Journal of Applied Sport Science Research
1991, Volume 5, Number 1, pp. 22-34

Anaerobic Metabolic Conditioning: A Brief Review of Theory, Strategy and Practical Application

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ABSTRACT

Plisk, S.S. Anaerobic metabolic conditioning: a brief review of theory, strategy and practical application. *J. App. Sport Sci. Res.* 5(1):00-00. 1991.—*Anaerobic "sprint-interval" metabolic conditioning differs from aerobic in that exercise intensity is greater (supramaximal) and duration is shorter in the former; exercise modality may be identical, and is usually total-body in nature. Manipulation of the exercise:relief ratio should be based upon several criteria: competitive activity-inactivity profiles, bioenergetic kinetics and time courses of phosphagen repletion and lactate clearance during recovery. While there is discussion in the literature regarding manipulation of chronic training variables (especially exercise intensity, duration, volume, frequency, program progression and duration, concurrent "cross-training" compatibility and testing) and of the functional significance of such training in previously untrained subjects, data on athletic populations are scarce. However, it appears that sprint-interval training yields specific, positive physiological adaptations in the neuromusculature. Directions for future research should include the mechanisms and trainability of substrate repletion and metabolite clearance kinetics, concurrent strength-power and metabolic training compatibility and calcium metabolism.*

KEY WORDS: Bioenergetic, capacity, fatigability, fiber type, half-time, interval, kinetics.

Introduction

The purpose of this article is twofold. First, it is to provide an overview of the scientific theory and strategy underlying the sport-specific metabolic training effect. One assumption has been made: that the athlete of interest is anaerobically oriented, and thus special attention is devoted to training for those sports and athletic events traditionally considered as such. The focus of this review is directed toward peripheral (muscular) metabolic adaptations, since this is where the bulk of the available information lies. However, where appropriate, reference is made to accompanying central responses as well.

Second, in realizing that there are several common and valid approaches to training for such activites, discussion of certain program design considerations is focused on the common theory upon which these are based, without necessarily outlining specific program designs or models. Therefore, the practical implications for anaerobic training and testing are discussed in theoretical terms with respect to the available scientific evidence.

Strategy

Anaerobic-type metabolic conditioning can be characterized according to acute and chronic exercise response. Actually, the exercise stimulus must yield appropriate motor unit activity, substrate flux and force-speed production patterns such that the anaerobic bioenergetic pathways are preferentially

recruited in the active neuromusculature. Such periodic and systematic stress tends to yield chronic responses in the affected tissues, including some combination of specific neural, metabolic and architectural adaptations (9, 13, 21, 26, 27, 29-32, 44, 46, 67, 74, 85, 91, 94, 95, 104). These are associated with distinct augmentations in both physiological and performance-related exercise parameters.

Such training is typically accomplished through sets of repetitions, referred to as intervals or sprints, performed intermittently at variable exercise:relief ratios. For purposes of discussion, an exercise *interval* is defined as any multiple-sequence or whole-body activity of a continuous and rhythmical nature (e.g., running, cycling, swimming), of supramaximal intensity (above $\dot{V}O_{2\ max}$), and of up to 120 seconds duration (usually 30 to 90 seconds) per repetition. A *sprint* can be considered a subset of this definition, as it must meet the same criteria, with two modifications: intensity is of maximal or near-maximal anaerobic power, and duration is up to 15 seconds per repetition. Note that while traditional resistance-type training may meet these criteria in certain cases, it will be excluded from these definitions.

The concept of interval-type training was originally designed with two basic purposes: to train the oxidative pathways via an overload stimulus of high (maximal or supramaximal) intensity, and to increase the total exercise time capability at that intensity when compared with exercise of continuous duration (28, 62). This was accomplished by imposing exercise intervals interspersed by relief or rest periods, usually at a specific ratio. By manipulating these variables according to certain principles, it has become possible to stress anaerobic metabolism more so than aerobic, and to elicit profound adaptive responses in the former, as will be discussed shortly.

The intensity and duration of both the exercise and relief fractions, and the actual exercise:relief ratio itself, are to a large degree based on four theoretical relationships: the specific nature of competitive activity, the kinetics of bioenergetic system recruitment, the time course of phosphagen repletion during recovery and the time course of pyruvate-lactate clearance during recovery.

Specific Nature of Competitive Activity

Obviously, conditioning for a particular sport or athletic event involves more than determination of force-velocity profiles or simply strengthening the major muscles deemed necessary for competition. The competitive physical activity (and inactivity) patterns must be closely scrutinized and evaluated (by film analysis of actual competition if necessary). What are the intensities and durations of the exercise fractions during competition? Can these be further subdivided into brief spurts of activity and inactivity? Are the relief periods active or inactive? What is the average (and total) exercise time? If the athlete participates in a team sport, are there position-specific differences in these variables? What is the frequency of media and other time-outs?

Based on these observations, it should be possible to construct a profile of the competitive activity-inactivity pattern. This profile then serves two purposes: first, it provides insight into the bioenergetic system(s) emphasized during competition, and second, it provides a training structure to be explicitly mimicked during at least part of the athlete's conditioning program, depending on how adequately this is addressed by skill-technique training during each phase of the training cycle.

