Dose/Response Effects of Exercise Modeled from Training : Physical and Biochemical Measures

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This study has measured the pattern of elevated serum enzyme activity (ESEA) during extended daily training in a dose-response manner and compared ESEA to the pattern of accumulated fitness and fatigue predicted from a mathematical model previously described. Blood samples were taken regularly during the study from each subject and the activity of lactate dehydrogenase (LDH), creatine kinase (CK), and aspartate aminotransferase (AST) in the serum was measured.

Although no single physiological/biochemical correlate of the hypothesized fatigue compartment of performance is firmly identified it is significant that the pattern of variation of model fatigue and ESEA throughout training were similar although slightly out of phase. With continued hard training, model fatigue began to plateau and concomitantly ESEA declined exponentially from its initial high value in early training. During relative rest throughout a tapering period following training both ESEA and fatigue reverted quickly towards baseline and follow the similar but earlier time course in blood of a degradative membrane enzyme phospholipase A2 observed in clinical studies.

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Recent papers in the literature draw attention to the maintenance of performance ability for a considerable period after training is either markedly reduced or increased. These papers reflect a growing interest in the dose/response effects of exercise. However the majority remain empirical lacking a firm theoretical base, and are individually, quite contradictory. Thus Coyle et al. (1984) observed a decreased \dot{VO}_2 max of 7 % after 12 days of minimal activity following 1 year of training in a group of well-trained runners. Houmard et al. (1990) found only an insignificant loss of $\dot{V}O_2$ power (1.4 %) from a similar level of attained $\dot{V}O_2$ max following a 70 % reduction in training throughout 2 weeks, in experienced runners. On the other hand Hickson et al. (1982) first found $\dot{V}O_2$ power and physical endurance on treadmill and cycle ergometer increased throughout 15 weeks of reduced training following 10 weeks of training and later found almost the exact opposite (Hickson, 1985). In addition Kirwan et al. (1988) found elite swimmers able to double their training for 10 days without detriment to their physical performance. Costill et al. (1991) have recently suggested that increased training does not enhance performance in swimming. Even for training lasting 25 weeks little absolute improvement in

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swimming endurance and power was noted after the 8th week in 2 groups of swimmers one of which trained, for a period, twice as much as the other. While some of these recent empirical physical, biochemical and physiological observations on training and detraining in humans mirror relationships already quantitatively evident in the growth and decay of cellular, biochemical indices accompanying training stimuli in animal studies (Holloszy 1967, Booth et al 1977, Baldwin et al. 1977, Dudley et al., 1982; Terjung 1979) considerable other of these data cannot be rationalized with the training producing them. This is probably because the complex, cellular activity which is induced at a microscopic level by training (Mader 1988, Pette and Staron 1989, Pette 1989) is often quite out of phase with the resulting structural and physiochemical changes grossly observed. The former still remain relatively inaccessible to serial, quantitative investigation in humans.

Systems Analysis

Despite this difficulty some insight into the above processes may be gained from the technique of systems analysis by making informed guesses (either from previous literature or by experiment); about the nature of the system being studied; about its components and relationships and about the value of the system's parameters (time delays, time constants, steady state limits, etc.). This characterization may be aided by in vitro or animal studies of sub-systems of the whole. How well the initial configuration of the system and parameter estimation is made determines how successfully the systems model mirrors experimental reality and how well it rationalizes underlying physio-chemical mechanisms of action (Stegemann 1971, Johnson 1991, Barstow et al. 1991, Mader 1988, Fukuba et al. 1989).

The present report uses a quantitative mathematical model to relate physical and biochemical responses to the training determining them.

Systems Model of Training

The systems model employed in this paper (see Appendix) specifies two general components symbolizing induction/synthesis or repression/degradation processes. These are respectively named fitness and fatigue, and are estimated from the quantity of daily training. The components are then used to predict physical performance (Calvert et al. 1976; Morton et al. 1990, Banister 1991, Fitz-Clarke et al. 1991). Physiological and biochemical definition of the model components has been attempted previously using serum iron and hormonal responses to training respectively (Banister and Hamilton 1985, Busso et al. 1990). Exercise-induced elevated serum enzyme activity (ESEA) is now studied, in order to evaluate its temporal relation with the hypothesized responsive elements influencing performance described by the model.

