

1401A INVITED
PRESENTATION

MODELING DOSE RESPONSE EFFECTS OF EXERCISE

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Introduction:

Considerable empirical training data is contradictory and not easily rationalized. Quantitative, serial, kinetic analysis of specific physical and biological responses to training and detraining in humans are sparse (Saltin *et al.*(1), Janssen *et al.*(2), Morton *et al.*(3)). This may be because the complex microscopic cellular activity which is induced by training and integrated to produce the gross structural and physio-chemical changes observed remains relatively inaccessible to investigation in human studies.

Systems Analysis

Despite this difficulty some insight into the above processes may be gained from the technique of systems analysis by making informal 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 states, 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 are made determines how well the systems model mirrors experimental reality and how well it rationalizes underlying physio-chemical mechanisms of action (Simon (4), Mader (5), Fukuba (6), Morton *et al.*(3)).

Systems Model of Training

The systems model employed here, Banister *et al.*(7), Calvert *et al.*(8), specifies two general components symbolizing induction/synthesis or repression/degradation processes. These are respectively named **Fitness** and **Fatigue**, and are estimated from the quantity (dose) of daily training $w(t)$. Dose of training is defined as a function of exercise duration and the exercise fractional elevation of maximum Heart Rate above rest. This may be expressed in arbitrary units of a product called **training impulse** $w(t)$ by the equation:

$$w(t) = \text{Duration of Training} \times \frac{HR_{EX} - HR_{REST}}{HR_{MAX} - HR_{REST}}$$

$$= D \times \Delta \text{ HR Ratio}$$

$w(t)$ may be weighted to emphasize strenuous training by introducing the multiplying factor Y given by:

$$Y = e^{bx}$$

Y reflects the exponential rise of blood lactate as the fractional elevation of exercise heart rate above rest ($\Delta \text{ HR Ratio}$) approaches unity where

$x = \Delta \text{ HR Ratio}$ and b is a constant, different for men (1.92) and women (1.67) respectively. Thus:

$$w(t) = D \times \Delta \text{ HR Ratio} \times Y$$

The **Training Impulse** $w(t)$ may be used to determine 2 components of performance termed **fitness** $g(t)$ and **fatigue** $h(t)$ at the end of the day t of training characterized by their decay time constants τ_1 , τ_2 in the interval i between successive bouts of training so that:

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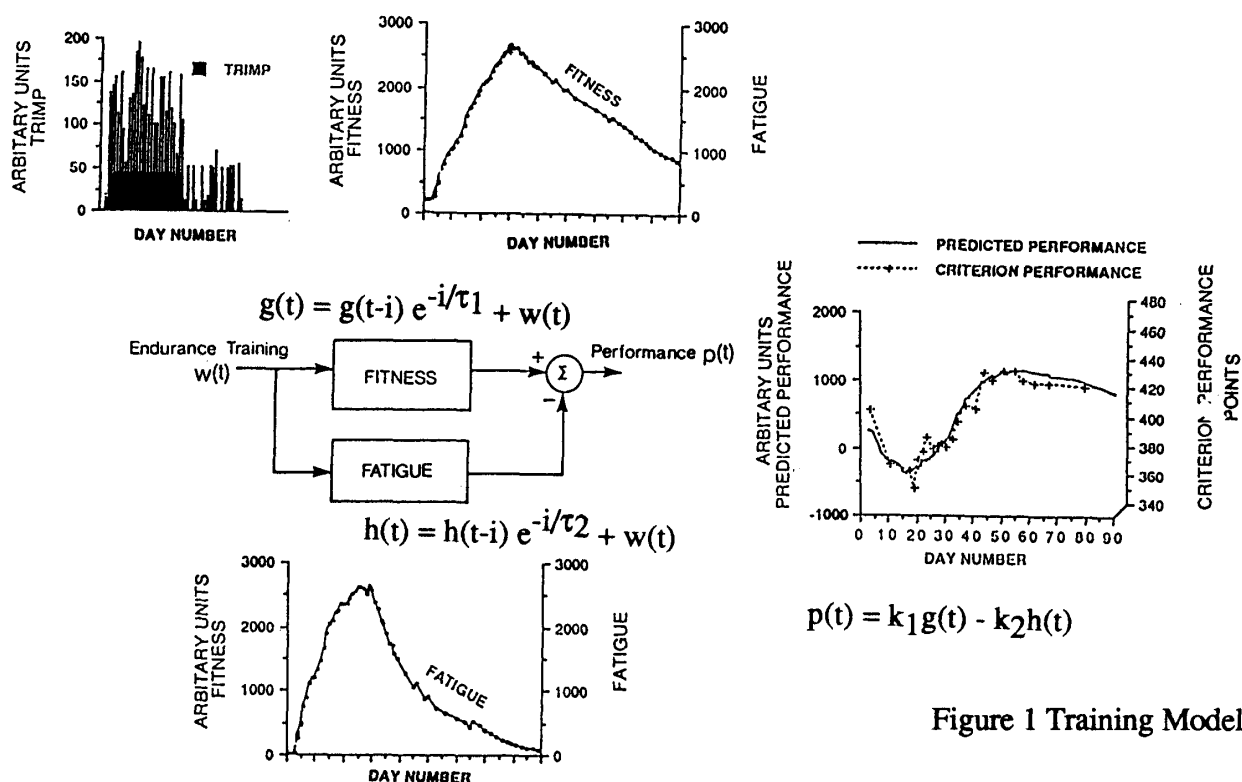