Kinetics of Bioenergetic System Recruitment

We will refer to the classic model of the bioenergetic systems (**Figure 1**) (20). The metabolic strategy behind each of these three systems or "biochemical pathways" is maintenance of the cellular energy charge via degradation of one or more organic

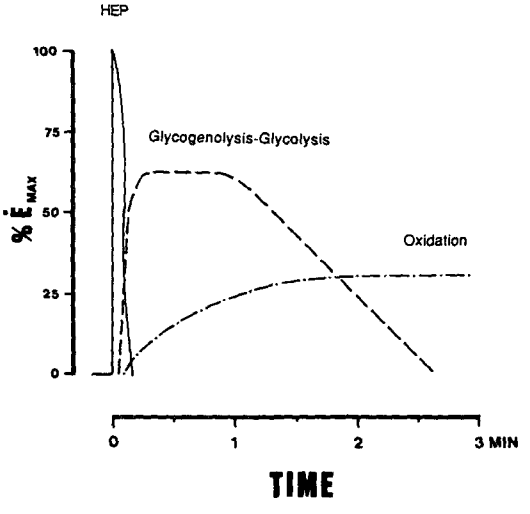

Figure 1. The three systems in human skeletal muscle: Bioenergetic system recruitment according to exercise intensity, or energy production rate (E; vertical axis), and duration (horizontal axis). High energy phosphagen (HEP) system activity is represented by the solid line; glycogenolytic-glyolytic system activity, by the dashed line; and oxidative activity, by the dash-dot line. Modified from Edington and Edgerton (20).

substrates in order to yield adenosine triphosphate (ATP), the chemical intermediate of energy transduction during muscular contraction (14, 56). The high-energy phosphagen (HEP) and glycogenolytic-glycolytic pathways are essentially supplemental systems that support the oxidative pathways, especially during workload onset or transition and when work production (and ATP synthesis) requirements approach or exceed one's aerobic power. While it is beyond the scope of this paper to review the specifics of these processes, it is important to note the following:

First, the three bioenergetic systems are recruited according to, and represent a trade-off between, intensity and duration of workload, or power and capacity of energy production. Second, the area under each curve is equivalent to total exercise or energy production. Third, the absolute and relative power and capacity of each system are subject to some variation, depending on individual genetic endowment, and on training, nutritional and hydration status. Fourth, these pathways, while distinct, are closely interrelated in both function and regulation, and are metabolically synergistic in effect. Fifth, the HEP and glycogenolytic-glycolytic pathways are traditionally considered anaerobic; however, as we shall see, their recovery is largely achieved aerobically and thus they are (at least indirectly) quite oxygen-dependent (11, 16, 33, 39, 47, 49, 53, 54, 78).

HEP. The HEP system, responsible for the degradation and resynthesis of phosphagens, is typically associated with activites of up to several seconds in duration and of near-maximal exercise intensity or energy production rate (E) (17, 53-56, 60, 66, 89). Intramuscular phosphagen turnover rates are linearly related to exercise intensity and are extremely rapid during intense exercise (53, 54, 60). They are, in fact, coincident with actomyosin cross-bridge formation cycles. Muscular E may increase up to 100-fold compared with resting metabolic rate by virtue of this system, a value up to three-fold greater than that of maximal aerobic power (17, 55, 56, 89). The activity of this pathway is most significantly affected by sprint-type training (13, 74, 104).

Glycogenolysis-glycolysis. The glycogenolytic-glycolytic system (Embden-Meyerhof pathway), responsible for carbohydrate degradation, is fully activated within three to five seconds of intense activity and can sustain peak E for up to 40 to 45 seconds, achieving values up to 1.5-fold greater than those of oxidative mechanisms (17, 56, 57, 60, 66, 82, 88, 89). The activity of this pathway is most significantly affected by interval-type training (21, 29, 31, 67, 74, 85, 94). Note that these systems account largely for the anaerobic energy yield (i.e., oxygen deficit) at the onset of, or transition to, square-wave aerobic-type work as well.

Oxidation. The oxidative system achieves steady-state E within two to five minutes of continuous activity. This $\dot{V}O_2$ on-respones operates as a first-order exponential function with a half-time ($t_{1/2}$) usually ranging from 20 to 40 seconds, depending on training and warm-up status, as well as on posture and workload (11, 12, 83, 84). These oxidative or mitochondrial pathways include the tricarboxylic acid (TCA) cycle, β-oxidation cycle, and respiratory electron transport chain, and largely support traditional endurance-type activities of supramaximal intensity and prolonged duration via oxidation of fats and glycolytic end-products (and possibly protein constituents). They also play a prominent role in recovery from intense exercise (11, 16, 33, 39, 47, 49, 53, 54, 78, 86). These relationships dictate certain program design considerations, to be addressed in the training kinetics discussion.

Time Course of Phosphagen Repletion

Conceptually, HEP metabolic training effects are likely to include one or more of three possibilities: phosphagen concentration, enzyme activity and phosphagen repletion kinetics. Note that phosphagen repletion during recovery from exercise is largely achieved oxidatively (11, 16, 43, 54). Thus, we see an indication of the potential value of oxidative metabolism (i.e., aerobic power) for the sprint- or power-oriented athlete, especially as supramaximal efforts are required repeatedly and on short notice.