Elevated Serum Enzyme Activity (ESEA)

It seems evident that some pathophysiology is attendant upon demonstrated ESEA, since the latter condition is also often accompanied by medical emergency in cardiac muscle, following myocardial infarction (Lee and Goldman 1966; Apple & McGue 1983; Apple and Rhodes 1988) and by soreness, fatigue or loss of function in skeletal muscle following marathon running (Dressendorfer and Wade 1983; Hagerman et al. 1984; Lignen et al. 1988; Noakes et al. 1982; Siegel et al. 1980) and following a variety of other forms of acute muscle stimulation (Armstrong et al. 1983; Arrko et al. 1983; Clarkson and Tremblay 1988; Galun et al. 1988; Newham et al. 1986b; Sanders & Bloor 1975). Peak activity in ESEA may occur either 6 to 24 hours (Donelly et al. 1988; Galun et al. 1988; Schwane et al. 1983; Tiidus and Ianuzzo 1983) or 4-6 days (Clarkson & Tremblay 1988; Newham et al. 1986a) after a single acute stressful event depending on the particular enzyme.

Myoglobin, normally found only in muscle cells is a specific index of muscle breakdown when found extra-cellularly. Schiff et al., (1978) found a linear correlation between serum myoglobin concentration and serum LDH and CK elevation. Fine structural studies of sore muscle (biopsied) by Hagerman et al., (1984) showed that both pre-marathon training and marathon running itself induced focal muscle necrosis and degenerative changes similar to rhabdomyolysis and myoglobinuria. Frieden (1984) found no necrotic fibers in human muscle following intense eccentric exercise, but found disruption (streaming) of the myofibril Z band. Both Apple and Rhodes (1988) and Janssen et al. (1989) have estimated exercise-induced skeletal muscle damage quantitatively from serum CK elevation.

Although the concept that exercise-induced ESEA induced by exercise is related to loss of function, injury or overtraining is not without criticism (Kirwan et al. 1988, 1991 and Costill et al. 1991) it certainly seems possible for it to be a useful physiochemical phenomenon readily identifiable with both disruptive and adaptive processes (fatigue and fitness) and therefore associated with the time course of change in performance, induced by a strenuous training program. In light of the significant biphasic character of the change in ESEA accompanying many types of both acute and persistent exercise it is important to define the dose-response characteristics of exercise eliciting such effects and possibly use the relationship as a guide to formulate the necessary amount of training to produce optimal performance, safely.

METHODS

Subjects and Training Procedures

Two subjects (RHM and EWB) trained at least once each day, for twenty eight consecutive days and then tapered for 32 days. Before commencing the program each was medically approved to participate and signed the informed consent approval required by the University Ethics Rules on Human Experimentation. Previous to beginning training RHM and EWB were moderately fit when measured by an incremental test to exhaustion (Table 1). Each subject trained 7 days per week, had a morning blood sample taken for analysis twice per week, and completed a timed run over a prescribed distance, to the best of their current ability at least twice per

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Subject	Sex	Age	Height	Weight	VO _{2max}
	-	yr	cm	kg	$ml \cdot min^{-1} \cdot kg^{-1}$
EWB	М	57	180	86	48.8±2.1
RHM	Μ	42	178	73	51.3 ± 1.7

week. This was termed a Criterion Performance (Cp). Cp was expressed by a points score X (T), calculated from the Cp times, which was given by the equation:

$$X(T) = 294.7 \times 1n \frac{11.9}{(11.9 - T)}$$

where T = Cp expressed in decimal minutes (Morton et al. 1990). The Cp was usually completed at the beginning of a regular training bout and was thus incorporated in training. Training commenced on a cycle ergometer for a period of 45 minutes per day but on the sixth day activity was changed to running for a similar period. On the 11th day twice-a-day periods of training were instituted each lasting from 40 to 45 minutes and this regimen was continued up to the 28th day when a period of relative rest, or taper began which continued for a further 32 days. During this period, the Cp tests were the only physical activity performed.

Blood Sampling and Analysis

Overnight fasting blood samples were taken one or two times per week from each subject between 8 a.m.-10 a.m. after a period of quiet sitting. Samples were allowed to clot and were centrifuged to separate serum. Serum was refrigerated at -20 °C until analysis, within 2 days. Total lactate dehydrogenase and aspartate transaminase activity in the serum was measured on a Roche Cobas Bio analyzer at 30 °C using Sigma Reagent according to the proce348

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dure of Henry et al. (1960). Total creatine kinase activity (N-acetylcysteine activated and optimized to the standards set by the "Deutsche Gesellschaft fur klinische Chemie") was measured on a Roche Cobas Bio analyzer at 30 °C using BMC reagent (Boehringer-Mannheim CmbH, Mannheim, West Germany) according to the procedure of Szaz et al. (1976). The coefficient of variation in repeated daily analyses of a know standard of these enzymes ranged between 3-5 % during the period of the study.

