

Systems Modelling of the Relationship Between Training and Performance

Tim Taha and Scott G. Thomas

Graduate Programme in Exercise Sciences, Faculty of Physical Education and Health, University of Toronto, Toronto, Ontario, Canada

Contents

Abstract	1061
1. Existing Models	1062
2. Quantification of Training	1065
3. Quantification of Performance	1066
4. Evaluation of Models and Model Components	1067
4.1 Descriptive Ability	1067
4.2 Quantification of Training Inputs	1069
4.3 Relationship to Underlying Structures	1069
4.4 Ability to Predict	1071
4.5 Applications	1071
5. Conclusion	1072

Abstract

Mathematical models may provide a method of describing and predicting the effect of training on performance. The current models attempt to describe the effects of single or multiple bouts of exercise on the performance of a specific task on a given day. These models suggest that any training session increases fitness and provokes a fatigue response. Various methods of quantifying the training stimulus (training impulse, absolute work, psychophysiological rating) and physical performance (criterion scale, arbitrary units) are employed in these models. The models are empirical descriptions and do not use current knowledge regarding the specificity of training adaptations. Tests of these models with published data indicate discrepancies between the predicted and measured time course of physiological adaptations, and between the predicted and measured performance responses to training. The relationship between these models and the underlying physiology requires clarification. New functional models that incorporate specificity of training and known physiology are required to enhance our ability to guide athletic training, rehabilitation and research.

Coaches and health professionals typically approach the relationship between training and performance empirically. Predictions of the effect of training on an individual's performance are guided

by on-going monitoring, and art and experience. Experimental studies to investigate the physiological adaptations underlying training responses have produced an extensive body of knowledge^[1] that is seldom systematically related to performance.^[2]

Researchers use models to develop and test hypotheses, and to identify important parameters and variables for measurement. For those interested in applications, mathematical models might provide a method of describing and predicting the effect of training on physical capability. While mathematical models are frequently used to describe the response to acute exercise,^[3] relatively few investigators have attempted to quantitatively relate chronic training stimuli to change in performance. The few proposed models have not achieved widespread use by researchers, coaches or health professionals. A review of current models may suggest approaches to improving their utility.

Dose-response models have been used to evaluate the relationship between regular exercise and health.^[4] However, those analyses assume that a constant 'dose' of exercise is maintained over the duration of the study period. They regard deviations from the prescribed training as imperfect compliance. The dose-response models do not examine how rapidly a desired response is achieved but at least obliquely address the relationship of the effect to underlying physiological responses.^[5] Compared with pharmacological studies that employ the dose response concept, the exercise studies only crudely quantify the input dose and make infrequent measures of the responses.

At the extremes, models may be purely empirical (models of data) or precisely based on underlying structure.^[6] However, most modelling is based on simplified abstractions of the underlying complex structures. The question arises of how much underlying structure should be incorporated into models of the relationship of training and performance.

The tension between structural and empirical approaches was evident in the first mathematical model of training and performance that started with cardiovascular, strength, skill and psychological components as possible determinants of perform-

ance.^[7] However, the skill, psychology and strength components were soon dropped due to difficulties in identifying useful measures and in relating the components. In subsequent studies, some authors have explored the relationship between their model terms and physiological measures,^[8] while others treat the models as purely empirical descriptions. Existing models do not use current knowledge regarding physiological adaptations to training. In sections 1, 2 and 3 of this review, the components of the present models are examined. In section 4, the common features are reviewed and the strengths and shortcomings of the models are discussed.

1. Existing Models

Until the work of Banister and Calvert^[7] and Calvert et al.,^[9] there were no published attempts at mathematically quantifying training and its relationship to performance. Banister, Calvert and colleagues encountered difficulty in combining components of their original proposed four-component model (cardiovascular, strength, skill and psychological).^[7] As a result, Calvert et al.^[9] used a simplified model to quantify the training and performance relationship of a swimmer. Their results were surprising and heartening, in that a clearer idea of the training process could be derived by fitting a mathematical equation and examining its parameters. The simple model was made up of three components. A single training impulse elicited two fitness responses that would increase performance, and a fatigue response that would decrease performance. The equation follows the general form (equation 1):

$$\begin{aligned} \text{Model performance} = & (\text{fitness from training model}) - \\ & K (\text{fatigue from training model}) \end{aligned} \quad (\text{Eq. 1})$$

where K is the constant that adjusts for the magnitude of the fatigue effect relative to the fitness effect. Mathematically, the model of Calvert and colleagues^[9] appeared as two simple first order differential equations that take the form (equation 2):

$$\frac{\partial p(t)}{\partial t} + \frac{1}{\tau} p(t) = w(t) \quad (\text{Eq. 2})$$

with an increase in the training impulse $[w(t)]$ resulting in a rise in performance $[p(t)]$ to a limit with a time constant in days (τ). Convolution of the associated transfer function results in (equation 3):

$$p(t) = w(t) * e^{-\frac{t}{\tau}} \quad (\text{Eq. 3})$$

where $*$ indicates the convolution and t is the day of the training impulse.

