REVIEW ARTICLE

Sports Med 2009; 39 (9): 779-795
0112-1642/09/0009-0779/$49.95/0

The Quantification of Training Load, the Training Response and the Effect on Performance

Jill Borresen and *Michael Ian Lambert*

MRC/UCT Research Unit for Exercise Science and Sports Medicine, Department of Human Biology, University of Cape Town, Cape Town, South Africa

Contents

Abstract

Historically, the ability of coaches to prescribe training to achieve optimal athletic performance can be attributed to many years of personal experience. A more modern approach is to adopt scientific methods in the development of optimal training programmes. However, there is not much research in this area, particularly into the quantification of training programmes and their effects on physiological adaptation and subsequent performance. Several methods have been used to quantify training load, including questionnaires, diaries, physiological monitoring and direct observation. More recently,

indices of training stress have been proposed, including the training impulse, which uses heart rate measurements and training load, and session rating of perceived exertion measurements, which utilizes subjective perception of effort scores and duration of exercise. Although physiological adaptations to training are well documented, their influence on performance has not been accurately quantified. To date, no single physiological marker has been identified that can measure the fitness and fatigue responses to exercise or accurately predict performance. Models attempting to quantify the relationship between training and performance have been proposed, many of which consider the athlete as a system in which the training load is the input and performance the system output. Although attractive in concept, the accuracy of these theoretical models has proven poor. A possible reason may be the absence of a measure of individuality in each athlete's response to training. Thus, in the future more attention should be directed towards measurements that reflect individual capacity to respond or adapt to exercise training rather than an absolute measure of changes in physiological variables that occur with training.

The ultimate goal of any sports coach and athlete is to produce a winning or personal best performance at a specific time, preferably in competition. The prescription of the training required to achieve this goal has been largely instinctive, resulting from years of personal experience. As such, the ability to achieve peak fitness and performance coinciding with dates of competition is met with varying degrees of success. It is generally believed that increasing training will result in improvements in sporting performance and physical well-being. However, although widely accepted, this vague approach to prescribing training may be tenuous, especially because random increases in training volume, intensity or frequency may also increase the likelihood of injury and symptoms of overtraining.[1-5] The role of scientific research in this process is becoming more important in order to prescribe optimal training programmes that prevent both under- and overtraining and increase the chance of achieving desired performances.

The frequency, duration and intensity of exercise all contribute to the nature and magnitude of the training effect. However, relatively little research has been conducted into the quantification of training programmes and their effects on physiological adaptation and subsequent performance. This is surprising because peaking for sporting performance requires an understanding of the quantifiable effects of training on performance so that optimal training and rest regimens may be planned in preparation for the event.

Optimizing training first involves quantifying what the athlete is currently doing. Several methods have been suggested to quantify exercise bouts, some of which will be reviewed below. Secondly, it needs to be established whether the athlete is adapting favourably to certain levels of exertion. Thereafter, training can be adjusted to optimize the athlete's improvement to meet a specific goal within a specified time. This review therefore presents methods currently being used to quantify training load, and assesses literature investigating the relationship between training load and the physiological response to training and performance. Finally, the importance of considering the variability in individual responses to training will be highlighted in the assessment of the training and performance relationship.

A PubMed search of the academic literature was performed using the following terms: 'quantification of training load', 'quantify exercise intensity', 'modelling training and performance', 'endurance training adaptations', 'training impulse', 'session RPE' and 'physiological response to training', limited to English papers and human subjects. Literature was also sourced from links to related articles, hand searches and the bibliographies of academic papers. The searches retrieved

2553 papers, of which 237 abstracts were reviewed and 114 full articles were evaluated.

1. Quantifying Training

1.1 Questionnaires and Diaries

Questionnaires and diaries obtain data recalled from the athlete (with diaries being completed more frequently [daily] than questionnaires), and are used to examine physical activity during the past week, month or even years.[6,7] The use of questionnaires to assess habitual physical activity and exercise, especially in large populations, is popular because their administration is easy, cost effective and does not impede training. However, their weakness is the fact that the athletes' responses are subjective.[6] Borresen and Lambert[8] studied the relationship between what athletes say they do in training and what they actually do. Twenty-four percent of the participants overestimated the duration of training they were doing, and 17% underestimated their training duration. Because this margin of error in self-reported data may significantly affect the prescription of training, it was recommended that the error be accounted for or, where possible, physiological measurements be used to corroborate self-reported data.[8] The use of data collected by questionnaires to quantify exercise load is also limited by inadequate reliability and validity compared with laboratory measures.[9] For example, reliability decreases as the time between the activity and recall increases, because this is dependent on human memory.[6,9]

A sports score derived from the Baecke questionnaire assesses the intensity of physical activity and has been used to estimate weekly training load.[10] The sports score is calculated using the duration (h/wk), frequency (mo/y) and intensity (dimensionless codes based on energy costs) of the activity.[10] There are, however, problems with questionnaires that assess the type, intensity, frequency and duration of the exercise and the environmental conditions in which the exercise was performed. For example, duration and frequency may be over-reported, especially if the person is influenced by the response he or she believes is sought by the investigators. Seasonal variations in duration and frequency of training may also not be taken into consideration. Perceptions of intensity may differ depending on experience or tolerance of the person, particularly if asked to report intensity as simply light, moderate, hard or very hard.

