, glucose to name a few. However, by the time the liver does its job, all of this sugar is converted to glucose which can be taken up by all cells. Muscle fibers take up glucose and either use it immediately, or store it in the form of long glucose chains called glycogen. During exercise, glycogen is broken down to glucose which then goes through a sequence of enzymatic reactions that do not require oxygen to proceed. All of these reactions occur out in the cell fluid, or cytosol. They can occur very rapidly and yield some ATP in the process. This pathway is called the anaerobic (no oxygen) glycolysis (glucose breakdown) pathway. Every single glucose molecule must go through this sequence of reactions for useful energy to be withdrawn and converted to ATP, the energy molecule, that fuels muscle contraction, and all other cellular energy dependant functions.

The Metabolic Fork in the Road

There is a critical metabolic fork in the road at the end of this chemical pathway. At this fork, glucose has been converted from one 6 carbon molecule to two, 3 carbon molecules called pyruvic acid, or pyruvate. This pyruvate can either be shuttled into the mitochondria via the enzyme pyruvate dehydrogenase, or be converted to lactic acid via the enzyme lactate dehydrogenase. Entry into the mitochondria exposes the pyruvate to further enzymatic breakdown, oxidation, and a high ATP yield per glucose. Conversion to lactate means a temporary dead end in the energy yielding process, and the potential for contractile fatigue due to decreasing cellular pH if lactic acid accumulation proceeds unchecked. Like a leaf floating in a river, the pyruvate molecule has no "say" in which metabolic direction is taken.

Which Way will MY pyruvate go during exercise?

I am sure you have surmised that that is a critical question with big implications for performance. I will try to answer the question at three levels: a single muscle fiber, an entire muscle that is active during exercise, and the entire exercising body.

The Muscle Cell at Work

In a single contracting muscle fiber, the frequency and duration of contractions will determine ATP demand. ATP demand will be met by metabolizing a combination of two energy sources: fatty acids and glucose molecules (ignoring the small contribution of protein for now). As ATP demand increases, the rate of glucose flux through glycolytic pathway increases. Therefore at high workloads within the single fiber, the rate of pyruvic acid production will be very high. If the muscle fiber has a lot of mitochondria (and therefore more Pyruvate Dehydrogenase), pyruvate will tend to be converted to Acetyl CoA and move into the mitochondria, with relatively little lactate production. Additionally, fatty acid metabolism will account for a higher percentage of the ATP need. Fat metabolism does not produce lactate, ever! If lactate is produced from glucose breakdown, it will tend to diffuse from the area of high concentration inside the muscle cell to lower concentration out of the muscle fiber and into extracellular fluid, then into the capillaries.

The Whole Muscle at Work

Now let's look at an entire muscle, say the vastus lateralis of the quadriceps group during cycling. At a low workload, glycolytic flux is low and the pyruvate produced is primarily shuttled into the mitochondria for oxidative breakdown. Since the workload is low, primarily slow twitch fibers are active. These fibers have high mitochondrial volume. As workload increases, more fibers are recruited and recruited fibers have higher duty cycles. Now ATP demand has increased in the previously active fibers, resulting in higher rates of pyruvic acid production. A greater proportion of this now is converted to lactic acid rather than entering the mitochondria, due to competition between LDH and PDH. Meanwhile, some Fast twitch motor units are starting to be recruited. This will add to the lactate efflux from the muscle due to the lower mitochondrial volume of these fibers. The rate of lactate appearance in the blood stream increases.

The Body at Work

The vastus is just one of several muscles that are very active in cycling. With increasing intensity, increased muscle mass is called on to meet the force production requirements. All of these muscles are contributing more or less lactic acid to the extracellular space and blood volume, depending on their fiber type composition, training status and activity level. However, the body is not just producing lactate, but also consuming it. The heart, the liver, the kidneys and inactive muscles are all locations where lactic acid can be taken up from the blood and either converted back to pyruvic acid and metabolized in the mitochondria or used as a building block to resynthesize glucose (the liver). These sites have low intracellular lactate concentration, so lactic acid diffuses INTO these cells from the circulatory system. If the rate of uptake or disappearance of lactate equals the rate of production or appearance in the blood, then blood lactate concentration stays constant (or nearly so). When the rate of lactate production exceeds the rate of disappearance, lactic acid accumulates in the blood volume, then we see the ONSET of BLOOD LACTATE ACCUMULATION (OBLA). This is the "Lactate Threshold" (LT).