Phosphagen concentration. Intracellular phosphagen concentrations are relatively low, regardless of muscle or fiber type, with a slight tendency for higher values in type I fibers (45, 55). ATP and phosphocreatine (PCr) are heavy compounds and are therefore stored in limited quantities under resting conditions (in the range of 4 to 6 and 16 to 20 $mmol \cdot kg^{-1}$ wet muscle weight, respectively) (21, 45, 47, 53, 56, 60-62). In light of the key role of the adenine-nucleotide energy charge in cellular metabolic regulation, these low concentrations may be strategically important since small fluctuations in absolute phosphagen concentration would represent larger relative fluctuations, thus allowing for greater regulatory sensitivity (43).

Several studies have shown that intramuscular ATP concentration ($[ATP]_m$) does not drop more than 40 to 60 percent, while $[PCr]_m$ is virtually depleted after several seconds of intense activity (53, 60, 61). Thus, it seems that ATP is spared to a degree at the expense of PCr and other back-up substrates (e.g., glycogen). While the notion of differential phosphagen turnover within or between fiber compartments is an intriguing possibility, methodological considerations make such determinations impractical as yet. Note also that PCr depletion is highly correlated with fatigue during

sprint-type activity (53, 60). Such fatigue may be a protective mechanism that prevents cellular energy levels from dropping below critical thresholds, possibly resulting in rigor, irreversible contracture and potential necrosis.

There is a shortage of data supporting the notion of significant intracellular phosphagen supercompensation with training (21, 61, 62). Indeed, such increases would probably be too small in absolute terms to be useful during maximal anaerobic performance efforts (41). It would seem more judicious to increase enzyme rather than substrate activites in response to training, since the former yields an effective augmenatation in metabolic power, capacity and control while doing little to adversely affect overall cellular bulk or ionic or osmotic balance (43).

Since resting phosphagen concentrations are not likely to vary significantly with training, increases in the total phosphagen pool may only be possible with an increase in muscle mass (41). Thus, a hypertrophic training response may have potential value if accompanied by a parallel increase in phosphagen deposition. Further investigation is needed to determine the nature and efficacy of such a response.

Enzyme activity. Increases in the activites of ATPase, creatine phosphokinases (CPK) and myokinase (MK) have been demonstrated following sprint-type training programs in previously untrained subjects (104), and correlate with an increase in alactic metabolic power (64, 103, 104). In one study, these increases were accompanied by a hypertrophic response characterized by significant increase in both types II and I fiber areas, as well as in the type II:I fiber area ratio (104). Since these enzyme activities are two- to three-fold greater in type II than in type I fibers (68, 103, 105), and exist in fiber type-specific isozymes (68, 105), the preferential hypertrophy of type II fibers alone may have affected these results.

Repletion kinetics. No less important is the rate of phosphagen repletion during recovery. The time course of PCr repletion is bi-phasic, with fast ($t_{1/2}$~20 to 22 seconds) and slow ($t_{1/2}$~170 seconds) components (**Figure 2**) (16, 47, 54). Thus PCr repletion may be essentially complete two to three minutes post-exercise. Since phosphagen repletion is achieved oxidatively, it may be possible (within limits) to positively affect the repletion rate, and thereby reduce recovery time requirements via supplemental aerobic-type training. While such data are lacking, it is likely that there is a happy medium of aerobic power for the sprint- or power-oriented athlete, especially in light of the controversy regarding concurrent strength and endurance training incompatibility (18, 19, 51, 52), and the possibility that this effect may extend to sprint training and anaerobic power or capacity. Further research is needed to determine the optimal and absolute magnitude of such a training effect, if it indeed exists at all.

It should also be possible, via aerobic-type training, to minimize or altogether negate the mitochondrial (69, 71, 73) and capillary (73, 102) dilution phenomena associated with hypertrophic responses to high-intensity resistance training. Until these issues are clarified, supplemental aerobic-type training for the anaerobically oriented athlete should be left to the discretion of the practitioner.

Time Course of Pyruvate-Lactate Clearance

In addition to phosphagen metabolism, pyruvate-lactate kinetics associated with glycogenolytic-glycolytic flux contribute to the exercise-related disruption in intracellular homeostasis as well. The end-product of the rapid anaerobic portion of glycolysis is pyruvic acid (PYR), a three-carbon unit with a number of metabolic fates. The most desirable of these during exercise is conversion to acetyl CoA and subsequent oxidation via TCA cycle. Note that only two net ATP are phosphorylated per glucose unit during the rapid initial portion of glycolysis, whereas

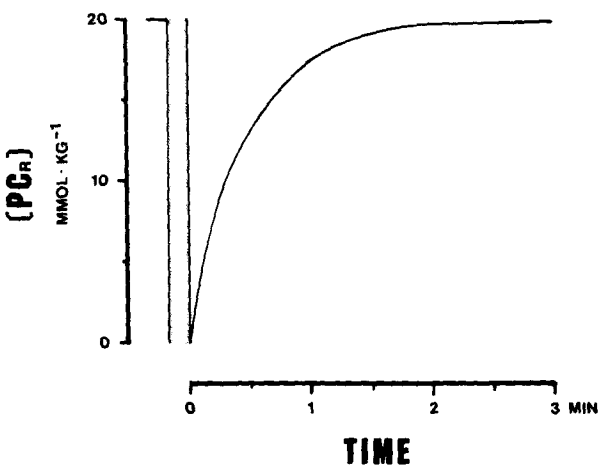

Figure 2. The bi-phasic time course of phosphagen repletion: $[PCr]_m$ as a function of recovery time (16,47,54). The shaded vertical bar represents a 10 second sprint workload during which PCr is depleted in active muscle tissue. Note the initial rapid phase ($t_{1/2}$~20 s) and latter slow phase ($t_{1/2}$~170 s) of repletion during recovery.

an additional 34 ATP are synthesized per glucose unit if PYR continues down this slow aerobic pathway (17, 66).