The environmental conditions under which the activity is performed may have important motivational, psychological and physical effects on the person, but these are often overlooked. Responses can also be influenced by differences in human understanding, which may be the result of cultural factors or of the translation of the questionnaire. The length of the questionnaire and the detail required from the participant may also affect results, as the person may become bored or confused with exhaustive questioning.[6,9] Therefore, although questionnaires may assist with monitoring general changes in population activity, attempts to quantify exercise dosage from data collected with questionnaires remain inadequate.[9]

1.2 Physiological Measures

1.2.1 Heart Rate

Heart rate monitoring has become a popular method for measuring exercise intensity.[11] This method is based on the principle that there is a linear relationship between heart rate and steady-state work rate.[6,12,13] Although absolute measures of intensity are commonly used, the relative equivalent may be more informative because considerable inter- and intraindividual differences may exist in the way people respond to various modes of exercise. Percent maximum/competition heart rate has been used to prescribe exercise intensity,[6] but Karvonen and Vuorimaa[14] suggest the use of percent heart rate reserve (equation 1) as a more accurate means of quantifying and prescribing intensity, because this method considers the fact that resting heart rate varies with age and fitness level, and maximal heart rate decreases with age.

$$\%\ \text{heart rate reserve} = \frac{(HR_{ex} - HR_{rest}) \times 100}{HR_{max} - HR_{rest}} \quad \text{(Eq. 1)}$$

where HR_{ex} is average heart rate of the exercise session, HR_{rest} is resting heart rate and HR_{max} is maximal heart rate.

Although heart rate monitors have been found to measure heart rate accurately during physical activity, many factors may influence the relationship between work load and heart rate. The day-to-day variation in heart rate is approximately 6 beats/min,[15] or <6.5%.[16] However, if the factors affecting heart rate, such as state of training, environmental conditions, diurnal changes,[13] exercise duration, hydration status, altitude[11,13] and medication, are controlled, the accuracy with which heart rate can be used as a marker of exercise intensity improves.[15]

1.2.2 Oxygen Consumption

Because it is generally accepted that the relationship between oxygen consumption ($\dot{V}O_2$) and steady-state work rate is linear,[13] $\dot{V}O_2$ has been promoted as a valid measure of exercise intensity during steady-state exercise, but not interval, supramaximal exercise bouts. In a review of the oxygen kinetics during exercise, Xu and Rhodes[17] point out that when exercising at a work rate below the lactate threshold, $\dot{V}O_2$ increases exponentially to a steady-state level, but when exercising above the lactate threshold, $\dot{V}O_2$ kinetics become more complex. Relative ($\%\dot{V}O_{2max}$) rather than absolute values of $\dot{V}O_2$ have been used to compare the exercise intensities executed by athletes of differing physiological and performance characteristics.[6] However, it has been found that $\dot{V}O_{2max}$ is exercise mode specific, so $\dot{V}O_{2max}$ needs to be determined for each mode before exercise can be prescribed or quantified using relative $\dot{V}O_2$ values.[18] Oxygen consumption reserve (VO_2R, equation 2) has been suggested as a more accurate means with which to prescribe exercise intensity than $\%\dot{V}O_{2max}$:

$$\%VO_2R = \frac{(VO_{2\,ex} - VO_{2\,rest}) \times 100}{VO_{2\,max} - VO_{2\,rest}} \quad \text{(Eq. 2)}$$

It has been shown in cycling and running that whereas calculating exercise intensity using heart rate reserve and VO_2R give similar results, the exercise intensities calculated using $\%\dot{V}O_{2max}$ differ.[19,20] Baldwin et al.[21] found that heart rate and plasma markers of exercise stress such as lactate, ammonia and hypoxanthine at $70\%\dot{V}O_{2peak}$ were different between trained and untrained individuals. This supports the suggestion that using $\%\dot{V}O_{2peak}$ does not necessarily produce the same physiological response in different people. It has also been found that the $\dot{V}O_2$ kinetics at the onset of exercise may differ with level of physical training, age and disease. As such, the use of $\dot{V}O_2$ may be inappropriate as a means with which to prescribe relative exercise intensity.[21,22]

1.2.3 Lactate

The measurement of blood lactate concentrations has become easier with the development of portable measurement instruments and requiring the collection of only one drop of blood from a finger prick. Nevertheless, it remains impractical to measure lactate frequently during every training session in order to prescribe or quantify intensity. Particular attention has been paid to determining the lactate threshold, which is defined as the exercise intensity at a fixed or maximal steady-state blood lactate level.[23] It has been proposed as a measure of endurance fitness, but also a means with which to standardize training intensity. The steady-state exercise intensity that elicits a lactate concentration of approximately 4 mmol/L has been suggested as the most favourable for inducing optimal physiological adaptations for endurance events.[24,25] However, Stegmann et al.[26] warn that this 'optimal' lactate level may range from 2 to 7.5 mmol/L among athletes.