Performance Implications

Lactic Acid production is not all bad. If we could not produce lactate, our ability to perform brief high intensity exercise would be almost eliminated. However, As I am sure you are aware, lactic acid is the demon of the endurance athlete. Cellular accumulation of the protons (increased acidity) that dissociate from lactate results in inhibition of muscle contraction. Blame those heavy legs on the protons! The bottom line is that exercise intensities above the OBLA point can only be sustained for a few minutes to perhaps one hour depending on how high the workload is above the intensity at OBLA. Exercise at or below this intensity may be sustainable for hours. The causes of fatigue at these sub-LT intensities include carbohydrate depletion and dehydration.

Factors that Influence the Rate of Lactate Accumulation in the body

- **Absolute Exercise Intensity** - for reasons mentioned above.
- **Training Status of Active Muscles** - Higher mitochondrial volume improves capacity for oxidative metabolism at high glycolytic flux rates.

Additionally, improved fatty acid oxidation capacity results in decreased glucose utilization at submaximal exercise intensities. Fat metabolism proceeds via a different pathway than glucose, and lactic acid is not produced. High capillary density improves both oxygen delivery to the mitochondria and washout of waste products from the active muscles.

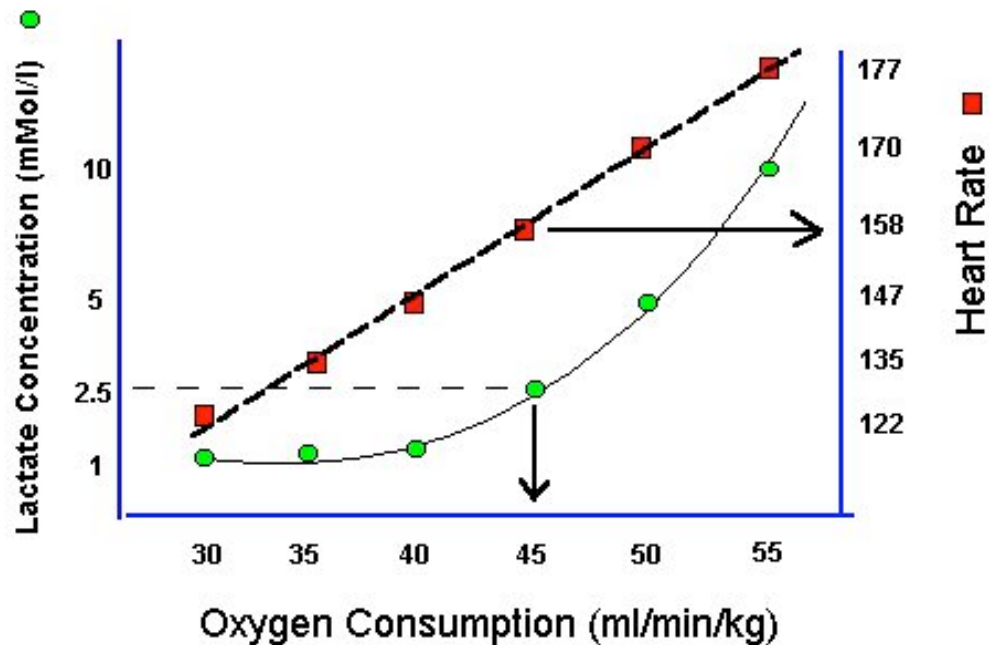
- **Fiber Type Composition** - Slow twitch fibers produce less lactate at a given workload than fast twitch fibers, independent of training status.
- **Distribution of Workload** - A large muscle mass working at a moderate intensity will develop less lactate than a small muscle mass working at a high intensity. For example, the rower must learn to effectively distribute force development among the muscles of the legs back and arms, rather than focusing all of the load on the legs, or the upper body.
- **Rate of Blood Lactate Clearance** - With training, blood flow to organs such as the liver and kidneys decreases less at any given exercise workload, due to decreased sympathetic stimulation. This results in increased lactate removal from the circulatory system by these organs.