RESULTS

Modeling Physical Performance

Figure 1 shows the effect of modeling the daily training impulse w(t) (top panel) to achieve a 'least squares' best fit of predicted p(t) to real criterion performance Cp (bottom panel) measured throughout the whole period of training and tapering. The beginning (default) values for decay time constants $\tau \ 1 \ \& \ \tau \ 2$ were 45 and 15 days respectively. These defined the impulse response of fitness g(t) and



Fig. 1

1 Showing (top) ; the daily training impulse measured from the duration and heart rate elevation of training, (middle); Growth in model fitness and fatigue (derived from the training impulus), (bottom); predicted performance p(t) (full line) plotted with criterion performance (dashed line joining crosses) throughout a period of training and tapering for two subjects derived from iterative computer modeling of predicted performance from training against real performance (Cp) to achieve at least squares fit. (Reproduced with permission from Morton et al., J.Appl.Physiol., 69: 1171-1177 1990)

fatigue h(t) (Equations 3 in Appendix) as each declined to a residual value between training sessions. The arbitrary, default, factors k1 and k2 weighting the relative contribution of fitness g(t) and fatigue h(t), to predicted performance p(t) were 1 and 2 respectively (Equation 2 in Appendix). The centre panels of Fig. 1, for each subject, show the final, respective time course of the model components, fitness and fatigue, defined from successive computer iteration of Equations 2 and 3 in Appendix until predicted performance p(t) matched the temporal pattern of actually measured performance (Cp) during the period of training and recovery (approx. 60 days). Model parameter definition, (τ 1, τ 2, k1, k2) is shown in Table 2. The r² values for regression of serially measured criterion performance (Cp) on model prediction from training p(t) were .71 and .96 respectively for each subject. These values were both highly significant at the $p \leq .001$ level.

 Table 2.
 Summary of model constants and statistics for least-squares regression of criterion performance on predicted performance

Subject	$ au_1$	$ au_2$	k1	k2	r²	FStat	df	Р	SE
EWB	50	11	1	1.8	0.71	59	4,21	≤0.001	±13
RHM	40	11	1	2.0	0.96	252	4,18	\leq 0.0001	± 7

It is assumed that when good coincidence between predicted and measured performance is achieved by modeling, the temporal pattern of both fitness and fatigue becomes a valid representation of the real time course of these hypothesized components.

Figure 2 shows the fatigue line, for each subject, abstracted from Figure 1 and plotted against the time course of ESEA for LDH (top), CK (middle) and AST (bottom) measured from serially taken blood samples throughout training. In each case ESEA parallels, the time course of fatigue although somewhat out of phase with it. After rising rapidly during the early training phase ESEA peaked by the 11th day of training and then declined between the 11-15th day from its initial high value and began to plateau, at a level still well above baseline (control). The model's fatigue component also plateaued despite continued heavy training, then declined exponentially during the taper period. On cessation of formal training from day 28 onwards ESEA closely paralleled the decline in the fatigue component of the performance model.

DISCUSSION

The temporal change in fatigue rather than fitness ($\tau \ 2 \ << \tau \ 1$) identifies better with the rapid return of ESEA to baseline in all of the enzymes studied when hard training ceased.

Single Bout exercise-induced ESEA

Previous reports have shown a similar but crucially different (see below, repeated-bout exercise), biphasic temporal response of ESEA to a single bout of exercise of a brief (Newham et al. 1986a, Salminen and Vihko 1983) or extended form (Newham et al. 1986b, Dressendorfer and Wade 1983) as is demonstrated in this report. In single-bout exercise ESEA usually rises to a peak ranging from 5 (Armstrong et al. 1983, Donelly et al. 1988) to 45 times (Newham et al. 1986, Schwane et al. 1983) the baseline of activity depending on the enzyme, between 6-24 hours after exercise. This single-bout response of ESEA is followed by a rapid exponential decay of enzyme activity towards the baseline between 48-90 hours later if no further exercise intervenes.