The inherent inter- and intraindividual differences in the extent to which lactate accumulates during exercise are two limitations of many in the use of lactate to prescribe exercise intensity. Extraneous factors such as ambient temperature and dehydration may influence the interpretation of lactate measurements. Mode of exercise can also be an influence, as it alters the muscle mass used during exercise[27] such that the same lactate concentration occurs at different $\dot{V}O_2$ levels during running and cycling. Exercise duration, intensity and the rate of change in exercise intensity may also influence lactate concentration, as may prior

exercise, diet and muscle glycogen content.[24,27] Exercising with damaged muscles has also been shown to cause an increase in lactate levels.[27] Improvements in training status as well as overtraining have both been associated with decreases in maximal and submaximal blood lactate concentration,[28-30] which may lead to erroneous interpretations of lactate measurements and incorrect exercise prescription.[27] The interpretation of lactate concentration may further be affected by sampling and measurement procedures such as the time and site of blood sampling, measurement techniques and dilution volume.[24,27] The extent to which the abovementioned factors affect the way lactate accumulates, independent of exercise intensity, makes the importance of the lactate threshold less definitive,[26] thus limiting its usefulness in monitoring and prescribing training intensity.

1.2.4 Rating of Perceived Exertion (RPE)

A rating of perceived exertion (RPE) is based on the understanding that athletes can inherently monitor the physiological stress their bodies experience during exercise, and thus be able to adjust their training intensity using their own perceptions of effort.[13] This principle has been demonstrated during steady-state exercise[13] and high-intensity interval cycling[31] where the athletes' reported RPE correlated well to average heart rate[13] and acute changes in heart rate.[31] However, poor correlations have also been found between heart rate and RPE responses during short-duration, high-intensity soccer drills[32] and during step dance sessions.[33] A meta-analysis of the literature concluded that although the Borg scale has been shown to be a valid measure of exercise intensity, the validity coefficients between the Borg 6–20 RPE scale and physiological criterion variables are not as high as previously thought.[34] For example, the weighted mean validity coefficients were 0.62 for heart rate, 0.57 for blood lactate, 0.64 for $\%\dot{V}O_{2max}$, 0.63 for $\dot{V}O_2$, 0.61 for ventilation and 0.72 for respiration rate. Thus, further research is required to ascertain the physiological mechanisms behind our cognitive perception of effort, which may clarify exactly what RPE represents.

1.2.5 Critical Power

Critical power is a theoretical concept that represents an estimation of the maximal power output that can be maintained at physiological steady state without fatigue.[35,36] The identification of such a marker would be useful for the prescription of training or the assessment of training response following an intervention.[37] However, if critical power has physiological meaning, then a steady-state physiological response during exercise at critical power would be required.[37] This hypothesis has been investigated. Brickley et al.[37] found that the work rate when exercising at critical power was approximately $80\%\dot{V}O_{2max}$ and that a physiological steady state was not reached, with oxygen uptake, blood lactate concentration and heart rate increasing over time. There was also considerable interindividual variability in the time that exercise could be sustained.[37] For subjects cycling at 20 W above the maximal lactate steady state (close to their critical power), Pringle and Jones[36] found that blood lactate concentration, $\dot{V}O_2$ and $\dot{V}_E$ increased significantly over time. Although critical power was significantly greater than the maximal lactate steady state, the two variables were strongly correlated.[36] Brickley et al.[37] concluded that critical power does not represent a sustainable steady-state intensity and that a more appropriate definition for critical power would be "the highest, non-steady-state intensity that can be maintained for a period in excess of 20 min, but generally no longer than 40 min".[37]

Dekerle et al.[35] found that critical power, calculated for a range of exhaustion times with subjects exercising at self-selected cadence on a cycle ergometer, was significantly higher than maximal lactate steady state, suggesting that these two variables represent different physiological phenomena. The authors concluded that the physiological significance of the intensity at critical power remains unknown and that further research is required to define the accurate physiological meaning of critical power.[35] Vanhatalo et al.[38] investigated whether critical power may be an indicator of the heavy/severe domain of exercise intensity. They found that critical power was not significantly different from power output

towards the end of a 3-minute all-out cycling test. As such, the authors suggest that there may be potential for the use of this test in the determination of critical power in place of the conventional protocol of multiple exhaustive exercise tests.[38]