The final form of Calvert's model had two components that made up the fitness function and one fatigue function (equation 4):

$$p(t) = [(e^{-\frac{t}{\tau_1}} - e^{-\frac{t}{\tau_2}}) - K e^{-\frac{t}{\tau_3}}] * w(t) \quad (\text{Eq. 4})$$

where τ_1 and τ_2 are the time constants associated with the two fitness functions and τ_3 is the time constant associated with fatigue. The time constants and the fatigue coefficient (K) were determined for the individual through use of an iterative process that was repeated for each athlete. As is typical of training-performance models, the constants are specific to the individual. Individual specific constants may incorporate the effect of known training response determinants such as past activity, initial fitness and genetic predisposition.^[10,11]

The systems approach to modelling training and performance was not further expanded upon until the work of Morton et al. in 1990.^[12] They further simplified the two-component fitness and fatigue model used by Calvert et al.^[9] to a form that had only one fitness and one fatigue component since the additional component was not statistically supported.^[13] Therefore, on a given day, cumulated fitness $[g(t)]$ and fatigue $[h(t)]$ are affected by a training input $[w(t)]$ (equation 5 and equation 6).

$$g(t) = g(t-i)e^{-\frac{i}{\tau_1}} + w(t) \quad (\text{Eq. 5})$$

and

$$h(t) = h(t-i)e^{-\frac{i}{\tau_2}} + w(t) \quad (\text{Eq. 6})$$

where $g(t)$ and $h(t)$ are arbitrary fitness and fatigue response levels at the end of day t , i is the

period of time in days between the current training session and the previous session and τ_1 and τ_2 are the decay constants associated with the decay time constants of each input.^[12]

These two equations were then combined to form a simple linear difference equation that included weighting factors k_1 and k_2 for fitness and fatigue, respectively (equation 7).

$$p(t) = k_1 g(t) - k_2 h(t) \quad (\text{Eq. 7})$$

At about the same time, Busso et al.^[13] also began looking at systems modelling and used a variation on the Morton and colleagues^[12] model to quantify training and performance. The model put forth by Busso et al.,^[14] appeared in a very useful format as follows (equation 8):

$$p_n = p^* + k_1 \sum_{i=1}^{n-1} w_i e^{-\frac{(n-1)}{\tau_1}} - k_2 \sum_{i=1}^{n-1} w_i e^{-\frac{(n-1)}{\tau_2}} \quad (\text{Eq. 8})$$

where p_n is the estimation of performance on day n from successive training loads w_i with i varying from 1 to $n-1$. p^* represents the initial performance level of the participant.

An alternative mathematical approach to modelling is the use of influence curves as proposed by Fitz-Clarke et al. in 1991.^[15] An influence curve is a template indicating how a function over a certain range (in this case time) affects a response at a specific point.^[15] While the actual model that the curve is based on is no different than the conventional training and performance systems model, the influence curve gives a clear picture of how a specific training session affects performance on a given day. This was further expanded upon by Busso et al. in 1994 who calculated the positive and negative influences of training throughout the entire training period.^[16] The findings showed that the positive and negative influences were closer to the variations in actual performance than the values calculated by the positive and negative functions.

By using the coefficients τ_1 , τ_2 , k_1 and k_2 determined by Morton et al.,^[12] Fitz-Clarke et al.^[15] were able to derive the period of time prior to a given performance when training should be stopped or

reduced in order to maximise performance (t_n) [equation 9].

$$t_n = \frac{\tau_1 \tau_2}{\tau_1 - \tau_2} \ln \frac{k_2}{k_1} \quad (\text{Eq. 9})$$

where \ln is the natural logarithm.

Fitz-Clarke et al.^[15] utilised the model and data of Morton et al.^[12] and found that the optimal rest period following the last bout of exercise was 15.8 ± 6.5 days prior to the date of performance. In their study, they noted that the theoretical upper limit corresponded to tapers performed by elite swimmers (21 days),^[17] and moderately trained young men and women who trained for 10 weeks and subsequently reduced their training by 70% for a further 15 weeks.^[18]

An additional derived parameter was the time to a maximal performance following a single bout of training (t_g) [equation 10]:

$$t_g = \frac{\tau_1 \tau_2}{\tau_1 - \tau_2} \ln \frac{k_2}{k_1} \frac{\tau_1}{\tau_2} \quad (\text{Eq. 10})$$

Fitz-Clarke et al.^[15] suggested by use of t_n and t_g , that it may be possible to optimise training to achieve a maximal performance.

Busso et al. examined t_n in three studies^[19] and observed that it appeared to vary according to training load. Moderate training resulted in a smaller t_n than was observed in response to intensive training by either untrained individuals or elite athletes. Individuals who differed in age and training experience were utilised in the three studies. They included a young elite strength athlete (aged 20 years),^[16] an untrained young individual (aged 20 years)^[13] and an older runner (aged 42 years).^[12] Busso et al.^[19] felt that this was indicative of changing model parameters throughout the course of a training period. As a result, they proposed to use time-varying parameters in the model. In order to calculate the parameters a recursive least square method was employed.

In the recursive least square method, model parameters are estimated each time data are collected,^[19] which for these models is each time a performance

was measured. The parameters, therefore, were obtained by using the present and previous data.^[19] In order to increase the importance of present data, the past values are exponentially weighted to decrease their value. The recursive function (equation 11):

$$S_n = S_{n-1}\alpha + (\hat{p}_n - p_n)^2 \quad (\text{Eq. 11})$$

was used to fit model parameters on day n . Changing α (between 0 and 1) allows changes in the sensitivity of the recursion to the data. A small α may be vulnerable to noise while if α approaches 1, the ability to adjust for short-term changes in the data is limited. α was set by the authors to give a parameter standard error of close to 0.5, with the intent of eliminating changes in the parameters due to artifacts in the data. Naturally, the time-varying parameters resulted in a better fit of the data as it followed changes in performance more closely.