Measuring the Lactate Threshold

We have previously discussed the value of a high maximal oxygen consumption for the endurance athlete (**see subcategory-(1.a.v) below**). A big VO₂ max sets the ceiling for our sustainable work rate. It is a measure of the size of our performance engine. However, the Lactate Threshold greatly influences the actual percentage of that engine power that can be used continuously.

Most of you will never have this measured in a laboratory, but a brief description of a lactate threshold test is still useful, because it will lead us into some specific applications for your racing and training. The test consists of successive stages of exercise on a treadmill, bicycle ergometer, swimming flume, rowing machine etc. Initially the exercise intensity is about 50- 60% of the VO₂ max. Each stage generally lasts about 5 minutes. Near the end of each stage, heart rate is recorded, oxygen consumption is measured, and a sample of blood is withdrawn, using a needle prick of the finger or earlobe. Using special instrumentation, blood lactate concentration can be determined during the test. After these measurements, the workload is increased and the steps repeated. Through a 6 stage test, we would expect to achieve a

distribution of intensities that are below, at , and above the intensity of OBLA or the lactate threshold. The data from a test would generally look similar to the example below.



Interpreting the Data

For purposes of interpretation, let's say that the athlete above had a maximal heart rate of 182, and a VO_2 max or 61 ml/min/kg. These were also determined using a bicycle test. So they are good values for comparison. Looking at the green dots, we see that blood lactate concentration does not begin to increase until during the 4th workload, from a concentration of about 1 mM to 2.5 mM. This is the break point. The subject's VO_2 was 45 ml/min/kg at this point. So we determine that his LT occurs at 45/61 or about 74% of VO_2 max. If we look at the heart rate at this point, it is 158. Now we have a heart rate at lactate threshold. $158 = \text{about } 85\%$ of his max heart rate. This is useful for the athlete. When he is cycling, he can judge his training intensities based on this important value. If he is a time trialist, this would approximate his racing heart rate for the hour long event.

So, Do I Race at My LT Intensity?

This depends on your race duration. If you are rowing 2000 meters, running a 5k race etc, your exercise intensity will be well above the AT. Consequently,

the blood lactate measured after these events is extremely high in elite athletes, on the order of 15mM (resting levels are below 1 mM). In races lasting from 30 minutes to 1 hour, well trained athletes also perform at an intensity above LT, but by a smaller margin. It appears that in these events, top performers achieve what might be termed a **"maximal lactate steady state"**. Blood lactate may increase to 8 to 10 mM within minutes, and then stabilize for the race duration. A high but stable lactate concentration may seem to contradict the idea of the LT. But, remember that blood lactate concentration is the consequence of both production and clearance. It seems likely that at these higher lactate concentrations, uptake by non-working muscles is optimized. At any rate, measurements in cyclists, runners and skiers demonstrate the fact that elite performers can sustain work levels substantially above the LT for up to one hour.

Specificity of the Lactate Threshold

It is important to know that the lactate threshold is highly specific to the exercise task. So if this cyclist tries to get on his brand new, previously unused, rowing machine and row at a heart rate of 158, he will quickly become fatigued. Rowing employs different muscles and neuromuscular patterns. Since these muscles are less trained, the cyclist's rowing LT will be considerably lower. This specificity is an important concept to understand when using heart rate as a guide in "cross training activities", as well as for the multi-event athlete.