1.3 Direct Observation

Direct observation is usually carried out by a coach during the training session and may record training components such as exercise mode, duration and absolute/relative intensity.[6] Speed, for example, may be a useful measure of intensity in swimming or on a flat, measured, indoor running track. However, in other modes such as cycling, skiing and cross-country running, the influence of factors such as terrain, environmental conditions and equipment may alter the accuracy with which speed reflects intensity.[39] Direct observation may also include subjective measures such as the coach's perception of whether or not an athlete is overtraining.[6] Perceptions by coaches and athletes of the same training have been studied by Foster et al.[40] They showed significant differences between the training that the coaches prescribed and the training the athletes actually did. This disassociation may have a significant impact on the effectiveness of the training the coach prescribes. The extent to which training can be quantified based on direct observations may therefore also be limited. Because this method requires the presence of an observer at every training session, which may be impractical or impossible, the amount of data able to be collected in order to monitor training accurately may be inadequate.[6]

New technology using a global positioning system (GPS) offers innovative ways to track distance covered and speed during training.[41-43] The accuracy of these techniques has improved so that for distance mean errors of 0.04–0.7% have been found, and for position mean errors of 1.94–2.13 m have been found.[44] Schutz and Herren[45] found that the accuracy of speed prediction had a standard deviation of 0.08 km/h for walking and 0.11 km/h for running, yielding coefficients of variation of 1.38% and 0.82%, respectively.[45] Studies have shown that GPS can be used to quantify training load in horses.[46,47] Although to our knowledge there are no published studies using this technology to quantify training load in humans, there does appear to be potential for its use under certain circumstances.

1.4 Indices of Training Stress

1.4.1 Training Impulse (TRIMP)

Banister et al.[48] proposed a method of quantifying a training session into a unit 'dose' of physical effort. They suggested that a person's heart rate response to exercise, along with the exercise duration, collectively called a training impulse (TRIMP), may be a plausible measure of physical effort, as it is based on the extent to which exercise raises heart rate between resting and maximal levels.[48,49] A TRIMP is calculated using training duration, maximal heart rate, resting heart rate and average heart rate during the exercise session (equation 3).

$$\text{TRIMP}(\text{w(t)}) = \text{duration of training (min)} \times \Delta\text{HR ratio} \times \text{Y}$$
$$\text{where } \Delta\text{HR ratio} = \frac{\text{HR}_{\text{ex}} - \text{HR}_{\text{rest}}}{\text{HR}_{\text{max}} - \text{HR}_{\text{rest}}} \quad \text{(Eq. 3)}$$

where $Y = 0.64e^{1.92x}$ for males, $Y = 0.86e^{1.67x}$ for females, $e = 2.712$ and $x = \Delta HR$ ratio.

Y is a weighting factor that emphasizes high-intensity exercise and is also applied to the equation to avoid giving disproportionate importance to long-duration, low-intensity exercise compared with intense, short-duration activity.[48] The Y factor is based on the lactate profiles of trained men and women relative to increases in exercise intensity. The ability to quantify and reduce training to a single figure/factor, as is possible with this equation, is appealing in terms of its practical application. However, the use of this method of quantification is limited by the necessity to use heart rate monitors throughout training. It is also understood to require steady-state heart rate measurements, thus limiting the accuracy with which exercise of an interval nature can be quantified. Busso et al.[50] simplified the TRIMP equation by multiplying the average fraction of maximum aerobic power output during exercise to the session duration, thereby

limiting the training stimulus to external loading. A further practical limitation of TRIMP as a measure of training load is the inability to quantify non-aerobic modes of exercise such as resistance training. This is because heart rate increases disproportionately during resistance exercise, and the heart rate responses required for the calculation of TRIMP are not elicited. The equation of Busso et al.[50] was thus adapted to be used in weightlifting by replacing heart rate reserve with %1RM and duration with number of lifts. However, alternative attempts to resolve this limitation resulted in the inclusion of RPE in the quantification of exercise intensity.[51-54]

1.4.2 Session RPE

In an attempt to simplify the quantification of training load, Foster et al.[55] introduced the use of session RPE instead of using heart rate data or having to measure the intensity or type of exercise being performed. The session RPE is a rating of the overall difficulty of the exercise bout obtained 30 minutes after the completion of the exercise. Session load is calculated by multiplying session RPE by session duration of aerobic exercise (in minutes).[55]

