# MODELING THE RESIDUAL EFFECTS AND THRESHOLD SATURATION OF TRAINING: A CASE STUDY OF Olympic Swimmers

# Philippe Hellard,<sup>1</sup> Marta Avalos,<sup>2</sup> Gregoire Millet,<sup>3</sup> Lucien Lacoste,<sup>1</sup> Frederic Barale,<sup>1</sup> and Jean-Claude Chatard<sup>4</sup>

<sup>1</sup>Research Department of the French Swimming Federation, France; <sup>2</sup>HeuDiaSyC Laboratory, UMR CNRS 6599, Compiègne University of Technology, France; <sup>3</sup>Faculty of Sports Sciences, Montpellier, France; <sup>4</sup>Laboratory of Physiology GIP Exercise, Saint-Etienne University School of Medicine, France.

ABSTRACT. Hellard, P., M. Avalos, G. Millet, L. Lacoste, and J.C. Chatard. Modeling the residual effects and threshold saturation of training: A case study of Olympic swimmers. J. Strength Cond. Res. 19(1):67-75. 2005.—The aim of this study was to model the residual effects of training on the swimming performance and to compare a model that includes threshold saturation (MM) with the Banister model (BM). Seven Olympic swimmers were studied over a period of  $4 \pm 2$  years. For 3 training loads (low-intensity  $w^{LIT}$ , high-intensity  $w^{HIT}$ , and strength training  $w^{ST}$ ), 3 residual training effects were determined: short-term (STE) during the taper phase (i.e., 3 weeks before the performance [weeks 0, 1, and  $\hat{2}$ ]), intermediate-term (*ITE*) during the intensity phase (weeks 3, 4, and 5), and long-term (LTE) during the volume phase (weeks 6, 7, and 8). ITE and LTE were positive for  $w^{HIT}$ and  $w^{LIT}$ , respectively (p < 0.05). Low-intensity training load during taper was related to performances by a parabolic relationship (p < 0.05). Different quality measures indicated that MM compares favorably with BM. Identifying individual training thresholds may help individualize the distribution of training loads.

KEY WORDS. mathematical model, performance, swimming

# INTRODUCTION

he training-performance relationship is an important issue for elite sports coaches in search of reproducible indicators useful for organizing the athlete's training program. Many authors have studied the relative influence of training (23, 27) and have found that reactions to training depend on volume, intensity, and frequency of the training sessions. Others have reported divergent results (4, 9), perhaps related to the fact that residual effects were not taken into account (3, 5, 6, 9). These residual effects are defined both in terms of the retention of physical changes following a summation of many training sessions (delayed effects) and in terms of the results of a summation of many training sessions (accumulative effects) (9).

The model described by Banister (4, 5) and its variations (6, 7, 8) have commonly been used to describe the dynamics of training. This model is based on 2 antagonistic functions, both calculated from the training impulse (4, 5). Studies on cellular adaptability reactions to exercise (4) have demonstrated that the negative function can be taken to be the complete set of fatigue reactions caused by training. The positive function can be compared to a fitness gain resulting from the organism's adaptation to training (4, 5, 8, 22). Expressed as an exponent, the functions account for the decreasing impact of the training effect. When iterative training sessions are considered, the time course of performance is described by:

$$p_t = p_0 + k_a \sum_{s=0}^{t-1} e^{-(t-s)/\tau_a} w_s - k_f \sum_{s=0}^{t-1} e^{-(t-s)/\tau_f} w_s$$

where  $p_t$  is the known performance at week (or day) t;  $w_s$  is the known training load per week (or day) from the first week of training to the week (or day) preceding performance  $p_i$ ;  $k_a$  and  $k_f$  are the fitness and fatigue multiplying factors, respectively;  $\tau_a$  and  $\tau_f$  are the fitness and fatigue decay time constants, respectively; and  $p_0$  corresponds to an initial, basic level of performance.

In the initial Banister model (BM), the training load was quantified as the product of training quantity (distance or duration)  $\times$  training intensity, measured by heart rate (5), oxygen consumption (7), or lactate concentration (22). However, because swimmers train with a wide range of different exercises (low intensity, high intensity, strength training), the immediate and long-term training effects cannot be grouped in a single regimen. A new approach, taking into account the residual effects of the various types of training loads, would be preferable (4, 22).

In the classical linear periodized model, a distinction has always been made between volume training, which is designed to develop aerobic capacities, and intensity training, which is designed to develop qualities specifically linked to performance, such as anaerobic capacities related to efficient technique (2, 3, 9, 13, 14, 23). For example, it is recommended to engage in volume training in the early part of the season (2, 9, 13, 14, 27), and to increase high intensity specific training as the season progresses and during taper phases (5, 15, 22). This model was suggested in order to prevent overtraining and to achieve peak physical performance for major competitions (2, 9, 11, 13). Such a schedule is also based on the assumption that the different physiological systems vary in the retention and rate of loss or gain of training level (9, 13, 14, 24). Nevertheless, the impacts of the various types of training loads on performance have an upper limit above which training does not elicit further adaptation of the subjects (12, 19, 21). Mader (19) described the balance between protein synthesis and degradation as a function of protein degradation rate by a transcription-translation activation control loop. Steadystate protein balance and active adaptation vary according to the level of functional activity induced by the training load. If the training stimulus is too intense, protein degradation exceeds synthesis, leading to catabolic processes, excessive and damaging immune system response, chronic tissue disruption, and subsequent muscular atrophy and degradation of physical capacities (5, 12, 19). Other observations have emphasized the importance of maintaining the intensity and duration of the training stimulus below a threshold limit in order to obtain an optimal development of physical capacities (6, 20, 21). It is noteworthy that Busso (6) suggested recently that the relationship between daily amounts of training and performance may be stronger if defined by a parabolic relationship. Such a relationship would mean that when the amount of training exceeds the optimal level, performance could decline because of the fatigue induced by over-solicitation (6, 19).