Effect of Training

For reasons mentioned above, training results in a decrease in lactate production at any given exercise intensity. Untrained individuals usually reach the LT at about 60% of VO₂ max. With training, LT can increase from 60% to above 70% or even higher. Elite endurance athletes and top masters athletes typically have LTs at or above 80% of VO₂ max. Values approaching 90% have been reported. The lactate threshold is both responsive to training and influenced by genetics.

b) Efficiency, Economy and Endurance Performance

So far, if you read the two previous articles regarding "The System", you know that high level endurance performance depends on 1) a high maximal oxygen consumption, or VO₂ max, and 2) a high lactate threshold, or point of

OBLA. Your VO2 max sets the upper limit for your sustainable work potential. For the elite endurance athlete, a high VO2 max is like the invitation to the big dance. Having an invitation to the dance does not ensure you will dance with the prettiest girl. But, not having one ensures you won't! The lactate threshold tells us something about how much of the cardiovascular capacity you can take advantage of in a sustained effort. It is determined by skeletal muscle characteristics and training adaptations. Multiplying VO2 max x LT (Oxygen Consumption at Lactate Threshold) gives us a measure of the effective size of your endurance engine. Now we come to efficiency. What does efficiency have to do with endurance performance? Victory goes to the person with the biggest endurance engine right? Well, let's use a racecar analogy.

If I build a powerful, well-tuned engine that can run at redline RPMs for hours, and then drop it into a Ford truck chassis, the truck might go 120 mph. But if I drop it into a streamlined Ferrari chassis, I might hit 200 mph (in theory, personally I am afraid I would soil my pants and hit the brakes long before I reached 200 mph). That is a big difference. Engine performance didn't change, but performance velocity did. To some extent, the same efficiency effect is observed in every endurance sport. Efficiency is critical to maximizing performance velocity!

Physiological Efficiency Defined

In an exercise setting, efficiency is defined as the percentage of energy expended by the body that is converted to mechanical work (another form of energy).

Work Efficiency = Mechanical work / Chemical energy expended

We can measure the mechanical work performed using an ergometer, like a bicycle ergometer, or rowing machine. We can measure the energy expended by the body indirectly via its oxygen consumption at sub maximal workloads. With some basic biochemistry we can convert the oxygen consumption we measure during exercise to a standard measure of energy like **kJoules, or Calories**. And, we can do the same for the work we measure on the ergometer. Work/time = power. Power is measured in watts and is a measure of the intensity of work. Intensity (watts) x exercise duration (minutes) gives **us total work, again measured in kJoules or Calories**.

If we take a group of cyclists, or a group of rowers and perform sub maximal testing on them to determine how much energy they consume when

performing a standard sub maximal workload, we find that overall work efficiency will range between about 17 and 26%, with an average somewhere in the middle of that range. In other words for every 100 Calories of energy burned, we manage to convert 20 Calories of that energy to useful work on the pedals of the ergometer, or as pulling power on the rowing machine. Now, if your goal is to lose body fat during exercise, then I suppose it pays to be inefficient, since it is Calories burned that matter. However, if your goal is to move your body faster than the other guy, than being 25% efficient is way better than 18%! So, what are the sources of inefficiency and what, if anything can we do about them?

Sources of Inefficiency in the Performance Machine

Let's use a 40 km time-trial in cycling as an example. The goal is to propel your body on a bicycle over land, and through air at the fastest sustained speed. So where do the energy losses occur in the path from chemical energy stored in pasta to velocity sustained on the bicycle over a 40km distance?

1. Chemical energy conversion losses - Your body must generate ATP for muscle contraction by chemically converting food energy, using a process that ultimately requires oxygen (hence the need for a big oxygen delivery capacity), while minimizing the production of lactic acid (high LT). All of the chemical energy in food is not transferred to ATP. About 60% is lost as heat energy. This is why you get hot during exercise. This source of inefficiency is the same in everyone.

2. Fiber type differences in converting ATP energy to contraction force - The next source of energy loss is in the step in which the chemical energy trapped within the ATP molecule is converted to mechanical energy via muscle contraction. There is some recent data from one laboratory in the United States suggesting that fiber composition of the muscle influences efficiency of muscle contraction (Coyle et al., *Medicine and Science in Sports and Exercise*. 24:782-788, 1992). Cyclists with a high percentage of slow twitch fibers appear MORE efficient. This was observed as a smaller increase in oxygen consumption for a given increase in cycling power output, in a group of cyclists whose fiber composition varied between 35 and 76% slow twitch. The higher efficiency was also observed when performing repetitive leg extensions, suggesting the source of efficiency was in the muscle, not the riding technique. The way they get at this is to measure the DELTA efficiency which is just the change in energy demand for a given change in power. By