Foster et al.[56] compared the session RPE method with the summated heart rate zone score[57] (described below) during aerobic exercise and found that the pattern of differences between the two methods was very consistent. They proposed that session RPE was a valid and reliable measure of exercise intensity.[58] However, no correlation coefficients were provided in Foster et al.,[56] and although individual correlations between the two methods ranged between $r = 0.75$ and $r = 0.90$ in Foster[59] statistical methods were not explained. Impellizzeri et al.[60] found that individual correlations between the session RPE method and Banister's TRIMP method ranged between $r = 0.50$ and $r = 0.77$; individual correlations between the session RPE method and the summated heart rate zone method ranged from $r = 0.54$ to $r = 0.78$, and from $r = 0.61$ to $r = 0.85$ between the session RPE method and the TRIMP methods in soccer players during training and matches. They suggest that the session RPE-based score cannot yet replace the heart rate-based methods as a valid measure of exercise intensity, as only 50% of the variation they measured in heart rate could be explained by the session RPE.[60] A study by Borresen and Lambert[61] found correlations of $r = 0.76$ between TRIMP and session RPE and $r = 0.84$ between summated heart rate zone method and the session RPE method.

The complex interaction of many factors that contribute to the personal perception of physical effort, including hormone concentrations (e.g. catecholamines), substrate concentrations (e.g. glucose, glycogen and lactate), personality traits, ventilation rate, neurotransmitter levels, environmental conditions or psychological states, may limit the use of RPE in accurately quantifying or prescribing exercise intensity.[1] Although using objective physiological measurements such as heart rate may be a more accurate way of calculating training load, the subjective measure of RPE remains useful. Thus, if heart rate monitors are not available, or an easier means of reporting and calculating training load is required, then the RPE method may still give reasonably accurate assessments of aerobic training load.

For resistance exercise, session load is calculated by multiplying session RPE by the number of repetitions performed in resistance exercise.[51-53,55,59,62] The use of session RPE to quantify training load has potential in being a mode- and intensity-independent method that can be used for multiple types of exercise such as high-intensity or non-steady-state exercise such as resistance training, high-intensity interval training or plyometric training.[56] However, there remain limitations to its use in both aerobic and resistance training. RPE is influenced more by resistance load than by volume, so performing more repetitions with a lighter load is perceived as being easier than performing fewer repetitions with a heavier load.[51,62] Sweet et al.[51] and McGuigan et al.[52] found that the RPE varies significantly among different muscle groups used because of differences in muscle mass (and hence metabolic demand), range of motion and the number of joints involved in a movement. The order in which the exercises are performed, the fibre type of the muscle used, the mode of exercise for

which the athlete is trained (i.e. the level of experience the athlete has in resistance training) and the time at which RPE is reported may also affect RPE.[51,62]

1.4.3 Summated Heart Rate Zone Score

The summated heart rate zone score is a modification to the calculation of training impulses that facilitates the quantification of interval training.[57] The accumulated duration (minutes) spent in each of five heart rate zones is calculated (i.e. 50–60%, 60–70%, 70–80%, 80–90% and 90–100% of maximal heart rate) and then multiplied by a multiplier factor for each zone (50–60% = 1, 60–70% = 2, 70–80% = 3, 80–90% = 4 and 90–100% = 5). The results are then summated.[57]

Borresen and Lambert[61] attempted to identify characteristics that may explain the variance not accounted for in the relationship between the objective (TRIMP and summated heart rate zone score) and subjective (session RPE) methods of quantifying training load. The results suggested that for athletes who spent a greater percentage of their training time doing high-intensity exercise, the objective (heart rate-based) equations may overestimate training load compared with the subjective (RPE-based) method. Alternatively, the session RPE method may underestimate training load compared with the objective methods for these athletes. Conversely, in athletes who spent proportionally more of their training time doing low-intensity exercise, the heart rate-based methods may underestimate training load when compared with the training load calculated using the session RPE method, or the session-RPE method may overestimate training load.[61] The authors suggest that it may be the weighting system used in this equation that limits its accuracy. Because a weighting factor is applied to each zone comprising a range of heart rates, the lowest heart rate and the highest heart rate in each zone will be weighted the same, despite a difference in the physiological load. Under certain circumstances a change in heart rate of only 1 beat/min will change the weighting factor of the zone, thereby increasing or decreasing the calculated load disproportionately.[61] After an extensive review of the literature, there appears to be no evidence that this method of quantification has been validated. The summated heart rate zone equation may therefore have been derived theoretically and not through experimentation, raising the question of the legitimacy of validating the session RPE method against this heart rate-based method.