The 2 hypotheses tested in the present study were: (a) volume training has a long-term positive effect on performance, whereas intensity training has a short-term positive effect; and (b) the impact of training on performance is nonlinear and has an upper limit (for BM this impact is linear:  $k_a w_s$  and  $k_f w_s$ ).

# **Methods**

### **Experimental Approach to the Problem**

To investigate the hypothesis of this study, a modeling post facto longitudinal research design was applied. Indeed, this study was the first to quantify training loads during an Olympic cycle in finalist and medalist Olympic swimmers. When preparing for events such as the Olympic Games, these high-performance athletes require training programs that have been personalized for intensity, frequency, and duration of taper (3, 23). The design of studies may be problematic; the athletes have different intensity responses, so the variables, including amount of exercise per training period, training format, taper pattern, and rest periods, have to be individually tailored. Therefore, when experimental design is difficult, the modeling approach provides an attractive solution (3). In the first part of the study, in order to determine the residual effect of training, a multiple regression analysis was computed between performance (dependent variable) and the training variables (independent variables) for 3 training phases: short-term (STE; 3 weeks before the performance [weeks 0, 1, and 2]), intermediate-term (ITE; weeks 3, 4, and 5 before the performance), and long-term (*LTE*; weeks 6, 7, and 8). In the second part of the study, a modified model (MM) was tested that included a saturation threshold above which training did not elicit subjects' adaptations to BM.

# **Studied Population**

The training characteristics and performances of 7 elite swimmers (4 women and 3 men) were analyzed over a period of  $4 \pm 2$  years (mean  $\pm SD$ ). Mean age at the beginning of the study was  $19.3 \pm 2.3$  years, mean body weight  $60 \pm 3$  kg, and mean height  $169 \pm 3$  cm for the women, and  $20.2 \pm 2.9$  years,  $74 \pm 4$  kg,  $185 \pm 4$  cm for the men. The height and the weight of the swimmers remained stable throughout the entire duration of the study, signifying the absence of the pubertal maturing process. Subject #1 was an Olympic medal winner, subjects #2, #3, #6, and #7 were Olympic finalists, and subjects #4 and #5 were European Junior level swimmers. The study was reviewed and approved by the local University Committee on Human Research and written informed consent was obtained from each participant. Each swimmer trained according to the program prescribed by their coaches, and the characteristics of the training regimens or competition schedules were not modified by the present study.

#### **Training Stimulus**

Intensity levels for swim workouts were determined as proposed by Mujika et al. (22). An incremental test to exhaustion was performed at the beginning of each season to determine the relationship between blood lactate concentration and swimming speed. Each subject swam 6  $\times$  200 m at progressively higher percentages of their best personal competition time over this distance, until exhaustion. Blood lactate concentration was measured in blood samples collected from the fingertip during 1-minute recovery periods separating the 200-m swims. All swimming sessions were divided into 5 intensity levels according to the individual results obtained during this test. Intensities I1, I2, and I3 represented swimming speeds below ( $\approx 2 \text{ mmol} \cdot L^{-1}$ ), at ( $\approx 4 \text{ mmol} \cdot L^{-1}$ ), and just above ( $\approx 6 \text{ mmol} \cdot L^{-1}$ ) the onset of blood lactate accumulation, respectively. High swimming work producing blood lactate concentrations of  ${\approx}10~mmol{\cdot}L^{{\scriptscriptstyle -1}}$  was defined as intensity *I4*, and maximal swimming work as intensity I5 (22). Training was quantified in meters covered in each intensity zone. The measurements were repeated 4 times per season, and training intensity was adjusted to the swimmer's response to training (26).

# **Strength Training**

The subjects participated in a supervised strength-training program, with a training frequency of 4 sessions per week during the volume phase, 3 sessions per week during the intensity phase, and 2 sessions per week during the taper phase. Strength training (I6) included dry land workouts, which involved various strength exercises. After a standardized 20-minute warm up, each training session included 1 exercise for the leg extensor muscles (bilateral knee extension exercises), 1 exercise for the arm extensor muscles (bench press), and 6 exercises for the main muscle groups of the body (chest press, shoulder press, isokinetic swim bench, surgical tubing, and medicine ball pullovers for the upper body; abdominal crunch for the trunk extensors). Each exercise was performed at 50-60% of a single maximal repetition (1RM) at the stroke rate corresponding to the specific swimming stroke rate. During the volume phase, the subjects performed 20-40 repetitions per set and 2-3 sets of each exercise. During the intensity phase, the number of sets was reduced and the subjects were required to complete as many repetitions as possible at the stroke rate corresponding to the specific swimming stroke rate. Finally, during the taper phase, the number of repetitions was reduced to 8-16, and the subjects had to maintain the specific swimming stroke rate, but had to perform each repetition as rapidly as possible.

Strength training was quantified in minutes of active exercise excluding resting periods. As each swimmer's stroke rate remained more or less stable during the course of the study, the method used to quantify strength

**Table 1.** The content of the 3 phases during the entire study period (137 training cycles) for 3 training loads ( $w^{LIT}$ ,  $w^{HIT}$ ,  $w^{ST}$ ), and the total volume of swim workouts (Total). Results are expressed in meters, minutes, and percentage of the total distance covered in each phase.

	Volume phase	Intensity phase		Taper phase				
$w^{LIT}$ (m)	$43,900 \pm 9,180$		$42,076 \pm 8,804$	†	$19,890 \pm 5,580$	ş		
$w^{HIT}$ (m)	$2,048 \pm 975$		$1,920 \pm 707$	†	$1,044 \pm 481$	ş		
$w^{ST}$ (min)	$90.6 \pm 30.1$	*	$77.3 \pm 42.8$	+	$26.6 \pm 30.2$	§		
Total (m)	$47,020 \pm 9,770$	*	$44,080 \pm 9,430$	‡	$20,968 \pm 5,820$	Ĩ		
$w^{LIT}$ (%)	$93.3 \pm 1.6$		$95.4 \pm 1.4$		$94.8 \pm 1.7$			
$w^{_{HIT}}(\%)$	$4.3 \pm 2.4$		$4.4\pm1.46$	‡	$5.3\pm1.8$			
$w^{_{ST}}(\%)$	$0.2\pm0.2$		$0.2 \pm 0.1$	‡	$0.1 \pm 0.1$	Ï		

\* p < 0.05 (volume phase vs. intensity phase).