measuring the change in oxygen consumption an athlete requires to increase his work rate from say 150 to 200 watts, while keeping his cadence and body position the same, this specific efficiency of the muscles can be determined. The differences are small. Perhaps a cyclist with 80% slow twitch fibers would have a **Delta efficiency** of 25%. A person with only 50% slow twitch fibers might be 21% efficient. So within the endurance community, the numerical difference in muscular efficiency seems small, but the impact on power output in a 40km time trial can be 8-10% independent of other variables. For example, data from Horowitz et al. (Int. J. Sports Medicine, 15:152-157, 1993) compared two groups of seven cyclists. The average VO₂ maintained during 1 hour of cycling (a function of VO₂ max and Lactate threshold) was the same in the two groups (4.48 vs. 4.46 l/min). However, the group with the higher average % slow twitch fibers (73 vs 48) achieved higher power output during the hour of cycling at voluntary maximal power (342 watts vs 315). They achieved 8% higher power output for the same physiological cost. How does this translate to velocity on a bike that actually moves?

3. The energy cost of moving the limbs - If you sit on a bicycle ergometer with the load set at zero and pedal at 80 rpms, you will discover that even though you are not doing any measurable mechanical work, you are still WORKING. It costs energy to just move your limbs, support your body, hold your balance, etc. The same is of course true for ANY movement, like running or skiing, or rowing. When this “unloaded cost of movement” is included in our measure of the mechanical work to energy expenditure ratio, then we get the GROSS Efficiency. Here, the word “gross” means “overall”, not “icky”.

One factor that impacts gross efficiency is movement frequency. That can be cycling cadence, or rowing stroke rate, or stride frequency in XC skiing. Higher cadences tend to cost more energy in general. And heavier limbs have been shown to be less efficient to move. However, there is a balance such that trained athletes tend to zero in on an optimal cadence for their body type and anatomy. When they are pushed away from that cadence, they use more energy to do the same work. Therefore, it is important to realize that the ideal movement frequency is not a universal, but varies from individual to individual. So, you should not try automatically to mimic your training partner's cadence if they are much taller or shorter, or more or less muscular than you.

Having said that, in sports like rowing and cross-country skiing, there is a general tendency that the best athletes with the big engines use it by pulling or pushing harder each stroke, not revving up their movement frequency.

This makes sense. Pushing harder each stroke means that more of the total energy goes to propelling the body and less to moving the limbs back and forth. And, you have no doubt seen the truth of this in watching how smoothly the great rowers, or skiers, or runners generate speed. Their technique looks controlled and powerful, not frantic or hurried. There is a fine balance though. If you try to work with huge powers at too low frequencies, then the muscles become overloaded, blood flow gets compromised and fatigue results. So, the endurance athlete seeks a balance between the efficiency of lower movement frequencies and the decreased muscle tension and blood vessel compression of higher frequencies for a given workload. Exactly where this balance point lies varies from athlete to athlete.

In sports that are very technique intensive, like cycling, XC skiing, or rowing, there is much to be gained in perfecting the biomechanics of the movement. However, in cycling, with its relatively basic movement pattern, there does not seem to be any difference in overall cycling efficiency between elite cyclists and cyclists that have not been training very long. Sure, efficiency is an advantage, but the research indicates that cycling efficiency does not get better and better with years of training.

Efficiency vs Economy

The difference between efficiency and economy in an exercise setting is that **economy is measured as movement velocity for a given energy consumption**, while **efficiency is mechanical power output for a given energy consumption**. When we measure economy we connect the power produced to the movement intended, like cycling as fast as possible over 40km.