LAC formation from PYR. Lactic acid (LAC) is a fixed, non-volatile acid (pK = 3.86) that rapidly dissociates a hydrogen ion (H^+) at physiological pH = 7.4, yielding H^+ and the lactate salt. LAC itself is neither a fatigue substance nor a dead-end metabolite. It is, in fact, a valuable substrate for both cardiac and skeletal muscle (especially types I and II_a), as well as an important gluconeogenic precursor during prolonged exercise and recovery (7, 78, 98, 99). Indeed, carbon flux through the lactate pool may be an important means by which anabolic-catabolic processes are coordinated, thus allowing for an integrated exercise response (7). However, the disruption in pH secondary to H^+ accumulation inhibits glycogenolytic-glycolytic enzyme activity (8, 14); displaces Ca^{2+} at the troponin-tropomyosin complex, thereby inhibiting actomyosin cross-bridge formation (38, 80, 100); and stimulates pain receptors, resulting in one manifestation of fatigue (57, 60, 87, 100). Intracellular pH may approach 6.2 to 6.3 during intense exercise (86, 87), and therefore such fatigue may be another protective mechanism that prevents cellular proteins from being denatured.

A variety of factors influence LAC formation from PYR and its subsequent accumulation in the tissues and blood. These include substrate selection, with carbohydrate becoming the preferred substrate over fat; increasing type II_b (fast glycolytic) fiber recruitment; positive catecholamine modulation of glycogenolysis via an adrenal response; and a mismatch between the blood lactate (LAC_b) rate of appearance (R_a) and a rate of disappearance (R_d) with increasing exercise intensity (7, 78, 98, 99). This latter response is largely a function of mitochondrial activity, which may be saturated with substrate beyond a certain exercise intensity; of lactate dehydrogenase (LDH) isozyme activity; of blood shunting away from the liver and kidney, major sites of LAC_b uptake, with increasing exercise intensity; and possibly of relative fiber compositon and capillary density (7, 78, 98, 99).

LAC production tends to be highest in type II fibers (22, 23, 100), which are higher in glycogenolytic-glycolytic enzyme activity than are type I (68). LAC diffuses into adjacent fibers, as well as into the plasma (22, 23, 77, 100). There is also clear evidence of a carrier-mediated LAC transport mechanism in mammalian cardiac tissue (15, 75). Indeed, a similar mechanism has been proposed for skeletal muscle in several studies of isolated muscle *in vitro*, contributing an estimated 50 to 75 percent total LAC transmembrane uptake over the physiological [LAC] range (59, 63, 76).

Clearance kinetics. It is important to note that intramuscular, not blood, PYR-LAC kinetics are the limiting factor during exercise. $[PYR + LAC]_m$ is linearly related to pH_m (86, 87). Peak $[LAC]_m$ (up to 30 $mmol \cdot kg^{-1}$ wet weight) can be achieved within one to two minutes of intense exercise, while peak $[LAC]_b$ (20 $mmol \cdot kg^{-1}$) can occur several minutes post-exercise (50, 57, 60, 86, 87). $[LAC]_b$ continues to increase linearly until essentially reaching equilibrium with $[LAC]_m$, at which point it follows a parallel decline in concentration. Note that resting $[LAC]_b$ is about 1 $mmol \cdot kg^{-1}$.

Systemic LAC clearance is a function of R_a and R_d. Post-exercise LAC clearance operates as a first-order exponential function, independent of concentration, and is markedly affected by both training status and type of recovery (active versus inactive; **Figure 3**) (33, 39, 49, 53, 78, 86). This clearance rate increases with relative workload up to an optimal range of 50 to 70 percent $VO_{2\ max}$, with $t_{1/2}$ LAC_b clearance ranging from 15 to 25 minutes during inactive recovery and approaching seven minutes during active recovery (i.e., exercise relief) in enduarance-trained subjects (33, 39, 49, 53, 86).