 $\dagger$ ,  $\ddagger p < 0.01$  and p < 0.05, respectively (intensity phase vs. taper phase).

 $\{$ ,  $\| p < 0.01 \text{ and } p < 0.05$ , respectively (volume phase and taper phase).

training in terms of time spent (in minutes) appears to gauge total volume correctly.

#### **Quantification of the Training Stimulus**

Quantification of the training stimulus was performed as proposed by Avalos et al. (3). The weekly amount of training in each training zone was notated as  $V_{i,i}$ , where i ( $i = I1, \ldots, I6$ ) is the level intensity and t is the week number ( $t = 1, \ldots, T$ ; T = the total number of weeks). The weekly training volume of each intensity level was expressed as a percentage of the maximal volume measured at the same intensity level throughout the period studied for each subject. Training volumes were then expressed as a percentage for the training volume of type i performed in week t:  $x_{i,t} = V_{i,t} / [Max_{1 \le t \le T} (V_{i,t})]$ . This normalization allowed comparisons of the training stimulus of different units or intensities, using the same scale of values.

In the first part of the study (analysis of the residual effects of training), 3 weekly training loads were determined according to 3 training zones. Low-intensity training load  $w_t^{LIT}$  was the mean of the  $x_{1,t}$ ,  $x_{2,t}$ , and  $x_{3,t}$ . High-intensity training  $w_t^{HIT}$  was the mean of the  $x_{4,t}$  and  $x_{5,t}$ . Strength training  $w_t^{ST}$  consisted of dry land workouts  $x_{6,t}$ . In the second part of the study (comparison of BM and MM), the total weekly training load,  $w_p$  was the mean of the weekly stimulus for each normalized training intensity for week t, and was expressed as a percentage of the maximal training stimulus performed by each swimmer during the period studied.

#### **Performances**

For each swimmer, performances were measured during real competitions for the same event, and during the entire study period. Performance at time t designated  $p_t$  was expressed as a percentage of the personal record of each swimmer.

### **Statistical Analyses**

All values were reported as mean  $\pm$  *SD*. For all variables, the hypothesis of a normal distribution was tested ( $p \leq 0.05$ ) with the Shapiro Wilk test for small samples (performances), and with the Kolmogorov test for large samples (training loads) (25). The Bartlett test was used to control performances' unequal variances (25). If heteroscedasticity or an abnormal distribution were observed, logarithmic (natural) transformation of the data was performed. Statistical significance was accepted as less than or equal to the type I error rate of 0.05.

#### **Residual Effects of Training**

A linear model of periodization characterized the training cycles (2, 11, 13): each training cycle, lasting between 8 and 12 weeks, commenced with high training volume and low intensity. As training progressed, volume decreased and intensity increased. For the whole group, 3 training phases were identified during each training cycle. The taper phase was defined as the last 3 weeks prior to the competitive period (weeks 0, 1, and 2). The intensity phase was defined as weeks 3, 4, and 5 prior to the competitive period. The volume phase was defined as weeks 6, 7, and 8. The training effects of these 3 phases were defined as short-term (STE), intermediate-term (ITE) and long-term (LTE) effects for the taper, intensity, and volume phases, respectively. Ultimately, 9 distinct training variables were defined:  $STE w_t^{LIT}$ , STE  $w_t^{HIT}$ ,  $STE w_t^{ST}$ ,  $ITE w_t^{LIT}$ , I $LTE w_t^{HIT}$ , and  $LTE w_t^{ST}$ . ITE  $w_t^{HIT}$ , ITE  $w_t^{ST}$ , LTE  $w_t^{LIT}$ .

The content of the 3 phases of 137 training cycles  $(w^{LIT}, w^{HIT})$ , and  $w^{ST}$  were compared using 1-way analysis of variance (Table 1).

To analyze the relationships between loads and performances within each training cycle, multiple regression analysis was computed between performances (dependent variable) and the training variables (independent variables). Each training variable was transformed by a quadratic function (25) to take into account a potential parabolic relationship between the quantity of training loads and the performances.

After testing the normality and homoscedasticity of the residuals, 95% confidence intervals (CI) were calculated for regression parameters.

# Comparison of Banister vs. Modified Model: The Modified Model

With BM, the training impulse effect was represented as a linear function of the amount of this impulse limit:  $k_a w_s$  and  $k_j w_s$  (4, 21). In MM, the Hill function (18) was used to model the existence of a threshold in the doseeffect relationship, using the equation

$$Hill(w) = \kappa \frac{w^{\gamma}}{\delta^{\gamma} + w^{\gamma}}$$

where  $\kappa$  is the value of the saturation threshold above which training loads no longer have an effect. The parameter  $\gamma$  expresses the sensitivity to training load and controls the time to reach  $\kappa$  (the higher the value of  $\gamma$ , the shorter the delay). The parameter  $\delta$  is the inertia of the



**FIGURE 1.** Hill function pattern for 3 different  $\gamma$  values when  $\delta = 1$  and  $\kappa = 10$  (A); and for 3 different  $\delta$  values when  $\kappa = 10$  and  $\gamma = 1$  (B). The saturation threshold is rapidly reached for high  $\gamma$  and low  $\delta$  values.

function to the threshold value. A low value of  $\delta$  expresses a strong effect of training load on performance. The effects of 3 different values of  $\gamma$  and  $\delta$  are shown in Figure 1.