This brings us to the Truck vs. Ferrari analogy. Having the biggest engine doesn't guarantee the fastest performance in car racing or bicycle racing (or rowing, running, and swimming). The Ferrari goes faster because it is lighter and slices very cleanly through the air, reducing aerodynamic drag. So does the cyclist who perfects an aerodynamic riding position. (***See discussion of Cycling Aerodynamics from expert Jim Martin in separate article***). The best distance runners display high running economy. This means that they can run at a given speed with less oxygen demand. A high economy can make up for a relatively lower VO₂ max. For example, Derek Clayton ran an incredible 2:08 marathon in 1969. His VO₂ max was "only" 69 ml/min/kg (well it was probably a bit higher than that, but this was data from one non-peak season test). Thanks to his high running economy, that time stood for 12

years and was not matched by talented runners such as Craig Virgin, Gary Tuttle, and Bill Rodgers, whose VO₂ max values ranged from 78 to 82 ml/min/kg! In rowing, both the hydrodynamics of the racing shell and the technical mastery of the rower contribute to rowing economy. However, even on a stationary ergometer, elite rowers are more efficient than well-trained but non-elite oarsman. This is not due to a difference in fiber composition. So, it appears that subtle changes in rowing technique can continue to contribute to improve rowing efficiency and performance with additional years of training.

In no sport is efficiency more important than in swimming. The best swimmers in the world do not stand out in physiological tests of raw endurance capacity when compared to other endurance athletes. This suggests that high efficiency, achieved through a combination of ideal anatomical structure and technical perfection of the stroke is critical.

The Big Picture - Going back to the performance model, I identified several anatomical and cellular characteristics that contribute to 1) maximal oxygen consumption, 2) relative work intensity at lactic acid threshold, and 3) efficiency of transfer of physiological work to movement velocity. The details differ with each sports discipline and the event duration. But these are the BIG THREE variables. Maximal oxygen consumption is limited by central cardiovascular function, but also dependent on the peripheral adaptations that occur in the trained muscles. A high lactate threshold is due to peripheral adaptations improving the muscle's ability to generate energy aerobically. And, a high efficiency/economy creates the link between the physiological engine and the actual performance goal, to maximize average velocity.

c) The Brain - Body Link and Adaptation to Training

Because most exercise physiologists focus our training on muscle and systemic physiology, we tend to treat the brain as a mysterious black box, but that is changing. I for one, am recognizing that I must learn more about the impact of exercise on the brain, and the impact of brain activity on physical function. The MIND-BODY link is becoming more than just mysticism.

Here I will try to present some material that comes from the 25 years of work by Dr. Heinz Liesen. He served as National Team Doctor for the German soccer (football) team that reached the WorldCup final in 86 and 90, despite only modest talent. He also served as team doctor for the very successful national Field Hockey team and the Nordic Combined (cross country skiing

plus ski jumping) team. Today his focus has returned to preventive medicine. The knowledge that he derives from following specific athletes (and non-athlete exercisers) for several years, with repeated measures of cellular immune reactivity, training pattern, performance, and even regionalized brain wave activity, is unique in the field. Some of the material also is based on a growing body of research generated in the United States and in Germany.

The Brain is the Center of Sports Performance

After all this talk about heart and muscles, this must sound pretty crazy. It is true though. The brain both initiates all of our voluntary movements and reacts to the stress that exercise creates. And, to some extent, stress seems to be a universal quality. The brain responds to the stress of job, driving, training, competition. The measurable impact of this stress is revealed in several ways:

- **Resting Catecholamine Levels.** Appropriate training tends to produce parasympathetic (rest and recover) dominance in the endurance trained athlete. However, if training stress becomes too severe, sympathetic (fight or flight) hormone levels remain elevated even at rest, an indication of incomplete recovery. An outward manifestation of this change is an elevated resting heart rate, although this may not be as sensitive as other measures. Another characteristic of the heart rhythm at rest is a certain degree of irregularity. There is considerable beat to beat variability in the heart rate, measured as minute changes in the interval between successive beats. This variability actually decreases in anticipation of a mental task, as sympathetic stimulation increases.

- **Testosterone / Cortisol Ratio.** Testosterone is an *anabolic* hormone that plays a role in regeneration and repair of muscle and tissue. Cortisol is a catabolic hormone that stimulates tissue breakdown. For example, cortisol levels are elevated in starvation situations when muscle tissue is catabolized for energy. Testosterone levels tend to be higher in individuals with a high capacity for hard training and recovery (and in those who receive it via a needle). Testosterone levels are naturally lower in women compared to men (about 10x lower). The testosterone/cortisol ratio has been shown in some (but not all) studies to be indicative of overtraining and staleness in elite athletes.