The time-varying parameters k_1 , k_2 and τ_2 were observed to be the most sensitive to changes in the intensity of exercise. This was not surprising in itself since Fitz-Clarke et al.'s^[15] sensitivity analysis of the effects of varying the model parameters indicated τ_1 did not influence t_n but varying k_1 , k_2 and τ_2 had a strong effect on altering the value of t_n . Using model parameters determined by Morton et al.,^[12] it was observed that a 10% change in the value of τ_1 resulted in only a $\pm 5\%$ change in t_g . A 10% change in one of τ_2 , k_1 and k_2 resulted in changes in t_n of 14–16%. Changes in t_g had a similar pattern of response to model parameter changes, except that the differences were not as large. Busso et al. concluded that as an individual exercises more intensely, it will take longer to recover (greater t_n).^[19]

A time-varying model has been used to determine how model parameters alter with different frequencies of training while intensity is held constant. While t_n increased significantly ($p < 0.01$) during the periods of higher frequency training, none of the individual model parameters (τ_1 , τ_2 , k_1 and k_2) changed significantly.^[20] The difference between amplitude coefficients ($k_2 - k_1$) increased, which suggests that there is a larger decrease in performance following frequent exercise.

2. Quantification of Training

In order to model training and performance, the training inputs must be quantified such that the parameters of intensity, frequency and duration are taken into account. Several methods of quantifying exercise training intensity using heart rate or lactate response, or expressing intensity relative to maximum heart rate or oxygen consumption, or relative to the ventilation or anaerobic threshold, have been used.^[21] However, only the subset of methods that have been employed in training-performance modelling will be reviewed.

A number of groups have categorised training into ordinal levels based on differences in intensity.^[9,22,23] These categories were either arbitrary divisions of intensity^[9] based on the individuals' reports, or the categories were based on a physiological measure such as swimming speed associated with a given lactate level^[22] or training heart rate.^[23] Each category is then assigned an arbitrary coefficient to weigh the values such that a training session with a higher intensity is given a greater weight. With these measures, training stimulus is a function of the physiological or psychophysiological response of the individual.

A second method to quantify training inputs was to use a unit of training termed the TRIMP (TRaining IMPulse).^[24] In order to calculate a single TRIMP, the training load (stress) is multiplied by the training intensity (strain). Therefore the TRIMP quantifies the training stimulus as a composite of external loading and physiological response.

For example, in an athlete who has a maximum heart rate of 185 beats/min and who runs 6km at an average heart rate of 130 beats/min, the TRIMP calculation would appear as follows (equation 12):

training volume (stress) = 6km; average heart rate = 130 beats/min; heart rate intensity (strain) = 70% of maximum heart rate

$$\begin{aligned}\text{TRIMP} &= \text{stress} \times \text{strain} / 10 \\ &= 6 \times 70 / 10 \\ &= 42\end{aligned}$$

(Eq. 12)

Therefore, that particular training session would contribute 42 TRIMPs. Later, TRIMPs were further modified to include a multiplying factor Y , which gave greater weight to high-intensity training by using the observed exponential rise in blood lactate associated with increased intensities of work.^[12]

Finally, the third method of quantifying training was to simply use the average fraction of the maximum aerobic power output sustained during the training session multiplied by the length of the training session.^[13,14,16,19,25] For example, an athlete may have a maximum oxygen consumption ($\dot{V}O_{2\max}$) of 4.0 L/min and train at 75% of $\dot{V}O_{2\max}$ for 30 minutes. This would yield the calculation (equation 13):

$$0.75 \times 4.0\text{L} / \text{min} \times 30 \text{ min} = 90\text{L of oxygen consumed}$$

(Eq. 13)

therefore, the work input would be 90L. With this method, the training stimulus is dependent only on the external loading. The concept of using average power is applied to strength as well as cardiovascular training. In strength training, a percentage of the individuals one repetition maximum for that exercise are utilised. In order to use the strength and cardiovascular training sessions together, a weighting factor was applied.^[14]

Two other methods quantified training based on using fractions of the maximal power generated during testing. A training session would correspond to a specific value for periods of intensive and lighter training when it is completed at the prescribed intensity.^[19] If the training was not conducted at the prescribed intensity, it was multiplied by the ratio between the actual percentage of the maximal power generated during the test and the assigned intensity. Busso et al.^[19] gave the following example. One workout assigned was 3 minutes of intensive exercise at 95% of maximal aerobic power determined during testing, followed by 3 minutes of active recovery. This sequence was repeated ten times, then 15 minutes rest followed by ten more sequences. If the intensive portions of the actual workouts were completed at 90% of maximal aerobic power, then the training dose of 100 would be multiplied by the ratio between actual (90%) and

prescribed intensities (95%). In this example, a training dose of 94.7 units would result.

In a later study, Busso et al.^[20] used a function of the exercise duration and intensity in reference to the average power generated during a 5-minute test ride. The average power generated during the 5-minute test ride is assigned a value of 100. Subsequent training sessions were divided into average power generated during 5-minute blocks and were multiplied by the ratio between the power output during the training and the testing.

Application of the models to such diverse training stimuli indicates that the boundary conditions are very broad. In addition, the different types of training quantification did not result in substantially different τ_1 and τ_2 . Changes, however, do occur in the amplitude coefficients which vary from 0.01–5.^[12,20] These differences also are due to differences in quantification of performance.