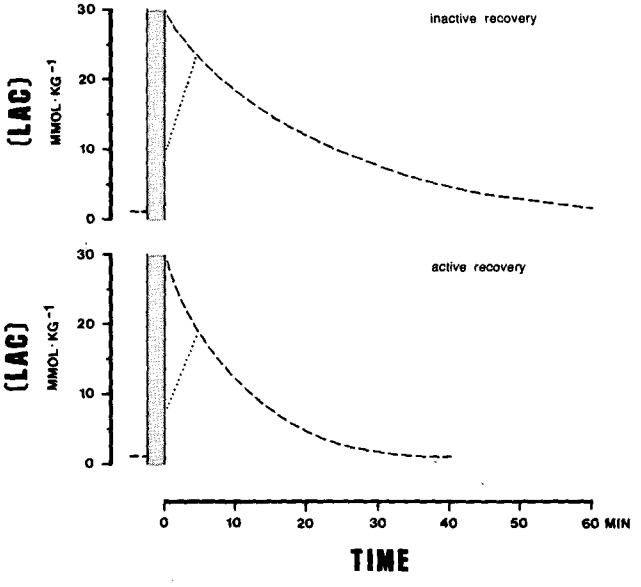

Figure 3. The time course of LAC clearance: [LAC] as a function of recovery time and type, and of training status (33,39,49,53,78,86). The shaded vertical bar represents a one-minute interval workload during which LAC accumlates in the tissues (dashed line) and blood (dotted line). a. During inactive recovery in untrained subjects, the $t_{1/2}$ LAC clearance may exceed 15 minutes. b. During active recovery, and with chronic aerobic training, $t_{1/2}$ LAC clearance approaches seven minutes.

Training. It has been well documented that aerobic-type training decreases the $t_{1/2}$ LAC clearance. This is due largely to increased mitochondrial activity (and therefore pyruvate oxidative capacity) (40, 42, 48, 91, 96), increased capillary density (96), and a shift from "M-type" LDH_{4-5} to "H-type" LDH_{1-2} isozyme activity, and thus a decreased tendency for LAC formation from PYR (95), in trained muscle.

Endurance training and preliminary aerobic-type warm-up activity have also been shown to reduce early lactate formation at the onset of a typical relative or absolute square-wave exercise bout. This is achieved largely via an improved $\dot{V}O_2$ on-response (11, 12, 82, 83), and thus a decreased dependency on anaerobic contribution to the exercise-onset deficit. The $t_{1/2}$ VO_2 on may exceed 40 seconds in untrained subjects, with a tendency for higher values in the upright (versus supine) position, and with increasing exercise intensity. However, the $t_{1/2}$ $\dot{V}O_2$ on may approach 20 seconds in trained and primed (warmed-up) subjects (11, 12, 82, 83). Twenty seconds appears to be the absolute limit established by muscle-to-lung blood transit time, and the kinetics of the respective rate-limiting enzymes in the working tissues (11, 12, 82). In contrast, the $\dot{V}O_2$ off-response ($t_{1/2}$ $\dot{V}O_2$ off 23.5 seconds) is not significantly affected by training status, posture or exercise intensity (11, 12, 82).

Thus, we see a second indication of the potential value of oxidative metabolism for the anaerobically oriented athlete. It should be possible, again within limits, to minimize LAC formation and maximize the LAC clearance and PYR oxidation rates via aerobic-type training, warm-up and recovery. More research is needed to determine the absolute and optimal limits of these training responses. However, it would seem wise to employ supplemental aerobic-type training in moderation for the anaerobic athlete, with the bulk of such training in the early off-season, and perhaps no more than weekly maintenance during pre- and in-season phases.

Training Kinetics

Figure 4 illustrates the author's conception of the theoretical contribution of the bioenergetic systems to acute square-wave sprint (**Figure 4a**) and interval (**Figure 4b**) exercise bouts based on existing data. Note that the time course and magnitude of the physiological responses illustrated are subject to some variation, based on genetic endowment and training and nutritional status. These diagrams represent two of several commonly used approaches to training the

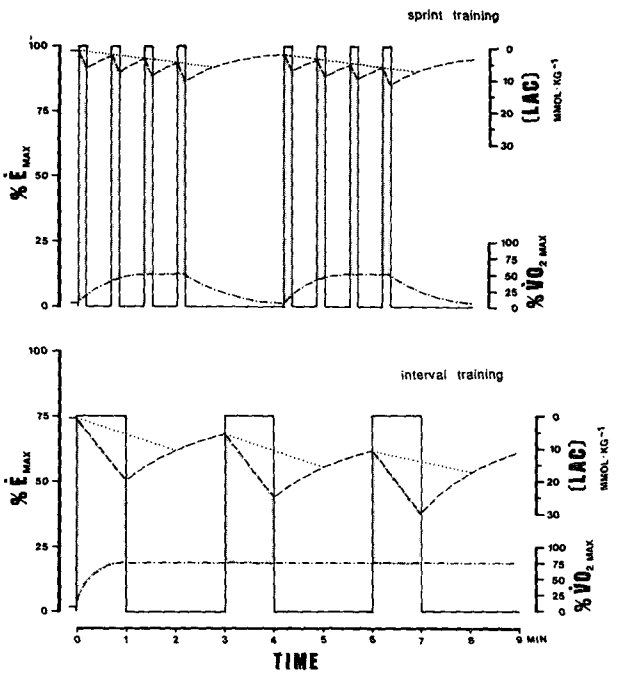

Figure 4. a. Sprint training kinetics. The vertical bars (solid line) represent 10-second square-wave sprint workloads of maximal intensity (100 percent E_{max}), with a 30-second recovery between repetitions. HEP pathway contribution, represented by the shaded area between the $\dot{V}O_2$ (dash-dot line) and inverted [LAC] curves)(LAC_m = dashed line; LAC_b = dotted line), is maximized. A 1:3 exercise:relief ratio is illustrated. **b. Interval training kinetics.** The vertical bars (solid line) represent 60-second square-wave interval workloads of three-quarter intensity (75 percent E_{max}), with a two-minute recovery between repetitions. Glycolytic contribution (represented by the inverted [LAC] curves) is maximized with successive repetitions. A 1:2 exercise:relief ratio is illustrated.