Repeated bout exercise-induced ESEA

Repeated bouts of standard exercise on succeeding days during an extended period however may be expected to complicate the time course of the singlebout, exercise ESEA response. Thus, depending upon the interval between training sessions, successive exercise stimuli will induce new cellular enzyme loss from further fibre and membrane damage, while leakage induced by a preceding stimulus (within 6-24 hours) is still continuing. When adapt-

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Fig. 2 Showing the temporal relationship of time course of fatigue for two subjects from Figure 1 with ESEA. In both subjects (left and right panels) the elevation of serum enzyme activity (ESEA) above baseline, precedes the time course of model fatigue somewhat out of phase with it. Adaptation of ESEA to continuing hard training is shown by the slow decline in ESEA towards a plateau above baseline. In both subjects when peaking or tapering commences ESEA and fatigue both quickly revert towards a baseline level with a parallel time course.

ing muscle begins to resist further damage, then subsequent stimuli will induce less enzyme loss and the initial rapid rise of ESEA will slow, peak and then decline to plateau at a new level possibly reflecting the existing degree of muscle cell adaptation to the training stress. This is exactly the effect apparent in Figure 2 and a similar relationship may be observed in several other published reports of ESEA induced by sustained daily activity. Dressendorfer and Wade (1983) measured this effect in male marathon runners engaged in a 20 day 500 km stage race. Apple and McGue (1983) observed a similar sub-peak plateauing of CK, still 3 times the baseline value, for young men during extended training. An earlier study of Sanders and Bloor (1975) indicated clearly the recursive nature of the rise and fall in daily ESEA values in proportion to the size of the daily stimulus since the training stimulus was continuously varied and blood was sampled immediately prior to, and after training each day.

Relation of ESEA to Phospholipase A2 activity

The relation of elevated serum enzyme activity to the defined, degradative model component of fatigue in this paper is consistent with the known degradative action of a widely distributed, sub-set of membrane-bound phospholipases seemingly induced by exercise. Collectively these are known as phospholipase A2, PLA2 (Chang et al. 1987). Various Physiological stimuli activate PLA2 and in a series of papers of Jones (1983, 1984) and Jackson (1984) attributed a role in the release of intracellular enzymes to blood, to a calcium-activated PLA2 hydrolysis of mitochondrial and other membrane phospholipids. This action gives rise to lysophospholipid and free fatty acid and their derivatives (prostaglandins, leukotrienes, and platlet activating factor); all widely implicated in the human inflammatory response. This latter effect, which can induce fibre damage and/or increased cell surface membrane permeability to soluble enzyme, is possibly a prime, degradative action, on cell membrane mediating the observed exercise-induced ESEA.

We have been unable to ascertain, from the literature, whether there is a concomitant serum phospholipase A2 elevation accompanying exerciseinduced ESEA in humans although in an isolated mouse muscle preparation, LDH release, induced by electrostimulation, was inhibited by the phospholipase A2 inhibitors dibucaine and chlorpromazine (Jackson et al. 1984). An uncontrolled elevation of serum PLA2 has also been observed to precede other developing patterns of cellular disruption such as post-traumatic, pulmonary insufficiency and septicemia in patients suffering from multiple organ failure (MOF) (Koeniger et al. 1989). In these patients there was overall, a better positive prognosis following remission of high serum PLA activity, either spontaneously or in response to steroid therapy (Koeniger et al. 1989, Baur et al. 1989). A hypothesized temporal relation between exercise-induced serum PLA2 activity, (modified from its time course observed in MOF), ESEA and the model fatigue component associated with these degradative actions is shown in Figure 3. Model fatigue parallels, the time course of both phenomena particularly the rapid decrease to baseline on cessation of training or recovery from illness. Convincing evidence of the role of PLA2 in ESEA awaits further serial study of its plasma concentration throughout a well defined period of stressful training, sufficiently long to establish development of the sub-peak plateau of ESEA observed in the present



Fig. 3 A proposed temporal relationship between model fatigue, ESEA and serum PLA2 activity modified from data of the present experiment and the only time course of induced serum PLA2 found in the literature (Koeniger et al. 1989), measured in multiple organ failure, accident patients. It may be seen that the model degradative function, fatigue, parallels the time course of succeedingly earlier degradative events (ESEA, PLA2) implicated (see text) in the etiology of muscle trauma accompanying a prolonged period of daily heavy exercise. This parallelism is especially evident in the decay of each curve to baseline from a peak or steady state level. (Reproduced with permission from Koeniger et al., Klin.Wochenschr., 67: 212-216, 1989).

study, followed then by non-training and ESEA recovery to baseline. Meanwhile there seems to be sufficient evidence to accept the modeled fatigue component as a quantitative mirror of the earlier time course of primary degradative processes induced by stressful training.