The positive and negative functions for week t ( $\omega_{p,t}$  and  $\omega_{n,p}$ , respectively) can be expressed as follows, with  $t = 1, \ldots, T$  and T being the total number of weeks:

$$\omega_{p,t} = \kappa_p \frac{w_t^{\gamma}}{\delta^{\gamma} + w_t^{\gamma}} \qquad \omega_{n,t} = \kappa_n \frac{w_t^{\gamma}}{\delta^{\gamma} + w_t^{\gamma}}$$

where  $\kappa_n$  and  $\kappa_p$  are the saturation threshold value of the negative  $\omega_{n,t}$  and positive  $\omega_{p,t}$  functions, expressed in arbitrary units (a.u.),  $\gamma$  is the time to reach the saturation threshold (a.u.); and  $\delta$  is the training load inertia coefficient (a.u.). The performance function at time *t* can be expressed as:

$$p_t = p_0 = \sum_{s=0}^{t-1} \omega_{p,s} e^{-(t-s)/\tau_p} - \sum_{s=0}^{t-1} \omega_{n,s} e^{-(t-s)/\tau_n}$$

where  $p_0$  is the initial basic performance level corresponding to the genetic endowment of the subject (7) expressed in the same units as performance, as a percentage (%) of each swimmer's personal record during the entire study period;  $\tau_p$  and  $\tau_n$ , the decay time constants (expressed in days or weeks) for positive and negative functions, respectively.

#### **Fitting the Models**

Model parameters were estimated for each subject using the iterative method of nonlinear least squares, by minimizing the residual sum of quadratic differences between the real and the modeled performances with a Gauss-Newton type algorithm (25). The starting values were chosen as follows:  $p_0 = 0.95$ ,  $k_a = 1$ ,  $k_f = 2$ ,  $\tau_a = 45$  days, and  $\tau_f = 15$  days for both BM and MM (20). All analyses were completed using Matlab 2000, 6.0 Optimization Toolbox, (Mathworks Inc., Boston, MA).

The determination coefficient was calculated as follows:  $r^2 = 1 - (\text{RSS/TSS})$ , where RSS is the residual sum of squares and TSS is the total sum of squares. Because increasing the number of parameters increases the determination coefficient, the adjusted determination coefficient ( $r_{adj}^2$ ) was calculated as follows:  $r_{adj}^2 = 1 - ([\text{number of pa$  $rameters } - 1]\text{RSS/TSS})$ . The adjusted determination coefficient takes into account the fitting gain with respect to the two parameters ( $\gamma$ ,  $\delta$ ) added by modifying BM.

Because BM and MM were not nested, the  $C_p$  score was computed as a comparison criteria (10, 25, 31):  $C_p = [RSS/(number of observations) + 2(number of parameters)\hat{\sigma}^2/(number of observations)], where <math>\hat{\sigma}^2$  is the standard unbiased estimator of the residual variance. A small value of  $C_p$  indicates a small prediction error (10, 25, 31).

## **Methodological Issues**

Although  $r^2$  is one of the most important indicators of adequacy of regression equations, a high  $r^2$  value is not a guarantee of accurate prediction. Several complementary measures are needed to confirm accuracy and sensitivity (25, 31). The analysis of variance applied to the residual sum of squares was not suitable to compare BM with MM, because they were not nested (models are nested when the parameters of one model are a subset of the parameters of another). The calculation of  $C_p$  is a useful statistical method that rewards models for good fit, but imposes a penalty for unnecessary parameters (10, 25, 31). To summarize,  $r^2$ and  $r^2_{adj}$  measure the goodness of fit, whereas  $C_p$  and CI measure the prediction error and the accuracy of estimation, respectively (10, 17).

#### **Bootstrap Method**

The bootstrap method (10) was used to compute the limits of agreements of estimated performances. Briefly, the procedure consisted of resampling the original data set with replacement, to create a number of "bootstrap replicate" data sets of the same size as the original data set. A random number generator was used to determine which data from the original data set to include in a replicate data set, and therefore, a given datum could be used more than once in the replicate data set, or not at all. This process was repeated 1,000 times. A 95% CI for the performances estimated was constructed from the estimates that fell between percentiles 2.5 and 97.5 of the 1,000 estimates (10). The number of actual performances included in the 95% CI for the performances estimated was compared for BM and MM.

#### **Positive and Negative Effects of Training**

To separate the short-term negative effects of the training doses from their long-term benefit, the positive and negative effects of training on performance were estimated as previously described (6, 23). The effect on performance on week t attributable to the amount of training during week s, for both BM and MM, was quantified as:

 $E(s/t) = k_1 w^s e^{-(t-s)/\tau_1} - k_2 w^s e^{-(t-s)/\tau_2}$ , where  $k_1$  and  $k_2$  corresponded to  $k_a$  and  $k_f$  for BM and  $k_p$  and  $k_n$  for MM, and  $\tau_1$  and  $\tau_2$  corresponded to  $\tau_a$  and  $\tau_f$  for BM and  $\tau_p$  and  $\tau_n$  for MM.

A negative or a positive value indicated a negative or

Subject #	Event (m)	Personal records	Best time of first season	Best time of last season	% improvement
1	100 Free*	0:55.11	0:56.63	0:55.44	0.27
2	200 Fly	2:10.80	2:12.85	2:10.80	0.38
3	200 Free	1:59.57	2:01.47	1:59.57	1.56
4	200 Mixed	2:00.75	2:01.08	2:00.75	0.27
5	100 Breast	1:03.17	1:03.85	1:03.61	0.38
6	200 Back	2:14.00	2:17.06	2:15.00	1.56
7	100 Free	00:51.47	0:51.58	0:51.47	0.27

**Table 2.** Individual swimming times at the beginning and the end of the study, and percentage improvement during the study period.

\* Free = freestyle; Fly = butterfly; Breast = breaststroke; Back = backstroke; Mixed = 4 styles alternated.

positive effect, respectively, of training on performance. Effects of training impulses at 100, 65, and 35% of the maximal training load were compared for BM vs. MM in subjects #2 and #3.