3. Quantification of Performance

The simplest way suggested to quantify performance converts the performance values into an arbitrary performance unit so that the initial value forms the baseline.^[13] Busso et al.^[13] converted the performance values to a common scale to avoid distorting comparisons between individuals. Mujika and colleagues^[22] used a similar method where all performances were converted into a percentage of personal best performances. However, these methods only work if the improvements in performance values are linear.^[12] The non-linearity of the relationship is evident for elite athletes where a small improvement in performance is the result of much greater training inputs than the larger relative improvement realised by a sedentary individual who has just begun to train. If the transformation is non-linear, a criterion point scale has been used.^[12]

Morton et al.^[12] suggested that the best way to model the criterion points scale was to look at the curve for athletic performance records over time. The mathematical form of the curve is (equation 14):

$$y = L + ae^{\frac{-x}{b}} \quad (\text{Eq. 14})$$

where y is the time or distance recorded for an athletic performance, L is the ultimate or perfect performance, a is an amplitude factor that is positive for running events and negative for jumping or throwing events, and b is a time parameter. It is the difference between the L and the performance of any able-bodied individual. This equation is rearranged to give (equation 15):

$$x = b \ln \left[\frac{a}{y - L} \right] \quad (\text{Eq. 15})$$

where x is the criterion points score and L is assigned a projected asymptotic value that theoretically cannot be bettered. If the present world record is assigned the value of 1000 points and the time or distance that any able-bodied individual can undertake is given a value of zero points then a and b can be solved for.

Morton et al.^[12] assumed for the 1500m run, a theoretical asymptote would be 3.1 minutes, the present world record (1990) was 3.5 minutes and the time that anyone could achieve was 15 minutes (arbitrarily assigned by Morton). These values are substituted into equation 15, which resulted in the criterion points (Cp) equation appearing as follows (equation 16):

$$Cp = 294.7 \ln \left[\frac{11.9}{y - 3.1} \right] \quad (\text{Eq. 16})$$

In 1996, Mujika et al.^[22] followed a group of swimmers for one competitive season and modelled performance utilising both the criterion points scale and an arbitrary unit system with each swimmer's personal best forming the baseline. The two methods produced time constants and fatigue and fitness magnitude coefficients that were statistically equivalent. Mujika et al.'s finding suggests that the sensitivity of the model to variation in the performance scaling is low.

Millet et al.^[26] examined triathletes where performances are difficult to compare since triathlon courses vary considerably both in terms of geogra-

phy and environmental conditions.^[26] As a result, performances were scaled subjectively from 0 (poor performance) and 10 (perfect performance) by both the coach and the athlete. Both ratings were averaged and expressed as a percentage. The validity of such a measure is doubtful, as it is difficult to determine which feelings or events during a competition make a good performance. For example, a victory over easy competition may give feelings of a good performance even if quantifiable measures such as time were slower. Until the subjective measures have been validated experimentally against an objective measure, performances that are not easily quantified should be avoided in future work.

4. Evaluation of Models and Model Components

There is a substantial literature on physiological modelling^[6] that examines the iterative process of developing and testing a model. The goals in experimental testing are either to modify the model to better match reality or to define the range of phenomena that can be accurately described and/or predicted by the model. In examining these training-performance models, their ability to describe phenomena, our ability to measure inputs and outputs, the relationship of model terms to the underlying structures, the models' ability to predict responses, and possible applications are discussed.

4.1 Descriptive Ability

In evaluating their model's descriptive ability, Calvert et al.^[9] chose a university intercollegiate swimmer and examined two 20- to 24-week seasons 2 years apart. After inputting all the actual performance points, a combination of iterative and interactive graphic approaches was used to estimate the individual time constants and coefficients that best related training to performance. While in general,

the predicted performances followed the profile of the actual performances, there were wide discrepancies at specific time points. The authors believe that the differences result not from oversimplification of the model or training inputs but from outside influences on the swimmer's life.^[9] They point out that fewer discrepancies were observed for the season that was modelled 2 years later and suggest this reflects the athlete's increased control of outside influences. Despite the differences in fit between the two seasons, the time constants and coefficients are remarkably similar (table I). This observation suggests the rate and magnitude of adaptation may be stable individual parameters. However, the similarity in parameters was not statistically tested.

Morton et al.^[12] used a similar method but instead of using a highly trained athlete, two of the authors were trained for 28 days and the data from their running programme and performances were modelled. Another important development was that Morton et al. obtained the model fit by using a least squares regression. An excellent agreement between measured and predicted performance ($r^2 = 0.96$) was obtained for one of the authors.

Busso and colleagues^[13] examined the goodness of fit of models with different numbers of components. Two components (fitness and fatigue) were found to have the same fit as a single-component model (fitness), but it was felt that the training programme was not of sufficient intensity to give much of a fatiguing effect.^[13]

The precision of the model fit is a concern especially when used to model changes in performance in elite athletes.^[22] Mujika et al.^[22] noted that their attempt to model performance of elite swimmers resulted in explained variations in actual performance of 45–85%. Explanations for this high variability in the ability to describe the data was attributed to imprecise quantification of training, and changes in the model parameters over a lengthy period of time.

Table I. Yearly changes in model parameters (reproduced from Calvert et al.,^[9] with permission)

Season	τ_1 Fitness-1	τ_2 Fitness-2	τ_3 Fatigue	K ₁ Fitness-1	K ₂ Fitness-2	K ₃ Fatigue
1970–1	50	5	15	1	1	2.0
1973–4	50	5	15	1	1	10.0

K = the constant that adjusts for the magnitude of the fatigue effect relative to the fitness effect; τ = time constant in days.