- **Immune System Function.** The immune system is a simple term for an intrinsic, adaptable cellular system that responds to invading foreign

substances and eliminates them, or minimizes their capacity to replicate. The immune system is modifiable both in the rapidity and magnitude of its response to a foreign invader. Exercise creates both acute and chronic changes in immune system function. Acute exercise has been shown to cause a transient depression of certain components of the immune system, creating a window of susceptibility to infection of several hours after an exercise bout. The stress of chronic exercise has a biphasic effect on immune function. This can be demonstrated several ways. First, the incidence of upper respiratory tract infections (URTI) **decreases** with moderate exercise, but **increases in hard training athletes** (J-shaped Incidence curve). Second, the magnitude of immune response to an applied antigen is decreased in overstressed athletes. Diagnostic kits are available which allow the controlled application of 7 allergens to the skin of the forearm. The total area of resulting skin reactions represent a quantitative measure of the vigor of the immune system (within a given individual). This measurement is routinely made by many national teams in Germany and Scandinavia.

Perhaps the most interesting information I can give you is also the most difficult for me to understand with my miniscule knowledge of brain chemistry. It appears that the brain interacts with the immune system and modulates immune responses. This has been poignantly demonstrated by Dr. Liesen. By comparing blood withdrawn immediately before, and 1 hour after an unanticipated, but stress inducing medical diagnosis, he observed dramatic changes in the antigenic responsiveness of blood leukocytes. This brain modulation of immune function appears to involve the release of specific immuno modulating chemicals by the brain in response to emotional stimuli.

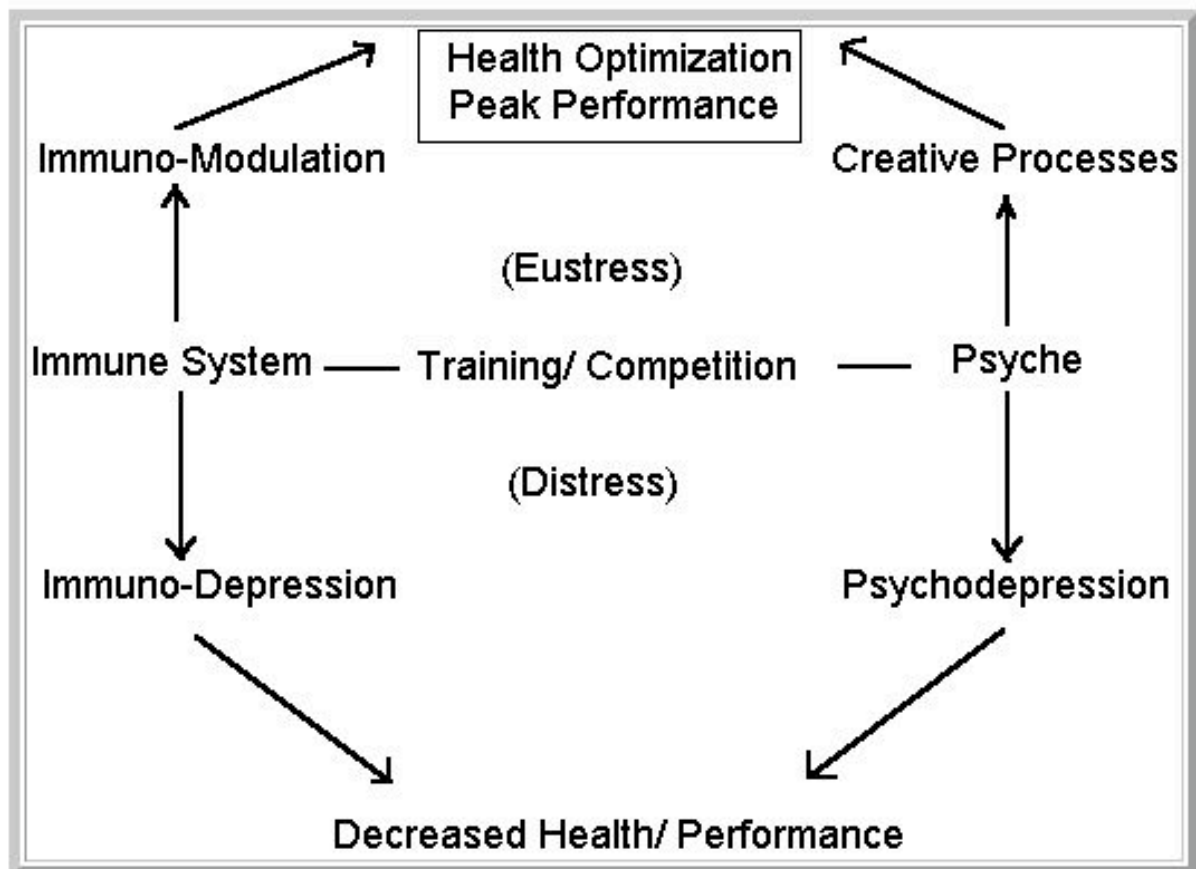
- **Psychological Profiles.** Several survey instruments have been developed which appear to be sensitive to emotional changes that accompany **or precede** the physiological and performance changes associated with an overtrained state. The instruments ask questions about current mood, anxiety, sleep quality, desire to train, etc.