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anaerobic pathways. Hybrid methods (e.g., tempo, fartlek) are often employed by endurance athletes due to their increased involvement of oxidative metabolism. This being the case, they are worth noting, but are beyond the scope of this discussion.

Discussion: Practical Applications

Repetition intensity-duration. It is generally accepted that the HEP and glycogenolytic-glycolytic pathways are best trained by increasing exercise intensity or rate rather than duration (3, 24). Since heart rate is a poor indicator of exercise intensity at supramaximal intensity, the conditioning coach must provide a goal pace (based upon predetermined target training times) at which the athlete is to be trained and tested (24, 92, 107). Extending the repetition duration beyond the thresholds dictated by the principles of bioenergetic system recruitment tends only to result in poor exercise quality and inordinately long recovery time requirements (3, 24, 72), and is thus contraindicated unless specifically dictated by the competitive activity-inactivity profile. This supports the concept of exercise quality (rather than quantity) being the critical factor in eliciting high-intensity training adaptation (3, 24).

Exercise:Relief. A second method of upgrading exercise quality involves a systematic decrease in the denominator of the exercise:relief fraction, allowing for the decrease in $t_{1/2}$ LAC clearance (and possibly phosagen repletion) with increasing fitness levels. One method during interval training is to allow the heart rate to recover to a specific range (e.g., 120 to 140 beats per minute) between repetitions, depending on training status (32). Empirically, this coincides with an exercise:relief ratio of 1:4 at the initiation of the metabolic training phase tapering to 1:2 or possibly 1:1.5 after four to six weeks (24, 28). As previously discussed, active recovery or work relief facilitates this recovery process.

In contrast, the sprint training exercise:relief ratio may taper from 1:5 to 1:3 over several weeks, with inactive recovery or rest relief allowance between repetitions in order to facilitate maximal phosphagen repletion (24, 28). A minimum of two minutes should be allowed between sets to accommodate such repletion (and therefore maximize work quality).

Total exercise volume. Information on the optimal exercise volume and duration per training session is scarce (26, 27, 46, 107). A range of 10 to 20 repetitions per workout has been proposed for the sprinter; 1.5 to 2.0 and 2.0 to 3.0 times total racing distance have been suggested as appropriate training volumes for middle distance runners performing fast (110 to 440 yard) and slow (110 to 880 yard) interval running workouts, respectively (107). However, these guidelines are based upon empirical evidence and are intended for competitive runners, and thus may be of limited use for other athletes or events. Until more information is available, manipulation of this variable is best left to the discretion of the practitioner.

Training frequency. The optimal frequency of sprint-interval training is currently the subject of some discussion. One study has documented similar significant increases in metabolic and functional performance parameters in previously untrained subjects regardless of training frequency (two versus four days per week) (67). However, such data on trained subjects are lacking.

In light of the concept that improved training status tends to dictate decreased training frequency (24), as well as the fact that metabolic conditioning may be coincident with other (e.g., resistance) training modalities, it would seem wise to limit conditioning sessions to two to three days per week for trained athletes. This should allow the coach and athlete to concentrate on work quality, allow for sufficient recovery time between workouts, and hopefully avoid an overtraining syndrome. While overtraining is a rather nebulous phenomenon, these guidelines are in keeping with the available data regarding gylcogen repletion following an acute high-intensity exercise bout (70, 84). The clever practitioner will account for these variables and will implement appropriate agility and skill-technique drills that provide part of the conditioning stimulus, thereby optimizing training time usage.

Program duration. The optimal duration of a preseason metabolic conditioning program is also open to discussion. Significant metabolic and functional adaptations to sprint-interval training have been documented in as little as four weeks in previously untrained subjects (21, 29, 31, 67, 85, 91, 94, 104). Motivational and training plateau factors may contraindicate preseason metabolic conditioning programs in excess of a 12-week duration (72). Due to the lack of available data on trained subjects, it would seem wise to implement conditioning programs within these guidelines until more information is available.

Value of resistance training. Preliminary aerobic-type training, while good metabolic preparation, may be insufficient architectural preparation for the mechanical power production-absorption profiles characteristic to high-intensity anaerobic training and competition. Ballistic stretch-shortening cycle activities such as sprint-running involve powerful eccentric contractions characterized by diminished motor unit activation relative to concentric contractions of similar force and velocity (5, 79). The resultant increase in force per muscle fiber has been associated with temporary ultrastructural muscle damage, myofibrillar disorganization (i.e., z-line streaming), soreness and strength reduction (10, 24-37, 97). Training that involves a controlled eccentric contraction component has been shown to have a prophylactic effect, significantly reducing the magnitude of these negative responses (10, 35). Thus, it is tenable that concentric-

eccentric resistance training has potential value in the preparation for such activity.