1.4.4 Lucia's TRIMP

Recently, a modified version of the summated heart rate zone equation has been used by Earnest et al.[63] and Lucia et al.[64] and referred to as 'Lucia's TRIMP' by Impellizzeri et al.[60] In this method the duration spent in each of three heart rate zones (zone 1: below the ventilatory threshold; zone 2: between the ventilatory threshold and the respiratory compensation point; and zone 3: above the respiratory compensation point) is multiplied by a coefficient (k) relative to each zone ($k = 1$ for zone 1, $k = 2$ for zone 2, and $k = 3$ for zone 3) and the adjusted scores are then summated. The original source of this equation, however, was not referenced in these studies. This method of quantifying training load shares the same limitation as the summated heart rate zone method, in so far as the weighting of each zone increases in a linear fashion, which does not reflect physiological responses to exercise above the anaerobic threshold.[65] Anaerobic threshold may vary between individuals with equal aerobic power, and therefore the metabolic stress experienced by individuals may be different even when exercising at the same percentage of maximal heart rate.[65]

The TRIMP, session RPE and summated heart rate zone methods are becoming popular methods of quantifying training load. The accuracy of these methods in assessing internal training stress is important if training is to be prescribed based on these results in order to produce more predictable performances. However, it is not only the quantification of training load but knowledge of the physiological mechanisms involved in the exercise response and the ability to measure and quantify training-induced adaptations that will allow more accurate prescription of training and prediction of performance.

2. Modelling the Relationship between Training and Performance

At the biological level, exercise training may be interpreted as a stimulus that causes a disturbance in homeostasis, which is restored during recovery after the training session. After several training sessions, the efficiency with which the physiological systems underlying homeostatic control function is altered, so that subsequent exercise at the same intensity may cause less disturbance to homeostasis. To produce optimal adaptations, training load and recovery must be balanced so that the athlete's physiological systems are sufficiently stimulated to adapt and yet recovery is not impaired.[59,66-68] To this end, the dose-response effect of training impulses on subsequent performances has been investigated.

Foster et al.[55] presented quantified observations of the performance response of competitive athletes to changes in training load but found no significant correlations between the improvements in time trial performance and training time, duration of high-intensity training, training intensity (reflected in RPE ratings) or training load (calculated as session RPE × duration). These findings further emphasize the complex relationship between a number of training variables that may contribute to training load, the body's adaptive response and subsequent performance. Models of the relationship between training and performance have been proposed that consider the athlete as a system in which the training load is the input and performance the system output. The systems models are attractive in their potential to allow more accurate prediction of performance at specific times, or conversely to enable the design of optimal training programmes towards a specific performance goal.[49,69,70]

2.1 Fitness and Fatigue

Banister et al.[48] proposed an equation to assess the training effect (dose) on performance (response) in an attempt to establish a quantifiable relationship. They suggested that, in its simplest form, performance could be defined by two components, a 'fitness impulse' and 'fatigue impulse', and that at any time their difference (fitness – fatigue) can predict an athlete's performance[48,49] (equation 4):

$$\text{Predicted performance} = \text{Fitness} - \text{Fatigue}$$
$$a(t) = k_1 w(t) e^{-t/\tau_1} - k_2 w(t) e^{-t/\tau_2} \quad \text{(Eq. 4)}$$

where k_1 and k_2 are weighting factors (initially $k_1 = 1$ for fitness and $k_2 = 2$ for fatigue) such that the fitness impulse ($k_1 w[t]$) and the fatigue impulse ($k_2 w[t]$) can be calculated by multiplying the training impulse ($w[t]$) by the appropriate weighting factor (k_1 or k_2).

The equation thus comprises two functions in which one represents a positive influence on performance and the other represents a negative influence on performance. Between training sessions the fitness and fatigue variables decline exponentially but at different rates. The contribution of a training impulse to fatigue is proportionally larger than to fitness. However, the decay time constant of fitness is longer. Banister et al.[48] suggested that the fitness decay time constant (τ_1) may be estimated initially as 45 days and the fatigue decay time constant (τ_2) as 15 days. These values, as with the initial values for the weighting factors ($k_1 = 1$ and $k_2 = 2$), are only estimates that allow for an approximation (prediction) to be made of future performance. Data from real performances are then collected and compared with the approximated (predicted) performance, and the decay time constants and weighting factor constants adjusted if discrepancies occur between the predicted and real performance.[48,49]

Busso et al.[70] subsequently tested the accuracy of a simplified form of the above model, comprising only the fitness impulse [$a(t) = w(t) k_1 e^{-t/\tau_1}$]. They found that it produced a similar fit of estimated and real performances, accounting for 61–87% of the total variation in estimated and actual performances. However, they pointed out that the fatigue effect may have been underestimated because of the low-intensity endurance training the subjects underwent, and as such the fatigue effect did not contribute substantially to changes in performance. They suggested that

future studies should include more strenuous and varied training programmes. The researchers further acknowledged the low precision of the performance measures, with the standard error of the estimated and real performances ranging from 3.6 to 5.9 performance units (estimated) and 97 to 152 performance units (real). They ascribed this low precision to external factors such as daily stress,[70] which cannot be controlled but needs to be recognized as an integral part of performance.