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APPENDIX

The model used is based on the stimulus-response behaviour of the body to exercise. Physical training at any time t provides a stimulus w(t) which produces a change in observed performance p(t) relative to a baseline level. As the training stimulus is short compared with the time course of adaptive response, it may be considered an impulse whose magnitude must be related to the intensity and duration of the session. A convenient measure of training for a single session is therefore given by the duration of the training interval in minutes multiplied by the ratio of exercise to maximum heart rate, both above resting value. This ratio ranges between a lower limit (arbitrarily set at 0) and an upper limit of 1 (for exercise at maximum heart rate) is defined as the delta heart rate ratio (delta HR Ratio). Heart rate measured during exercise may be denoted as HRex. While maximum heart rate (HRmax) may be measured directly or estimated as 220-age. An intensity weighting factor Y is used to give more credit to a short intense session which logically provides a greater training stimulus than a longer less strenuous session. This correction is based on the commonly observed exponential relationship between blood lactate concentration and training intensity (Green et al. 1983). Therefore,

Training stimulus = $\frac{\text{Duration}}{(D)} \times \frac{\text{HREX} - \text{HRrest}}{\text{HRmax} - \text{HRrest}} \times Y$ w(t) = D×delta HR Ratio×Y where: Y = exp (1.92x) (males) = exp (1.67x) (females) and x = delta HR Ratio

Each training impulse has two effects: (1) to increase fitness g(t) which adds to increase performance, and (2) to increase fatigue h(t) which subtracts from performance. Net performance p(t) at any time t, is given in arbitrary units as the difference between these two quantities. Thus:

 $p(t) = k1 \cdot g(t) - k2 \cdot h(t)$ (2)
where: k1 and k2 are arbitrary weighting factors for g(t) and h(t) respectively and: $g(t) = g(t-i) \exp(-i/\tau 1) + w(t)$

 $h(t) = h(t-i) \exp(-i/\tau 2) + w(t)$

where i is the interval (days) between each training impulse, and time t > 1. The four adjustable parameters in the model are the time constants for fitness $\tau 1$ and fatigue $\tau 2$, and fitness and fatigue weighting factors k1, k2 respectively. These constants may be determined by calibration of model predicted performances p (t) against real criterion performances Cp measured experimentally. Default values for model parameters for beginning parameter definition from modeling $\tau 1 = 45$ days, $\tau 2 = 15$ days, k1 = 1 and k2 = 2.0.

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(1)

(3)

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GLOSSARY OF TERMS:

Cp Criterion performance measured on a standard task first measured as a time and then converted to a points score; it is measured regularly to assess real performance response being effected by training, points

D - Duration of training session, min

- Dose Alternative expression for quantity of training w(t) absorbed in a single training session
- Fitness g(t) Hypothesized model component of performance ability termed fitness calculated from quantity of training undertaken, arbitrary units
- Fatigue h(t)- Hypothesized model component of performance ability termed fatigue calculated from quantity of training w(t) undertaken, arbitrary units
- delta HR Difference between two heart rates (one usually resting heart rate)
- delta HR ratio- Ratio of elevation of exercise to maximum heart rate, with both above resting value
- k1 Arbitrary weighting factor for fitness, dimensionless (initially 1)
- k2 Arbitrary weighting factor for fatigue, dimensionless (initially 2)
- p(t) Model-predicted performance determined from difference between weighted-model fitness kl·g(t) and weighted-model fatigue k2·h(t) at any time t during a training program, arbitrary units
- Response Term used in sense of pharmacokinetics but applied to training, expressing some measurable result or response arising from a known input of

training (dose) into a performance model

- Assessment of amount of training undertaken during a training session, also defined as a training impulse (trimp), or dose and calculated as the product of time (in min) spent training and HR ratio, arbitrary units
 - Weighting factor applied to calculation of w(t) to increase magnitude of quantity of training nonlinearly at higher training intensities, dimensionless

Y

 $\tau 1$

 $\tau 2$

- Time constant determining time course of decay in accumulated fitness g(t) between training sessions, days
 - Time constant determining time course of decay in accumulated fatigue h(t) between training session, days

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