To minimise the effects of the changes in model parameters, Banister^[27] suggests that periods of 60–90 days be used to ensure a better fit.^[27]

In 1997, Busso et al.^[19] compared the fit of a time-varying model to the standard time invariant model. The time invariant model calculates one set of parameters to describe the relationship of training and performance over the whole study period. The time-varying model recalculates model parameters each time data are collected. A better fit was obtained for the time-varying model for the two individuals over the course of the training programme. The coefficients of determination for the two individuals for the time-varying model were 0.875 and 0.879, and for the time-invariant model were 0.682 and 0.666, respectively. The authors suggest variations in parameters with the time-varying model reflect changes in training responsiveness. However, this approach precludes using the model and its parameters to predict the responses to future training, unless the parameters themselves change in a predictable manner.

Careful examination of the follow-up study indicates that the derived parameters such as t_n and $k_2 - k_1$ do change with the frequency of training. As frequency or total volume of training increases (volume = frequency \times duration \times intensity), so does t_n from just over a day to about 3.5 days.^[20] $k_2 - k_1$ shows changes with a similar pattern. As derived parameters such as t_n and $k_2 - k_1$ change in lock-step with variation in training volume, this suggests that there is a time-invariant component to the relationship between performance and training. However, the structure of current time invariance models cannot explain this relationship.

On the other hand, caution must be used in interpretations of derived parameters such as t_n , t_g and $k_2 - k_1$. Each assumes that there is a positive and negative effect of training. It has yet to be established statistically if a fatigue or negative component should be included in the model. Only two studies have examined statistically whether the additional fatigue component significantly reduces the residual sum of squares. Busso et al.^[13] found that the fatigue component only improved model fit in one of eight

study participants. It was suggested that the low workloads undertaken by the study participants may have not induced measurable fatigue. In a study that modelled the transfers of sport-specific training effects on performance in elite triathletes, who were exercising about 20 hours per week, only the training-performance models of running were found to benefit significantly from the inclusion of a fatigue component.^[26] The residual sum of squares in a swimming training-performance model and the model of the effects of cycle training on running performance did not benefit significantly from the additional fatigue component.^[26]

Using a fitness-only model would mean that one is unable to derive the parameters t_n , t_g or $k_2 - k_1$. As a result, their relationship to changes in intensity or frequency of training should be interpreted carefully.

Millet et al.^[26] used the training and performance mathematical model in a very novel approach in an attempt to elucidate the transfer effects of cross-training. Elite triathletes were monitored over the course of a training year and the effects of swim, run or cycle training on swimming, running or cycling performances were examined. The only significant cross-training effect was observed between cycle training and running performance. Only running training had a significant effect on total triathlon performance as measured by a subjective rating scale (discussed in section 3). As a result, it was concluded that cycle training was an acceptable means to improve running performance while reducing total running distance. The added benefit would be a reduced wear and tear on the joints due to the pounding of running. Alternatively, the relationship between cycling and running may have been a consequence of the periodisation of the training programme.^[28,29] It is unlikely that the coach would prescribe training that varies in intensity between modalities especially during the competitive phase. In other words, cycling volume would increase at the same time as running volume. As a result, the mathematical model would indicate a relationship between the training modalities.

4.2 Quantification of Training Inputs

The TRIMP concept, as originally put forth by Banister et al.,^[24] does not take into account the energy system-specific effects of training intensity. For example, a run of 3km at 50% of maximum heart rate produces the same TRIMPs (15 TRIMPs), and therefore the same fitness and fatigue effects on performance as a run of 100% intensity for 1.5km. Yet the subjective experience and experimental studies^[30] have demonstrated energy system-specific effects.

Morton et al.^[12] attempted to address this shortcoming by including a weighting factor in the equation that is based on the exponential rise in blood lactate as work load increases. Heart rate was used as an indicator of work rate. However, this still results in unsatisfactory results because the relationship between lactate and heart rate is not fixed.^[31] An endurance-trained athlete maintains a linear relationship between relative intensity and heart rate but there is a flattening of the curve between lactate and relative intensity (lower peak lactates) as training progresses.^[32] Also, the rate at which heart rate and lactate adapt in response to chronic exercise differ. Rapid changes in lactate level at a given workload can occur without a change in heart rate.^[31] These factors decrease the precision of using the lactate curve as a weighting factor.

Quantifying training with the ratio of training session power output to maximum power output multiplied by training session duration again produces training dose estimates that are equivalent for long sessions at a low relative power output and short sessions at a high relative power output. As a result, in contrast to the observed specific effects of low- versus high-intensity training, the modelled fitness and fatigue effects would be the same. Also, the effect of inter-individual differences in fitness on the response to a training input is not quantified and the approach is difficult to implement away from laboratory ergometers.

Using categories to define training intensities allows a simple quantification of the training without having to measure velocities for each individual training session.^[22,23] The difficulty, again, lies with

the fact that a long, slow training session is predicted to have the same fitness and fatigue effects as a short, high-intensity training session.

The present models implicitly assume that the performance activity matches the training activity and therefore do not consider the specificity of training. As a result, these models do not reflect real world training, in which there is a wide variety of training sessions. For example, in rowing, competitions typically last from 330–420 seconds but training sessions vary between maximum efforts for 1 minute and submaximal 90 minute workouts. The present models will predict the same results regardless of match between type of training and measured performance. Furthermore, the model-derived fitness and fatigue coefficients would not correspond to the individual's actual ability to adapt to training. By reducing the training inputs into one number, much vital information regarding the type of training is lost.