#### RESULTS

## **Training Characteristics**

In the whole study group, the training volume measured during a season was  $1,922 \pm 417$  km. Contents of the volume, intensity, and taper phases during the entire study period ( $w^{LIT}$ ,  $w^{HIT}$ , and  $w^{ST}$ ) are shown in Table 1.

Only training volume and strength training decreased between the volume and the intensity phases (p < 0.05), whereas training volume, low intensity, high intensity, and strength training decreased between the intensity and the taper phases (p < 0.05). The percentage of high-intensity training increased as the percentage of strength training decreased between the intensity and the taper phases (p < 0.05). The total training load ( $w_i$ ) was  $34.0 \pm 14.2\%$  of the maximal training stimulus (range, 0.12-85.3%) measured throughout the period studied.

#### **Competitive Performances**

During the entire study period, the mean number of performances recorded for each swimmer were  $48.7 \pm 9.1$ . For the whole group, the mean performance was  $96.6 \pm 1.9\%$ (range, 92.8-100%). Best performances between the beginning and end of the study improved by 0.67% (range, 0.27-1.56%) (Table 2).

#### **Residual Effects of Training**

The best solution ( $r^2 = 0.30$ , F = 8.73, p < 0.01) for the multiple regression was

$$p_t = 0.97 - 0.46(STE w_t^{LIT})^2 + 0.28(LTE w_t^{LIT})^2$$

$$+ 0.25(ITE w_t^{HIT}).$$

Only significant variables were included in the multiple regression (p < 0.05). A better adjustment of the transformed variable (*STE*  $w_t^{LTT}$ )<sup>2</sup> indicated a parabolic relationship between short-term, low-intensity amounts of training and performance (Figure 2). The 95% CI was [-0.26; -0.65] for (*STE*  $w_t^{LTT}$ )<sup>2</sup>, [0.12; 0.43] for *LTE*  $w_t^{LTT}$ , and [0.10; 0.40] for *ITE*  $w_t^{HTT}$ .

## **Fitting Accuracy**

The parameters of BM and MM are presented in Table 3. The relationships between training and performance were significant (p < 0.01) for the 2 models in all subjects. The determination coefficients ( $r^2$ ) were higher for MM than for BM:  $0.42 \pm 0.1 (0.30 \le r^2 \le 0.53)$  in BM vs.  $0.52 \pm 0.1$ 



**FIGURE 2.** Parabolic relationship between short-term, low-intensity training load (*STE*  $w_t^{LT}$ ) and performances  $(p_t)$ , for the whole group of subjects. Performance on the vertical axis is expressed as a percentage of the personal record of each subject. Training load on the horizontal axis is expressed as a percentage of the maximal training load performed by each subject during the course of the study.

 $(0.32 \le r^2 \le 0.58)$  in MM. With  $r_{adj}^2$ , the fit of BM  $(0.36 \pm 0.1 \ [0.23 \le r_{adj}^2 \le 0.49])$  was slightly lower than that of MM  $(0.43 \pm 0.1 \ [0.21 \le r_{adj}^2 \le 0.51])$ . When comparing MM with BM, the C<sub>p</sub> score was lower

When comparing MM with BM, the  $C_p$  score was lower in 5 subjects (#1, #3, #4, #5, #7) and higher in 2 (#2, #6). The fitting difference between BM and MM associated with CI for subject #5 is shown as an example in Figure 3.

The mean interval width of 95% CI in BM and MM (1.92  $\pm$  0.42 vs. 1.91  $\pm$  0.41%) were similar. The number of measured performances included in the 95% CI (PCI) was higher in BM than in MM (25.7  $\pm$  4.8 vs. 20.1  $\pm$  3.1). The 95% CI for subject #5 in both models is shown in Figure 3.

# **Positive and Negative Effects of Training**

The effects of training impulses at 100, 65, and 35% of the maximal training load were different for BM and MM. Two practical examples of subjects #2 and #3 are displayed in Figure 4A and Figure 4B, respectively. In BM, the magnitude of the responses was proportionally related to the amount of training impulses in both subjects. With MM, in subject #2, the 2 training doses (100 and 65%) induced markedly different responses (0.009 and 0.007 a.u., respectively). In subject #3, the training doses (100 and 65%) brought about two positive effects of similar magnitude (0.003 a.u.).

#### DISCUSSION

There were 2 main observations emerging from these analyses. First, the relationship between training load and per-

Table 3. Results and quality measures for Banister Model (BM) and Modified Model (MM).

Subject #	$M^*$	N	$p_0$	$ au_{\omega}$ $ au_p$	$ au_{ m p}$ $ au_n$	$k_{a}$ , $\kappa_{p}$	$k_{\beta} \kappa_n$	γ	δ	$r^2$	$r_{ m adj}^2$	$C_{ m p}$	PCI
1	BM	38	94.2	45	17	0.01	0.03			0.42	0.32	4.12	16
	$\mathbf{M}\mathbf{M}$	38	94.2	56	7	0.04	0.08	6	0.1	0.48	0.36	3.74	18
2	BM	57	95.4	40	10	0.03	0.08			0.30	0.24	1.52	22
	$\mathbf{M}\mathbf{M}$	57	94.2	24	23	0.78	0.74	1	10	0.32	0.21	1.60	29
3	BM	62	96.2	56	14	0.01	0.03			0.47	0.45	2.33	18
	$\mathbf{M}\mathbf{M}$	62	97.1	37	30	0.43	0.67	6.9	0.4	0.54	0.48	2.28	24
4	BM	41	97.3	45	28	0.01	0.02			0.39	0.32	3.24	21
	$\mathbf{M}\mathbf{M}$	41	99.1	39	21	0.22	0.42	0.1	1.2	0.57	0.48	2.76	31
5	BM	51	93.3	55	14	0.02	0.09			0.52	0.48	1.69	17
	$\mathbf{M}\mathbf{M}$	51	95.4	54	14	0.06	0.24	1.2	1.6	0.58	0.51	1.66	31
6	BM	52	90.3	45	18	0.01	0.06			0.53	0.49	1.92	24
	$\mathbf{M}\mathbf{M}$	52	89.1	40	18	0.43	0.67	0.9	4.3	0.57	0.51	2.02	24
7	BM	41	91.5	61	33	0.01	0.07			0.34	0.23	1.93	23
	$\mathbf{M}\mathbf{M}$	41	90.1	56	11	0.44	0.11	0.4	5.1	0.55	0.44	1.91	23
Mean	BM	48.7	94.0	49.6	19.1	0.01	0.05			0.42	0.36	2.39	20.1
SD		9.1	2.5	7.7	8.3	0.01	0.03			0.1	0.1	0.95	3.1
Mean	$\mathbf{M}\mathbf{M}$	48.7	94.2	43.7	17.7	0.34	0.42	2.36	3.24	0.52	0.43	2.28	25.7
SD		9.1	3.6	12.1	7.8	0.26	0.28	2.83	3.54	0.1	0.1	0.75	4.8