Clearly, it is not appropriate to train sprinters like competitive weightlifters or powerlifters. However, the merit of resistance (and flexibility) training is apparently based on these observations. It is reasonable to propose that such training can provide the muscle and connective tissue integrity needed to augment athletic performance and minimize the incidence and severity of overuse-type soft-tissue injury (2, 25), of which eccentric-induced muscle damage can be considered one type. It is also generally accepted that resistance training is prerequisite to the explosive-type movements (e.g., plyometrics, olympic style lifts) in common use during the speed-strength and strength-power phases of many current training programs (4).

Training progression. While it is not the purpose of this paper to outline a specific periodization paradigm, it is important to note that the bioenergetic systems are to be trained progressively (1, 3). The importance of oxidative mechanisms for anaerobic metabolism implies that a fitness base be established via endurance-type training prior to the pursuit of higher intensity training activity. In practice, training phases may overlap one another and should decrease in length with increasing fitness levels (1, 72).

In-season maintenance. Depending on the demands of seasonal skill-technical training and competition, there may be a deconditioning effect in athletes who neglect a conditioning maintenance program. Thus, metabolic (and resistance) training may be required on a weekly maintenance basis in-season, and possibly as often as every four to five days, depending on the competitive schedule. Perhaps the practitioner's greatest challenge at this point is to allow for sufficient recovery time between training sessions and competitive events such that preseason performance levels are adequately maintained without overtraining or undertraining the athlete(s).

Testing. Several in-depth reviews of the various approaches to laboratory and field testing for anaerobic power and capacity are available to the coach (6, 65, 92, 106). Briefly, laboratory tests usually consist of force-velocity, vertical jump, staircase and cycle ergometer tests for anaerobic power; and all-out or constant-load ergometric tests, with possible O_2 debt, O_2 deficit or peak $[LAC]_b$ measurements, for anaerobic capacity (65, 92, 106). Note that some laboratory tests are useful in field situations as well.

Field tests can be categorized as follows (6, 92): a single explosive movement of less than 1-second duration (i.e., muscular power; e.g., vertical jump), a continuous sequence movement of less than 15 seconds duration (i.e., alactic power or capacity; e.g., 40- to 100-yard dash), and a continuous sequence movement of 15 to 120 seconds duration (i.e., lactic power or capacity; e.g., 220- to 880-yard dash). Timed, repeated multiples of each of these three approaches are perhaps more useful to the conditioning coach since they provide important data on fatigability as well as on metabolic power and capacity.

It should be pointed out that laboratory testing may have the advantage in terms of data collection accuracy, and thus has great value in a research setting. However, field testing obviously has the advantage of specificity for the field-trained athlete. There is no one test, either in the laboratory or field, that can provide data on all relevant components of anaerobic metabolism and performance. Based on scientific knowledge and practical experience, the conditioning coach must design and administer a valid and reliable test battery, specific to the training program utilized and to the competitive event, at strategic points throughout the competitive year such that the effectiveness of both pre- and in-season conditioning programs can be objectively evaluated.

Discussion: Functional Significance

The peripheral responses consequent to endurance, interval and sprint training are well documented in literature, and are beyond the scope of this review. Unfortunately, less effort has been devoted to documenting the correlation between specific physiological markers and anaerobic performance parameters. While such data are scarce, the significance of specific training responses is evident in light of the following relationships:

Strength. Absolute muscular strength has been highly correlated with muscle cross-sectional area (103) while high-velocity force production capability and muscular power have been highly correlated with percent type II fiber compositon (64, 103). Thus, force production capability is determined more so by fiber type composition than by metabolic profile, especially at high contraction velocities (103). The athlete so endowed genetically, or by selective type II fiber hypertrophy, has a distinct advantage in such events. Sprint training may positively affect these parameters, in light of a hypertropic response characterized by an increase in type II:I fiber area ratio observed in previously untrained subjects (104). The effects of interval training on force production capability have not been investigated.

Endurance. Local muscular endurance, recovery and aerobic capacity have been highly correlated with capillary density, and with key aerobic and anaerobic

enzyme activities (103). Specifically, fatigability is directly related and recoverability inversely related to the LDH:TCA cycle enzyme activity ratio (103). Thus, metabolic profile (i.e., high LAC formation capacity alone, or in combination with low aerobic power) rather than fiber type composition tends to determine local muscular fatigablility and recoverability (103). While endurance training has been shown to positively affect this ratio, as well as the LDH isozyme pattern and capillary density, both sprint and interval training have been shown to have no effect on, or increase, both fractions (85, 95, 104). More research is needed, as the exact functional nature of such a response is unclear.

LAC. $[LAC]_b$ has been very highly correlated with percent type II fiber composition, glycolytic enzyme activity and the PFK:TCA cycle and LDH:TCA cycle enzyme activity ratios (64, 101, 103). Likewise, the exercise intensity required to produce the "onset of blood lactate accumulation" is inversely related to these factors, as well as to capillary density (64, 101, 103). Interval training may have a positive effect in light of a decreased $[LAC]_b$ observed at submaximal exercise intensity in previously untrained subjects consequent to training (21, 67, 85), and of an increased cellular buffering capacity in previously untrained subjects (88, 93) as well as in elite anaerobic athletes (81). Note that endurance training elicits no such adaptation in buffer capacity (81, 93). The effect of sprint training on these relationships is unknown.