4.3 Relationship to Underlying Structures

The proposed models^[9,12-14] are empirical systems models; meaning that a relationship to underlying structures is not explicitly assumed. However, as indicated in Morton's review of training-performance models,^[33] several attempts have been made to relate the fitness and fatigue components of the model to physiological responses.

In Busso's 1990 study,^[14] it was proposed that there were significant relationships between calculated fitness and fatigue and certain hormonal levels. The performance (weight lifted in the clean and jerk) and hormonal responses of six elite weightlifters were monitored over the course of 1 year. Four of the six study participants had decreases in the fitness component over the year of training and actual performance did not change. The authors observed correlation coefficients of 0.5–0.97 between modelled and actual performance. They observed similar time constants between fitness (23 days) and fatigue (22 days) parameters. The authors observed generally high, positive correlations between modelled fitness and serum testosterone ($r = 0.61$ – 0.92) but this relationship was significant for

Table II. Model parameters from published training-performance models

Study	Study participants (age)	Time constants (days)			Amplitude constants (arbitrary units)		
		τ_1 fitness-1	τ_2 fitness-2	τ_3 fatigue	K_1 fitness-1	K_2 fitness-2	K_3 fatigue
Calvert et al. ^[9]	Male swimmer (college age)	50	5	15	1	1	2.0
Morton et al. ^[12]	Male (57y)	50	NA	11	1	NA	1.8
	Male (42y)	40		11	1		2.0
Busso et al. ^[13] (2 component)	8 fit males (19–22y)	38	NA	1.8	0.048	NA	–0.117
Busso et al. ^[19] (time invariant)	Male (36y)	60	NA	4	0.0021	NA	0.0078
	Male (29y)	60		6	0.0019		0.0073

K = the constant that adjusts for the magnitude of the fatigue effect relative to the fitness effect; **NA** = not used in model; τ = time constant in days.

only two individuals. Surprisingly, the modelled fatigue and serum testosterone was also positively correlated ($r = 0.34\text{--}0.88$; one significant coefficient). It is difficult to suggest why the same physiological variable may be positively related to both fitness and fatigue.

In terms of physiological parameters, the fatigue consequent to training session has not been quantified. The physiological basis of exercise-induced decrements in force production have been extensively reviewed.^[34,35] Prolonged fatigue following high-intensity exercise has been documented in response to exercise.^[36] However, identifying relationships between modelled fatigue and physiological variables has seldom been ventured. Banister and colleagues^[37] observed that the fatigue component and serum levels of certain enzymes (creatine kinase, lactate dehydrogenase and aspartate aminotransferase) followed similar, although phase shifted, curves. However, these observations in two individuals were not statistically tested nor have subsequent studies supported these observations. As indicated in the paragraph above, Busso et al.^[14] observed a correlation between testosterone and modelled fatigue. In addition, they observed a significant correlation between the fatigue and the testosterone to cortisol ratio in one of six study participants.

James and Doust^[38] subjected runners to a single session of severe interval training and observed a performance decrement (24%) 1 hour, but not 72

hours later. This would suggest that the combination of fatigue and fitness response is negative for less than 72 hours. Also, performance has been seen to increase within 10 days of moderate-intensity, long-duration exercise.^[39]

The model-based estimated time course of change in fitness and performance (table II) differs from experimental observations with various groups of study participants.^[39–41] The current models, with slow development of fitness and rapid development of fatigue (table II), predict impaired performances in response to short-term training.

The model of Morton et al.^[12] quantifies training with TRIMPs and sets out default time course and magnitude values for the fatigue and fitness coefficients. If the data of Green et al.^[39] are examined using Morton's model, the daily training load is 366.5 TRIMPs. Utilising this value in Morton's equation, it predicts that 10 days of training would cause severe fatigue and a reduction in performance from baseline. The model predicts performance would not increase until 48 days after beginning training, which contrasts with the 4.3% increase observed by Green et al.^[39] at the 10-day mark.

The training intensity used by Green et al.^[39] (59% of $\dot{V}O_{2\max}$) may not be effectively represented utilising TRIMPs. If this is the case, it suggests an important limitation on the range of application of the model. Alternatively, the fitness and fatigue coefficients may be completely different between the study participants used by Green et al.^[39] and the

default values obtained by Morton et al.^[12] However, the consistency of the time coefficients across modelling studies (table II) argues against that possibility.

When physiological parameters such as resting heart rate or blood volume are examined, no fatigue or negative stage is observed within the first few days of beginning an exercise programme.^[42,43] In fact, it was shown that plasma volume increased 20% and total blood volume increased by 12% with 3 days of light-load cycling.^[42] Resting heart rate also did not show a fatiguing or negative effect with training, decreasing within 8 days.^[43] While these physiological parameters do not necessarily reflect athletic performance, the models presented are unable to describe the changes in the parameters that are believed to underlie fitness.

4.4 Ability to Predict

The original article by Banister and colleagues^[7] expressed the hope that the model might predict the effects of training. We used the model suggested by Busso et al.^[16] to model the published data of Hickson and colleagues.^[44] In Hickson et al.^[44] nine individuals were exercised for 40 minutes per day for 6 days per week. After 4 weeks, the training loads were increased. We observed that over the first 4 weeks of training, change in performance is adequately described by the model with a fitted set of coefficients (error = $\pm 1.5\%$). However, applying the same coefficients to the second period results in significant error ($\pm 6\%$). These observations place some doubt on the present model's ability to predict future performances. The time-varying mode might be employed to better describe the training-performance relationship in the second period. However, to retain the possibility of predicting future performance, new models will have to be formulated.