\* M = model; N = number of performances;  $p_0$  = basic performance level (%);  $\tau_a$  and  $\tau_f$  = fitness and fatigue decay time constants, respectively, for BM (days);  $\tau_p$  and  $\tau_n$  = positive and negative decay time constants, respectively, for MM (days);  $k_a$  and  $k_f$  = fitness and fatigue multiplying factors, respectively, for BM arbitrary units [a.u.];  $\kappa_p$  and  $\kappa_n$  = saturation threshold for the positive and negative components of training, respectively, for MM (a.u.);  $\gamma$  = time to reach threshold (a.u.);  $\delta$  = sensitivity to training load (a.u.);  $C_p$  = score accounting for prediction error; PCI = actual performances in 95% CI for performances estimated.



**FIGURE 3.** Modeled and actual performances (indicated with an irregular curve and diamonds at each data point) for subject #5, calculated with modified model (MM) and Bannister model (BM). Performance on the vertical axis is expressed as a percent of the personal record. Time on the horizontal axis is expressed in weeks. The adjusted determination coefficient and 95% CI for modeled performances are also represented. The 95% CI included 17/51 actual competitions for BM, vs. 31/51 for MM.

formance varied according to the training phases and training loads. The short-term effect of training was related to performance by a parabolic relationship for  $w^{LIT}$ , the intermediate-term effect was positive for  $w^{HIT}$ , and the long-term effect was positive for  $w^{LIT}$ . Second, BM and MM were significantly fitted with the training load-performance relationships. However, MM, by including a saturation threshold, improved the fit between training and performance significantly, compared with BM.

One of the major limitations of this research concerns its nonexperimental schedule. The lack of random sampling or random assignment to groups makes it difficult to generalize these findings to other situations. Experiments are better than observational studies, because there are fewer grounds for doubt. Experiments often settle questions faster. Despite this, experiments are not feasible in some settings.

Furthermore, the quantification method remains overly restrictive and does not take into account all types of training intensities.

The most important short-term effect was derived from training performed below and just above the onset of blood lactate accumulation ( $w^{LIT}$ ), which usually accounts for the greatest proportion of training in swimmers (3, 22, 23, 27). Tapering enables swimmers to recover from fatigue accumulated during intermediate- and long-term training, while maintaining previously acquired physical adaptation (16, 22, 23).

Nevertheless, the best regressor in the equation was  $(STE \ w^{LIT})^2$ , the relationship between low intensity training and performance as a parabolic relationship (Figure 2), indicating that *LIT* training amount has to decrease to 40–50% of the maximal training load during the taper phase; a further decrease may cause loss of training effect (24).

The effect of  $w^{HT}$  on performance was positive. This range of intensity optimizes aerobic and anaerobic energy production (23), and improves swimming techniques (28). Several authors have emphasized the importance of this training period, during which the increase in training intensity delays the stimulation of biological adaptations via an overcompensatory process (3, 13, 20, 27).

Low-intensity training had a positive effect on performance over the long term. These results suggest that an important low intensity training volume probably efficiently develops the physiological mechanisms necessary for subsequent intensity training (9, 14, 27). Aerobic training, as-



**FIGURE 4.** Time response of performance for subject #2 (A) and #3 (B) to 3 training impulses of 100, 65, and 35% of the maximal training load, for Bannister model (BM) ( $\_\_$ ) and modified model (MM) ( $\_\_$ ). Time in horizontal axis is expressed in weeks. In BM, training impulses were proportional to the training loads, with a higher load being related to more positive or negative effects. In MM, the relationship between loads and impulses was nonlinear and had an upper limit. In subject #2, the responses to the 100% vs. 65% of the maximal training load were distinctly different, consistent with a high upper limit. Conversely in subject #3, training impulses at 100 and 65% of the maximal training load elicited similar response patterns, suggesting a low upper limit.

sociated with a lactate production equal to or below the onset of blood lactate accumulation increases oxidative capacity, lowers lactate production at a given swimming speed, increases critical speed, and increases training capacity while lowering the fatigue threshold (30).

The 95% CI of the different parameters of the multiple regression between performance and training variables confirmed an accurate estimation. The practical implications of these results remain to be clarified, because the training variables explained only 30% of the variations in performance. There are several explanations. First, the swimmer's response to a given training volume may vary among consecutive seasons, reducing the statistical significance of the relationship between training and performance (3). Indirect effects of training may also interfere. For example, aerobic training may hasten the recovery from fatigue caused by anaerobic training (9). Variations in technique may also explain a large part of the variations in performance (29). Furthermore, swimmers react differently to the same training loads (3). Finally, during the study period, performance improved by less than 1%, suggesting that, after several years of high-level training, the performance of elite athletes reaches a plateau (14, 28). Therefore, as variations in training do not directly imply variations in performances, statistical relationships are lower.