Figure 1 Training Model

the recursive equation:

$$p(t) = k_1 g(t) - k_2 h(t)$$

then defines a unique **performance** $p(t)$, predicted to result from the accumulation of **fitness** and **fatigue**, weighted respectively by the arbitrary constants k_1 and k_2 , under the influence of a daily **training impulse** $w(t)$. The balance between these components determines the quality of competitive performance first as **fatigue** predominates throughout heavy training when **performance** naturally may be expected to decline. This is followed by an increasingly better **performance** reaching a peak as relative rest from training, termed peaking or tapering, ensues.

Visual representation of the processes hypothesised in the model of Figure 1 are shown in Figures 2 and 3, Morton *et al.*(3).

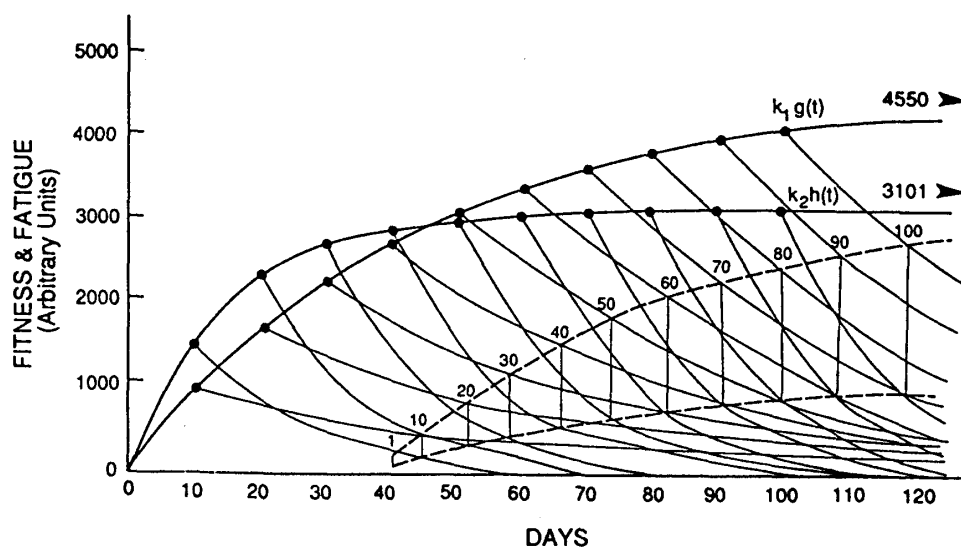


Figure 2.
Fitness and Fatigue
curves produced by
a standard amount
of 100 trimps
daily training.

Figure 2 illustrates the behavior of fitness and fatigue during and after uniform daily training of 100 trimps. Bold lines indicate exponential growth of fitness ($k_1 g(t)$) and fatigue ($k_2 h(t)$) under this training

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regimen with $\tau_1 = 45$, $\tau_2 = 15$, $k_1 = 1$, and $k_2 = 2$. If training is terminated at any time t , recovery takes place as functions decay (thin lines). Maximum positive differences between fitness and fatigue are shown by vertical bars. These maximum bars are located at days on which they actually occur after completing the number of training days (t) heading each bar. The lengths of each bar indicate the magnitude of each performance in arbitrary units.