Examination of the time constants from one year to the next in the only study which actually looks at the multiple seasons,^[9] shows a remarkable consistency (table I). This suggests that in a more highly trained individual, these parameters are stable and, as a result, do have some predictive ability.

4.5 Applications

The model has been used by Mujika et al.^[22] to examine the changes in performance during training and tapering periods in elite swimmers. It was noted that the enhancement of performance following the taper period was due to a decrease in the negative influence of training rather than an increase in positive influences. Positive influences rose rapidly, following the commencement of that modelled year's training to a level that was relatively steady. Negative influences increased during periods of heavy training but disappeared during periods where training was reduced or tapered in preparation for competition. The reduction of training during a tapering period caused a greater fall in the negative influences than in positive influences. Mathematically, this would be due to the slower decay (larger time constant) associated with the fitness component than with the fatigue component.

Banister et al.^[45] used a training model to suggest which type of taper is most effective and confirmed these results in a field test of the taper types using triathletes. However, the predictive nature of the model was not tested statistically as the study modelled training loads and performances after the athletes completed them. Essentially, the results obtained from the athletes confirmed the ability of the model to describe the data obtained in the field study. The use of the model in such a way does represent a substantial improvement to the structuring of future studies in tapering as it allows different methods to be tested theoretically prior to the beginning of a lengthy study. Additionally, Banister et al.^[45] confirm the findings of prior studies that a higher intensity, low volume (frequency \times duration) taper will give the best results.^[46,47] The useful additional information to some from this study is that a taper should reduce volume sharply at its beginning to eliminate fatigue effects as quickly as possible.^[45]

Morton^[33] argues that if the model parameters to describe an individual athlete's response are known, then a training programme may be devised that will maximise performance while avoiding overtraining. While experimental verification remains to be gathered, this would be a substantial contribution.

5. Conclusion

From the original model proposed by Calvert et al.,^[9] new information was gathered regarding the dose/response effect of exercise. The most interesting hypothesis was the concept of fatigue; a negative influence on performance brought on by a training session.

The current models, in particular the time-varying model of Busso et al.,^[19] are very successful in describing the relationship between training and performance in their studies. However, the discordance with known physiology and the absence of training response predictions suggest that another approach should be developed. One possibility is to move to a functional model^[3] that would incorporate information about underlying structure.

Models have to be tested to see whether they can predict future performances. A predictive model would achieve a much greater utility for athletics, rehabilitation and research. In physiological terms, a predictive training-performance model that incorporated knowledge about physiological adaptations would allow hypothesis-driven research. The end result would be the ability to detect unusual physiological responses to training as well as the ability to see which physiological responses to training have the greatest impact on performance.

Acknowledgements

This work was supported through a research contract with the Canadian Forces. The authors have no conflicts of interest that are directly relevant to the content of this manuscript.

References

- Keul J, König D, Huonker M, et al. Adaptation to training and performance in elite athletes. *Res Q Exerc Sport* 1996; 67 (3): S29-36
- Coyle EF. Integration of the physiological factors determining endurance performance ability. *Exerc Sport Sci Rev* 1995; 23: 25-63
- Swanson GD. Assembling control models from pulmonary gas exchange dynamics. *Med Sci Sports Exerc* 1990; 22 (1): 80-7
- Bouchard C, Rankinen T. Individual differences in response to regular physical activity. *Med Sci Sports Exerc* 2001; 33 (6 Suppl.): S446-51
- Haskell WL. What to look for in assessing responsiveness to exercise in a health context. *Med Sci Sports Exerc* 2001; 33 (6 Suppl.): S454-8
- Keener J, Sneyd J. *Mathematical physiology*. New York: Springer, 1998
- Banister EW, Calvert TW. A systems model of training for athletic performance. *Aust J Sports Med* 1975; 7: 57-61
- Banister EW, Hamilton CL. Variations in iron status with fatigue modelled from training in female distance runners. *Eur J Appl Physiol* 1985; 54 (1): 16-23
- Calvert TW, Banister EW, Savage MV, et al. A systems model of the effects of training on physical performance. *IEEE Trans Syst Man Cybern* 1976; 6 (2): 94-102
- Sonna LA, Sharp MA, Knapik JJ, et al. Angiotensin-converting enzyme genotype and physical performance during US Army basic training. *J Appl Physiol* 2001; 91: 1355-63
- Thomas SG, Cunningham DA, Rechnitzer PA, et al. Determinants of the training response in elderly men. *Med Sci Sports Exerc* 1985; 17: 667-72
- Morton RH, Fitz-Clarke JR, Banister EW. Modeling human performance in running. *J Appl Physiol* 1990; 69 (3): 1171-7
- Busso T, Carasso C, Lacour JR. Adequacy of a systems structure in the modeling of training effects on performance. *J Appl Physiol* 1991; 71 (5): 2044-9
- Busso T, Hakkinen K, Pakarinen A, et al. A systems model of training responses and its relationship to hormonal responses in elite weight-lifters. *Eur J Appl Physiol* 1990; 61 (1-2): 48-54
- Fitz-Clarke JR, Morton RH, Banister EW. Optimizing athletic performance by influence curves. *J Appl Physiol* 1991; 71 (3): 1151-8
- Busso T, Candau R, Lacour JR. Fatigue and fitness modelled from the effects of training on performance. *Eur J Appl Physiol* 1994; 69 (1): 50-4
- Neufer PD, Costill DL, Fielding RA, et al. Effect of reduced training on muscular strength and endurance in competitive swimmers. *Med Sci Sports Exerc* 1987; 19 (5): 486-90
- Houmard JA, Costill DL, Mitchell JB, et al. Reduced training maintains performance in distance runners. *Int J Sports Med* 1990; 11 (1): 46-52
- Busso T, Denis C, Bonnefoy R, et al. Modeling of adaptations to physical training by using a recursive least squares algorithm. *J Appl Physiol* 1997; 82 (5): 1685-93
- Busso T, Benoit H, Bonnefoy R, et al. Effects of training frequency on the dynamics of performance response to a single training bout. *J Appl Physiol* 2002; 92 (2): 572-80
- Martin D, Carl K, Lehnertz K. *Handbuch Trainingslehre*. Schorndorf: Verlag Hofmann Schorndorf, 1993
- Mujika I, Busso T, Lacoste L, et al. Modeled responses to training and taper in competitive swimmers. *Med Sci Sports Exerc* 1996; 28 (2): 251-8
- Rowbottom DG, Keast D, Garcia-Webb P, et al. Training adaptation and biological changes among well-trained male triathletes. *Med Sci Sports Exerc* 1997; 29 (9): 1233-9
- Banister EW, Calvert TW. Planning for future performance: implications for long term training. *Can J Appl Sport Sci* 1980; 5 (3): 170-6
- Busso T, Hakkinen K, Pakarinen A, et al. Hormonal adaptations and modelled responses in elite weightlifters during 6 weeks of training. *Eur J Appl Physiol* 1992; 64 (4): 381-6
- Millet GP, Candau RB, Barbier B, et al. Modelling the transfers of training effects on performance in elite triathletes. *Int J Sports Med* 2002; 23 (1): 55-63
- Banister EW. Modeling elite athletic performance. In: MacDougall JD, Wenger HA, Green HJ, Canadian Association of Sports Sciences, editors. *Physiological testing of the high-*