2.2 Physiological Correlates of Fitness and Fatigue

Wood et al.[71] explored possible physiological and psychological correlates of the positive and negative components of the Banister model in an attempt to validate the parameters with physiological markers. They found that running speed at ventilatory threshold and running economy correlated with the fitness parameter ($r = 0.94$ and $r = -0.61$, respectively), whereas a fatigue subset of the Profile of Mood States (POMS) questionnaire correlated moderately ($r = 0.75$) with the fatigue parameter of the equation. The authors suggested that the fatigue subset of POMS might reflect a more global fatigue (comprising various stressors of occupation, lifestyle and illness, etc.), whereas the fatigue component of the equation may represent only exercise-induced fatigue. The validity of the fatigue component will thus remain unclear until an accurate measure/marker of exercise-induced fatigue is found. Either that or the fatigue component of the performance equation does not accurately represent exercise-induced fatigue.[71] Lambert and Borresen[72] reviewed several methods being used to monitor fatigue, including the evaluation of recovery and the assessment of muscle soreness after exercise training. In addition, methods of quantifying accumulated (chronic) fatigue were reviewed, including the Daily Analysis of Life Demands for Athletes test, POMS questionnaire, and heart rate measurements of variability, recovery and resting heart rate.

Many other physiological adaptations that occur in response to prolonged exercise training have been investigated as possible markers to directly measure and monitor fitness, fatigue, overtraining and recovery. Jones and Carter,[73] in their review of the effects of endurance training, identify four key parameters of aerobic fitness, namely $\dot{V}O_{2max}$, exercise economy, lactate and ventilatory thresholds, and critical power. They suggest that an improvement in any one or more of these parameters will produce an improvement in performance. However, it must be acknowledged that many other factors, such as environmental conditions, race tactics and psychological factors, may also influence the outcome of a competitive performance.[73] Submaximal blood lactate concentration has also been proposed as a means with which to monitor changes in endurance fitness, because blood lactate concentration decreases at the same absolute and relative intensity after endurance training.[24] The absolute work rate at which the onset of blood lactate accumulation occurs also increases after 6 weeks of training.[74] Pyne et al.[23] found a direct relationship between improvements in lactate parameters and maximal 200 m test time in swimmers after a 20-week training period. However, lactate parameters were unrelated to international competition performance. Because numerous factors besides training have an effect on blood lactate concentration, and as a result of the necessity for standardized testing conditions, the usefulness and accuracy with which lactate profiling can be used to monitor training adaptations remains questionable.[23]

The free circulating testosterone : cortisol ratio has been proposed as an indicator of physiological anabolic/catabolic balance.[75] A low free testosterone : cortisol ratio (<30%) has been suggested as a marker of a catabolic (overtrained or over-reached) state.[2,75] The circulating testosterone : cortisol ratio has also been proposed as a predictor of performance. However, consensus has not yet been reached on how testosterone and cortisol concentrations change in response to training and how this relates to performance.[76] It has been proposed that the measurement of serum iron, serum ferritin and transferrin may be used to identify the inflammatory response to muscle damage, as well as the state of acute and chronic recovery.[77] Serum iron and ferritin

concentrations have been found to be reduced in chronically exercising individuals, particularly those training at higher intensities.[78-80] It has been suggested that such decreases may have a negative effect on performance.[78,79]

Hawley and Stepto[81] presented a theoretical model of training-induced adaptations in skeletal muscle that are likely to influence performance in elite cyclists. The model includes changes in muscle morphology such as increases in neural recruitment, capillary density, enzyme concentration and activity, and type I fibre content.[81] The decrease in muscle glycogen utilization and increase in intramuscular triglyceride oxidation[81-84] during prolonged submaximal exercise is also included. The model also considers acid-base status in terms of an increased lactate threshold and transport capacity. However, Hawley and Stepto[81] conclude that knowledge of the effect of physiological adaptations on performance is limited. Despite years of research, consensus has not yet been reached concerning the effects of training and overtraining on each of the many physiological 'markers' that have been investigated. As such, no single measure has been identified that can accurately assess how an athlete is responding to training. The correlation between training and the observed changes in these physiological variables is highly personal and dependent on individual tolerance of an exercise load, which may be a culmination of many internal and external factors.

2.3 Influence Curves

Fitz-Clarke et al.[69] proposed the use of influence curves to show conceptually how each consecutive day's training affects a subsequent performance. The influence curve is a line representing the effect of a training impulse imposed at any time on performance at a specific future time. Performance on a specific day may be considered as a summation of the contributions of each day's training impulse prior to the day of competition and decayed over the time between the training impulse and competition day. Each training impulse adds a contribution to performance according to its initial magnitude. The impulse response encompasses both the negative and positive influences of each day's training from the start of a programme until competition day. The influence curve thus allows for the identification of the number of days before competition, at which time training load needs to be reduced because training after that day will contribute more to the fatigue impulse than to the fitness impulse. The influence curve can also identify the number of days prior to competition when training is most beneficial to performance on competition day. This model therefore has the potential to design an optimal training programme able to produce a specific performance at a particular time. Influence curves may therefore be useful when giving training advice for one event. However, analysis of training and performance for several competitive events during a season, as is common for elite athletes, is more complex. Influence curves show that the prescription of optimal training for each event becomes a challenge, as the ideal training and rest periods for one performance will impact suboptimally on subsequent performances.[69]