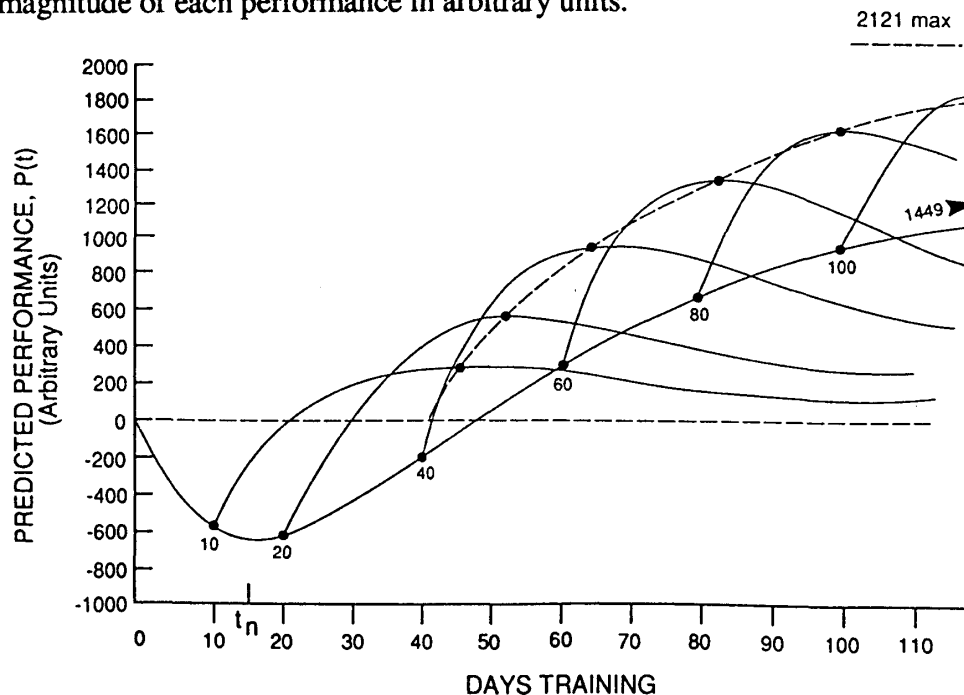


Fig.3
Predicted performance
throughout a period
of training.

Figure 3 illustrates the behavior of performance during and after uniform daily training of 100 trimps. The lower full curve represents performance that would result if training at current rate continued indefinitely. In this case, recovery to baseline performance takes ~47 days. For continuous daily training at 100 trimps, performance asymptotes toward a value of 1,449 as t becomes large. If instead training is terminated after an interval t_s , then performance increases described by a family of curves branching upward ($t = 10, 20, 40, 60, 80, 100$ days). The dashed line curve passes through loci of respective performance peaks at time t_m (bars of Fig.2), asymptoting toward the eventual value of 2,121 units that would occur 16 days after end of an extended period of continuous training. A better performance could only result from a new training regimen at a higher training impulse value per day. This process could theoretically proceed up to the limit of a person's genetic potential. Equation 1 may also be represented by an Influence Curve, Fitz-Clarke *et al.*,(9), which is a line showing the effect of a unit training impulse, $w(t)$ at any time point, t on performance at a specific future time $t(p)$. Thus for the equation:

$$\begin{aligned} p(t_p) &= k_1 g(t_p) - k_2 h(t_p) \\ &= \int_0^{t_p} [k_1 e^{-(t_p-t)/\tau_1} - k_2 e^{-(t_p-t)/\tau_2}] w(t) dt \\ &= \int_0^{t_p} L(\mu) w(t) dt \end{aligned}$$

and the influence curve is simply

$$L(\mu) = k_1 e^{-\mu/\tau_1} - k_2 e^{-\mu/\tau_2}$$

where $\mu = t_p - t$ is time measured previous to performance at t_p , when training has been under taken for a time t . The influence curve is then simply:

$$L(\mu) = k_1 e^{-\mu/\tau_1} - k_2 e^{-\mu/\tau_2}$$

and is defined by model parameters k_1 , k_2 , τ_1 , τ_2 specifically determined for an individual. Figure 4A shows the cumulative effect of several single training impulses on performance p at $t(p)$ both plotted from the method of Figure 2 and from the influence curve Fig 4B. Figure 5 demonstrates how peak performance at specified times $t_1 \dots t_p$ within a training period are affected by the placement of training relative to competition.