With both models, the fit between training and performance was significant in all subjects. The determining coefficients were similar to those reported by Avalos et al. (3) who used a linear mixed model with 13 competitive swimmers over 3 seasons. They were, however, lower than reported in earlier studies of swimmers (22, 23), probably due to a larger number of performances for each swimmer and a longer study duration. For small samples, the mean  $r^2$ value may be high despite the absence of relationship between predictor and response variable (1). The current study resulted in  $r_{adj}^2$  values for BM that were smaller than those reported by Busso (6) (0.36  $\pm$  0.11 vs. 0.88  $\pm$  0.04). However, in that study, sedentary subjects were trained over 15 weeks and improved their performance by approximately 30% during the period studied, an improvement much greater than can be expected in elite athletes (1-4%)(15)

Moreover, both BM and MM assume that the parameters remain constant over time, an assumption that is inconsistent with observed time-dependent alterations in responses to training (3, 5, 6, 8). Although performance is specifically and largely influenced by training, athletes also adapt to other factors whose influence may increase over time, including personal involvement, intensity swimming techniques, external factors, altitude training, and time-lag during travel (3, 22, 23).

On average, the  $r_{adj}^2$  coefficients were slightly higher (p < 0.05) in MM than in BM (0.42 ± 0.10 vs. 0.36 ± 0.13). This result is consistent with the adjustment increase reported by Busso (6) by comparing BM to a nonlinear model that took into account the magnitude and duration of exercise-induced fatigue (0.88 ± 0.04 vs. 0.94 ± 0.01).

Since the mean interval width of 95% CI was similar (~1.9  $\pm$  0.4%) in both models, the highest number of measured performances included in the 95% CI for MM indicates a higher accuracy and best specification of the latter model (31). Thus, MM can be considered to be a complementary tool to BM for modeling the relationship between training and performance. Moreover, Olympic level of the subjects, the long duration of the study, and the use of CI contributed to the validation of BM. Indeed, from a practical point of view, the CI values were accurate (e.g., for subject #5, CI was 1 second for a 100-m event performed in 1 minute, 3 seconds).

On average, positive function decay rates were shorter in MM than in BM (43.7 ± 12.1 days vs. 49.6 ± 7.7 days), whereas negative function decay rates were similar (19.1 ± 8.3 days vs. 17.7 ± 7.8 days). The positive and negative function values in BM and MM were close to those reported previously (4, 5, 8, 23). The fitness and fatigue acquisition coefficients  $k_{\omega}$   $k_f$  in BM were lower than the positive and negative saturation thresholds  $\kappa_p$   $\kappa_n$  in MM (0.01 ± 0.01 vs. 0.34 ± 0.26 a.u. and 0.05 ± 0.03 vs. 0.42 ± 0.28 a.u.). The differences between these values may be explained by the differences in the models' structures. In MM, the saturation coefficients for the positive and negative function,  $\kappa_p$  and  $\kappa_n$ indicating a threshold limit beyond which training no longer has any effect on performance, do not have the same meaning as the fitness  $k_a$  and fatigue  $k_f$  acquisition coefficients in BM, representing fitness and fatigue acquisition amplitudes (Figure 1).

#### **PRACTICAL APPLICATIONS**

Summarizing, in this study of elite swimmers, low-intensity training was related to short-term performance by a parabolic relationship, whereas its long-term effect during the volume phase was positive. These results underscore the positive effects of low-intensity training during the volume phase and suggest that this type of training should be maintained around 40–50% of the individual maximal values during the taper phase. Moreover, MM may better take into account, for each swimmer, the limit above which training does not elicit subjects' adaptations and the delay in reaching this limit. For example, subject #2, who was an Olympic finalist in the 200-m butterfly event, responded quite differently to the 2 training doses (100 and 65%), as shown in Figure 4A, and may respond well to high training loads. Conversely, subject #3, who was an Olympic finalist in the 200-m freestyle event in Atlanta in 1996 and Sydney in 2000, determined similar response patterns to training impulses of 100 and 65% (Figure 4B), suggesting a poor response to high training loads. These results are consistent with those reported by Avalos et al. (3) who used a linear mixed model. Subject #3 was included in the group of swimmers responding poorly to short- or midterm high training loads. Conversely, subject #2 was included in the group of swimmers responding well to short-term high training loads (3). Finally, adaptation to training is known to be a highly individual phenomenon (3, 23). Thus, these results suggest that training programs must be highly personalized, adapted to each individual swimmer's profile.

#### Perspectives

The present MM could be further refined. First, the change in  $p_0$  could be integrated into the modeling process (for example, at the beginning of each season), because  $p_0$  fluctuates from year to year, whereas in shorter studies (6, 8) it seems preferable to use time-varying positive and negative parameters. Second, MM could be computed with a mixed procedure (3), to integrate the intra- and interindividual variability and to take into account the temporal closeness of the competitions.

The results of this study need to be generalized. A batch of experimental studies based on longitudinal research design would make it possible to compare several training programs over an entire training cycle. Considering the popularity of periodized training, there are surprisingly few studies examining the effectiveness of periodized training loads. The first step would be to compare linear periodization with a nonperiodized training involving constant volume and intensity training loads. The second step would be to investigate any residual effects and the threshold effects of training by comparing progressive multiple-training-load programs. For instance, it would be highly instructive to compare the effects of 2, 3, and 4 sessions of high training loads per week throughout the intensity phase (weeks 3, 4, and 5). By the same token, it also would be appropriate to study the optimal training volume during the volume phase (weeks 6, 7, and 8). Finally, studies examining periodized models other than the traditional volume/intensity periodized model are also needed.