Running velocity. Absolute running velocity has been directly related to MK and CPK activities, and inversely related to total LDH activity (64). Thus, the enzymes involved in rapid ATP turnover and PYR-LAC interconversion, rather than muscle strength or fiber composition, tend to be critical determinants for running speed (64). While sprint training appears to have a positive effect on MK and CPK activities (with potentially increased myofibrillar contractility and ATP resynthetic capacity, respectively) (13, 41, 74, 104), again the effect of interval training is unclear in light of its effect on LDH activity (85, 95) and of its apparent lack of effect on MK and CPK activities. Since these enzymes (and contractile proteins) exist in fiber type-specific activities and isoforms (68, 103, 105), which are ultimately determined by innervation, (58, 90) movement velocity can be considered an indirect function of motoneural input, and is apparently modifiable through training.

Summary

Conceptually, these research data tend to support the functional training adaptations we are acquainted with empirically. These can be generally classified as follows: increased anaerobic power and capacity, increased recoverability and decreased fatigability.

Anaerobic power-capacity. If we refer back to **Figure 1**, we might expect to see the HEP and glycogenolytic-glycolytic curves grow taller and/or wider with specific training. The on-off responses may be positively affected, and may achieve greater absolute or relative values; thus, potential increases in various combinations of power, capacity and total work production. This is in agreement with functional training responses observed in numerous studies (11, 12, 21, 29, 31, 64, 67, 74, 82, 83, 85, 94, 103, 104). As the athlete attains increasingly higher levels of fitness, we can expect the magnitude of these responses to decrease.

Recoverability. As the abilities for substrate resynthesis (proposed; **Figure 2**) and metabolite removal (**Figure 3**) (40, 42, 48, 91, 95, 96) improve with training, we can expect more rapid recovery from intense activity. The magnitude of this response should also decrease with improved training status.

Fatigability. It is important to realize that absolute performance parameters are a function of, and are therefore limited by, a complex interaction of neurogenic and myogenic factors. Significant biochemical and structural adaptations may be functionally attenuated by the limitations of motor unit recruitment, rate coding and synchronization patterns, and vice-versa. While it is generally accepted that training can positively affect both neuromuscular components, we cannot help an athlete to exceed his or her genetic limits. For example, it may be unrealistic to expect a conditioning program to decrease an elite sprinter's 100-meter dash time by 0.5 second. However, he or she may recover more rapidly from that effort, and may maintain significantly higher performance levels over repeated efforts. Thus, while there may (or may not) be significant improvement in an athlete's single, all-out performance effort consequent to a metabolic conditioning program, his or her fatigability is functionally decreased. This is of great value to the athlete when performing repeated supramaximal efforts with limited recovery time allowances.

Conclusion

Training has come to be synonymous with endurance- or aerobic-type activity in much of the existing literature, exclusive of higher intensity anaerobic-type exercise. While both types of training involve a complex interplay of many physiological variables, the acute and chronic responses to each are quite distinct.

In this article, the strategy underlying program

design considerations for sprint-interval training is reviewed in light of the existing data regarding functional significance. It is hoped that this review will prove useful to the coach and athlete preparing for competition. Attention is also called to those areas where there is some debate or nescience, or simply insufficient documentation to support certain concepts or practices. While our empiricism and intuition as practitioners may be soundly based and are indeed valuable attributes, they alone are not enough. A thorough scientific understanding of the physiology of exercise is essential for the conditioning coach to effectively bridge the gap between theory and practice, and thereby augment athletic performance to an optimal degree.

In closing, several issues are in need of further research if we are to expand our working knowledge of the neuromuscular conditioning scheme:

1. The trainability and functional significance of phosphagen repletion kinetics during recovery.

2. The mechanism(s) and kinetics of the proposed lactate transmembrane carrier in skeletal muscle. Of particular interest are its physiologic importance in different fiber types, precise contribution to total lactate transport and trainability.

3. The relative compatibility of coincident metabolic and strength-power training. This will require extensive investigation of the interrelationships between pre- and posttraining cellular markers, training stimuli and functional parameters, respectively:

a. fiber type-specific difference in enzymatic and substrate activities (i.e., the presence or lack of dilution effects resulting from dissociated adaptations in sarcoplasmic and contractile compartments);

b. standardized manipulation of acute and chronic training variables (especially intensity, duration, frequency, volume, exercise:relief); and

c. indices of metabolic power and capacity, with respect to the pre-training status of the subject and its effect on the time course, magnitude, and mechanism(s) of adaptive response(s).

4. It would be difficult to overstate the fundamental importance of cytosolic calcium in initiating and modulating cellular events in striated muscle. Calcium is intrinsic to the structure, energy metabolism and contractile response of skeletal muscle, and indeed discussion of its significance during rest and exercise would be impossible in this paper due to the remarkable breadth of the subject. However, it must be noted that ongoing research of calcium-mediated processes (and their plasticity) is certain to yield profound insight into peripheral training adaptations and fatigue mechanisms.

Acknowledgements

The author wishes to gratefully acknowledge the expertise of Dr. David Pendergast (Department of Physiology, State University of New York at Buffalo), without whose friendship and professional insight this project would not have been possible; of Dr. Robert Mazzeo (Department of Kinesiology, University of Colorado at Boulder); and of Dr. Gary Klug (Institute of Neuroscience, University of Oregon at Eugene).

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