Figure 4a shows how performance $p(t)$ may be considered as summation of residuals of the contribution of each day's training impulse decayed to performance time as in this example of training for intervals t (equal to 0, 20, and 50 days) for performance $p(t)$ on day $t_p = 60$. Note that a training impulse of 200 units results in increments to fitness ($w(t) \times k_1$) and fatigue ($w(t) \times k_2$) of 400 and 200 units, respectively, on day $t = 0$. These values decay exponentially to $p(t_p)$ as shown. Each subsequent training impulse likewise adds a contribution according to its initial magnitude. The contribution of each training impulse to $p(60)$ is shown by the black area between curves and is negative when performance occurs before fatigue has decayed to 0. In figure 4b the same resultant may be calculated with more insight using a single influence curve, which shows relative contribution of each training impulse to performance at single specific future time. The right-hand origin of the influence curve (i.e., dimensionless ordinate of $L(\mu)$ extending from negative 1.0 when $k_2 = 2$ and $k_1 = 1$) is placed at point where optimal performance is desired and the relative contribution of each training impulse is immediately clear. Note the detrimental effect of the last training session (black area), as it is in the negative region of influence curve.

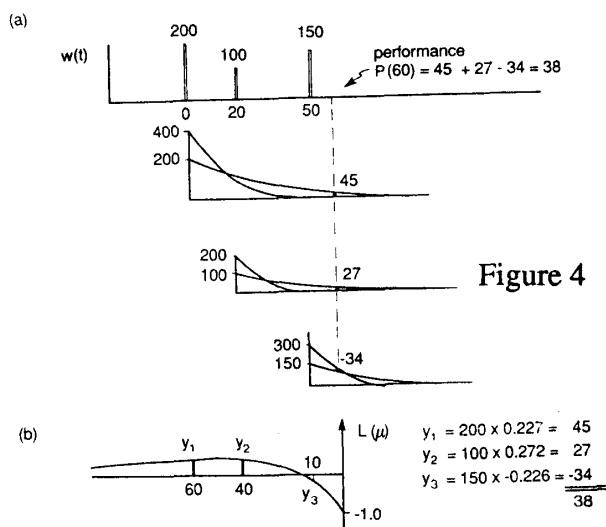


Figure 4

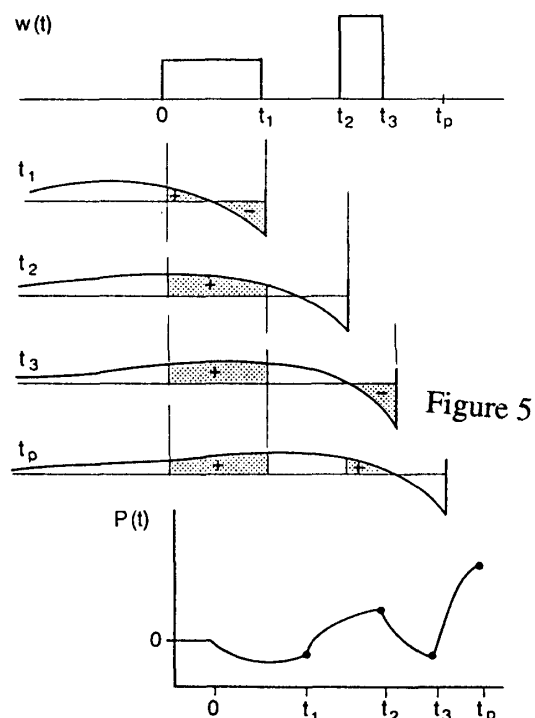


Figure 5

Figure 5. Indicates how the influence curve may be used as a moving template to track performance in time (t_1, t_2, t_3, t_p). Its origin is placed at point where performance is desired (successively from t_1 through t_p), and net (positive plus negative) hatched area of the influence curve shows the relative contribution of training $w(t)$ to performance $p(t)$. Note that performance at time t_p is highest because all training segments fall completely within positive region of influence curve for performance at time t_p .

The model of training effects presented here rationalizes seemingly contradictory evidence in the literature on the effects of training and detraining Houmarck et al., (10); Hickson et al., (11), Hickson et al (12), Costill (13), Kirwan (14).

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