## REFERENCES

- ALI, M.A. Effect of sample size on the size of the coefficient of determination in simple linear regression. J. Inform. Optim. Sci. 8:209-219. 1987.
- 2. AMERICAN COLLEGE OF SPORTS MEDICINE. Progression models in resistance training for healthy adults. *Med. Sci. Sports Exerc.* 34:364–380. 2002.
- AVALOS, M., P. HELLARD, AND J.C. CHATARD. Modeling the training-performance relationship using a mixed model in elite swimmers. *Med. Sci. Sports Exerc.* 35:838–846. 2003.
- 4. BANISTER, E.W., T.W. CALVERT, AND M.V. SAVAGE. A systems model of training for athletic performance. *Can. J. Sports Med.* 7:57–61. 1975.
- BANISTER, E.W., J.B. CARTER, AND P.C. ZARCADAS. Training theory and taper: Validation in triathlon athletes. *Eur. J. Appl. Physiol.* 79:182–191. 1999.
- BUSSO, T. Variable dose-response relationship between exercise training and performance. *Med. Sci. Sports Exerc.* 35:1188– 1195. 2003.
- BUSSO, T., C. CARASSO, AND J.R. LACOUR. Adequacy of a systems structure in the modeling of training effects on performance. J. Appl. Physiol. 71:2044–2049. 1991.
- BUSSO, T., C. DENIS, AND R. BONNEFOY. Modeling of adaptations to physical training by using a recursive least squares algorithm. J. Appl. Physiol. 82:1685–1693. 1997.
- 9. COUNSILMAN, B.E., AND J.E. COUNSILMAN. The residual effects of training. J. Swimming Res. 7:5–12. 1991.
- EFRON, B., AND R. TIBSHIRANI. An introduction to the bootstrap. In: *Monographs on Statistics and Applied Probability*. New York: Chapman and Hall, 1993. p. 237.
- FLECK, S.J. Periodized strength training: A critical review. J. Strength Cond. Res. 13:82–89. 1999.
- FRY, R.W., AND A.R. MORTON. Overtraining in athletes: An update. Sports Med. 12:32–65. 1991.
- FRY, R.W., A.R. MORTON AND D. KEAST. Periodisation of training stress—A review. Can. J. Sport Sci. 17:234–240. 1992.
- GASKILL, S.E., R.C. SERFASS, D.W. BACHARACH, AND J.M. KEL-LY. Responses to training in cross-country skiers. *Med. Sci. Sports Exerc.* 31:1211–1217. 1999.
- HOOPER, S.L., L.T. MACKINNON, AND E.M. GINN. Effects of three tapering techniques on the performance, forces and psychometric measures of competitive swimmers. *Eur. J. Appl. Physiol.* 78:258–263. 1998.
- HOPKINS, W.G., AND D.J. HEWSON. Variability of competitive performance of distance runners. *Med. Sci. Sports Exerc.* 33: 1588–1592. 2001.
- HUET, S. Statistical tools for nonlinear regression: A practical guide with S-PLUS examples. In: Springer Texts in Statistics. New York: Springer-Verlag, 1996.
- KRZYZANSKI, W., R. RAMACKRISHNAN, AND W.J. JUSKO. Basic pharmacodynamic models for agents that alter production of natural cells. J. Pharmacokinet. Biopharm. 27:467-489. 1999.
- MADER, A. A transcription-translation activation feedback circuit as a function of protein degradation, with the quality of protein mass adaptation related to the average functional load. *J. Theor. Biol.* 134:135–157. 1988.
- MORTON, R.H. The quantitative periodization of athletic training: A model study. Sports Med. Train. Rehab. J. 3:19-28. 1991.
- MORTON, R.H. Modelling training and overtraining. J. Sports Sci. 15:335–340. 1997.
- MUJIKA, I., T. BUSSO, L. LACOSTE, F. BARALE, A. GEYSSANT, AND J.C. CHATARD. Modeled responses to training and taper in competitive swimmers. *Med. Sci. Sports Exerc.* 28:251–258. 1996.
- MUJIKA, I., J.C. CHATARD, T. BUSSO, AND A. GEYSSANT. Effects of training on performance in competitive swimming. *Can. J. Appl. Sport Sci.* 20:395–406. 1995.

- MUJIKA, I., AND S. PADILLA. Detraining: Loss of training-induced physiological and performance adaptations. Part 1. Short term insufficient training stimulus. Sports Med. 30:78– 87. 2000.
- SEN, A., AND M. SRIVASTAVA. Regression analysis: Theory, methods, and applications. In: *Springer Texts in Statistics*. New York, Berlin, Paris: Springer-Verlag, 1990. pp. 40–41, 105–106, 237–240, 298.
- SHIMOYAMA, Y., AND T. NOMURA. Role of rest interval during interval training at OBLA speed. In: *Biomechanics and Medicine in Swimming VIII.* K.L. Keskinen, P.V. Komi, and A.P. Hollander, eds. Finland: University of Jyväskylä, 1999. pp. 459–463.
- STEWART, A.M., AND W.G. HOPKINS. Seasonal training and performance of competitive swimmers. J. Sports Sci. 18:834–873. 2000.

- TERMIN, B., AND D.R. PENDERGAST. Training using the stroke frequency-velocity relationship to combine biomechanical and metabolic paradigms. J. Swimming Res. 14:9–17. 2000.
- 29. TOUSSAINT, H.M., AND A.P. HOLLANDER. Energetics of competitive swimming—Implications for training programs. Sports Med. 18:384–405. 1994.
- WAKAYOSHI, K., T. YOSHIDA, AND M. UDO. Does critical swimming velocity represent exercise intensity at maximal lactate steady state? *Eur. J. Appl. Physiol.* 95:66–90. 1993.
- WETHERILL, G.B., P. DUNCOMBE., AND M. KENWARD. Regression analysis with applications. In: *Monographs on statistics and applied probablility*. London, New York: Chapman and Hall, 1986. pp. 27–28, 241–242.

Address correspondence to Marta Avalos, avalos@hds.utc.fr.