The Big Picture

In today's world of elite sport, the real limitation to continued improvement has moved from the quantity of training to the capacity of the mind and body for restitution. Many elite athletes are training 50 weeks per year, sometimes 3-4 hours/day. When this extreme physical stress is combined with the stress of more frequent competitions to satisfy sponsors, media pressure and a tendency to lose time or interest in mentally diverting creative activities, the

results are often disastrous. What we often see if we observe closely, is the sudden appearance of extremely talented performers, followed one or two years later by a decline in their performance or a complete disappearance from the scene. Behind these early burnouts is usually a coach or performance team that is pushing too hard.

Below is a model presented by Dr. Liesen based on his experience and research, depicting the potential for both positive and negative effects of physical exercise on health and peak performance.



The success of the teams and individual athletes managed by Dr. Liesen was not due to an intensification of their training. On the contrary, more careful application of low intensity, "recuperative" training and even, complete rest days was the key. Keep in mind that complete days without training for world class endurance athletes are bitter pills to swallow. Here in Norway, World and Olympic Champion cross country skier, Bjorn Dahlie was recently quoted in the paper, "A day without training is a day without value". Three weeks later

he had to withdraw from the National championships due to illness. Rest is important

How we rest is also important. For example, Dr. Liesen observed that the football (soccer) players tended to do nothing more than lie around and watch TV between training sessions, their mind assuming an almost vegetative state. To increase their mental creativity, he took his team members to museums, helped them study new languages, started them doing hand-crafts, all during the heat of training and World Cup competition. The results were outstanding. Modestly talented German teams advanced to the World Cup final in 1986 and 1990 (running out of talent both times in the Final). They were successful in large measure because they stayed healthy and strong throughout the tournament.

When you look at the model above, you see both exercise level and creative mental activity as potential modulators of health and performance. When we build a training program, we have to consider the brain as well as the body.

The typical masters athlete does not train at the same volume as elite athletes. So, you might think, "overtraining is not an issue for me since I only train 12 hours per week." But, do you have a career, children, an hour of rush hour traffic each day. Has every training session become intense? Does each training ride/run become a race? Have other hobbies disappeared from your routine. When you aren't training are you thinking about training? If you answered yes to most of these questions, you have to reevaluate your training program and your approach to exercise.

The Long Haul

In college and at the world class level, the clock is always ticking. Athletes feel pressure to reach their peak "this year". In many cases, this leads to yearly cycles that do not consider the ongoing development of the athlete "the next year". As a masters athlete, remember that you are in this game for the long haul. Training is a long process of learning and physical and technical growth. Medals go to those who combine talent with patience, and intensity with intelligence. Ultimately, no matter what your level of performance, the satisfaction of athleticism is sweetest when it enhances your life, not just your VO2 max!