- performance athlete. 2nd ed. Champaign (IL): Human Kinetics Books, 1991: 403-24
28. Matveev LP. Fundamentals of sports training. Moscow: Progress Publishers, 1981
 29. Fry RW, Morton AR, Keast D. Periodisation of training stress: a review. *Can J Sport Sci* 1992; 17 (3): 234-40
 30. McArdle WD, Katch FI, Katch VL. Exercise physiology: energy, nutrition and human performance. Philadelphia (PA): Lea and Febiger, 1991
 31. Gaesser GA, Poole DC. Blood lactate during exercise: time course of training adaptation in humans. *Int J Sports Med* 1988; 9 (4): 284-8
 32. Hurley BF, Hagberg JM, Allen WK, et al. Effect of training on blood lactate levels during submaximal exercise. *J Appl Physiol* 1984; 56 (5): 1260-4
 33. Morton RH. Modeling training and overtraining. *J Sports Sci* 1997; 15 (3): 335-40
 34. MacIntosh BR, Rassier DE. What is fatigue? *Can J Appl Physiol* 2002; 27 (1): 42-55
 35. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 2001; 81 (4): 1725-89
 36. Raastad T, Hallen J. Recovery of skeletal muscle contractility after high- and moderate-intensity strength exercise. *Eur J Appl Physiol* 2000; 82 (3): 206-14
 37. Banister EW, Morton RH, Fitz-Clarke J. Dose/response effects of exercise modeled from training: physical and biochemical measures. *Ann Physiol Anthropol* 1992; 11 (3): 345-56
 38. James DVB, Doust JH. Time to exhaustion during severe intensity running: response following a single bout of interval training. *Eur J Appl Physiol* 2000; 81 (4): 337-45
 39. Green HJ, Coates G, Sutton JR, et al. Early adaptations in gas exchange, cardiac function and haematology to prolonged exercise training in man. *Eur J Appl Physiol* 1991; 63 (1): 17-23
 40. Govindasamy D, Paterson DH, Poulin MJ, et al. Cardiorespiratory adaptation with short term training in older men. *Eur J Appl Physiol* 1992; 65 (3): 203-8
 41. Hickson RC, Bomze HA, Holloszy JO. Linear increase in aerobic power induced by a strenuous program of endurance exercise. *J Appl Physiol* 1977; 42 (3): 372-6
 42. Green HJ, Jones LL, Painter DC. Effects of short-term training on cardiac function during prolonged exercise. *Med Sci Sports Exerc* 1990; 22 (4): 488-93
 43. Swaine IL, Linden RJ, Mary DA. Loss of exercise training-induced bradycardia with continued improvement in fitness. *J Sports Sci* 1994; 12 (5): 477-81
 44. Hickson RC, Hagberg JM, Ehsani AA, et al. Time course of the adaptive responses of aerobic power and heart rate to training. *Med Sci Sports Exerc* 1981; 13 (1): 17-20
 45. Banister EW, Carter JB, Zarkadas PC. Training theory and taper: validation in triathlon athletes. *Eur J Appl Physiol* 1999; 79 (2): 182-91
 46. Hooper SL, Mackinnon LT, Ginn EM. Effects of three tapering techniques on the performance, forces and psychometric measures of competitive swimmers. *Eur J Appl Physiol Occup Physiol* 1998; 78 (3): 258-63
 47. Shepley B, MacDougall JD, Cipriano N, et al. Physiological effects of tapering in highly trained athletes. *J Appl Physiol* 1992; 72 (2): 706-11
-

Correspondence and offprints: *Scott G. Thomas*, Faculty of Physical Education and Health, 55 Harbord St, Toronto, M5T 2W6, Canada.

E-mail: scott.thomas@utoronto.ca