2.4 Recursive Least Squares Algorithm

In the equation of Banister et al.[48] the model parameters τ_1, τ_2, k_1 and k_2, which are initially estimates that are adjusted to suit the individual (after fitting the equation's predicted performance to a real performance), are subsequently kept constant (i.e. time invariant) for the duration of the study period.[48,49] Busso et al.[85] investigated using a recursive least squares algorithm incorporating parameters that are free to vary over time to more accurately illustrate changes in performance after training. They suggested that each training response may be influenced by previous training bouts and that the day-to-day variation in the model parameters may provide important information on the cumulative effects of training.[85,86] The relationship between predicted and real performances was better using a time-varying model than the time-invariant model, with coefficients of variation for the former being 0.875 and 0.879 for the two subjects tested, compared with 0.682 and 0.666 for the

time-invariant model.[85] However, because the parameters in the time-varying model are estimated at any given time from the previous and present data, this type of model would be limited in its ability to predict performance in response to future training, unless the parameters themselves change in a predictable way.[87]

Using their recursive least squares algorithm, Busso et al.[86] later studied the effect of an increase in training frequency on exercise-induced fatigue and found that the time needed to regain a previous performance level increased as training frequency increased. The positive effect of a given training load on performance also decreased when training frequency was increased. Therefore, reducing recovery time between training bouts resulted in an increase in accumulated fatigue. Subsequently, they proposed a model that would account for greater fatigue resulting from increased training frequency.[88] The model comprises a fatigue component that varies over time and with the intensity of past training bouts. It offers the possibility to more accurately describe the dose-response relationship between cumulative training loads and training response, and thus to study training periodization.[88]

2.5 Threshold Saturation

Hellard et al.[89] proposed a model that includes a saturation threshold in which the impact of training on performance is nonlinear and has an upper limit. This method introduces the possibility of identifying an upper limit to the training stimulus of an athlete so that training intensity and duration can be kept below this threshold in order to optimize physiological adaptations. Training maintained above this level may induce excessive chronic fatigue and lead to a decline in performance.[89] Hellard et al.[89] studied Olympic swimmers over 4 years and found that the modified model improved the fit between training and performance compared with the Banister model. However, the training variables still explained only 30% of the variation in performance. They suggested a number of reasons for this discrepancy: (i) an individual's response to the same exercise load may differ between seasons; (ii) there may be indirect effects of training, for example adaptations to one mode of exercise may influence/change the way in which the body responds to another exercise mode; (iii) variations in technique; or (iv) the fact that swimmers react differently to the same training stimulus.[89] The model parameters were also assumed to remain constant throughout the duration of the study, whereas regular adjustment of these parameters may have improved the fit between predicted and real performances.

2.6 Limitations to Modelling the Training-Performance Relationship

The proposed mathematical models attempt to describe the effect of training bouts on performance as a dose-response relationship comprising fitness and fatigue impulses. However, although attractive in concept, the accuracy of these theoretical models has proven to be poor, as is evident in weak correlations between the predicted and measured performances in response to training.[70,85] Considering that the smallest changes in performance could have a significant impact on the outcome of elite competition, it becomes vitally important that models predict performance with only the slightest margin of error. These disparities may be the result of the fact that the values for the fitness impulse, the fatigue impulse and their decay rates are initially estimates and that the adjustment of these values for each individual requires regular criterion performance measures comparable with competition conditions. Not only is this difficult and impractical, but it allows only retrospective adjustments to be made to the equation rather than the ability to prospectively prescribe training to achieve a desired performance. More importantly, the equations lack an accurate measure of individualism in how each athlete responds to training, which may contribute substantially to the inaccuracy of the model. Thus, although much has been reported on the correlations between endurance training and physiological adaptations, specific markers that facilitate the quantification of a dose-response relationship between

training, adaptation and performance remain to be identified.

3. Variability in the Physiological Response to Training

An important limiting factor in the establishment of a quantifiable relationship between training, physiological adaptations and performance is that the variability in the way individuals react to training is not being accounted for. The inter-subject variance in training-induced adaptations may be the result of several factors such as: age; sex; training history; psychological factors; initial training status; mode, duration, intensity and frequency of training;[90] recovery potential; exercise capacity; non-training stress factors; stress tolerance; [73,91] and genetics.[92] Jones and Carter,[73] in their review of the effects of endurance training on the parameters of aerobic fitness, note that the magnitude of change in $\dot{V}O_{2max}$ may be governed by many of the above factors, and that exercise economy differs significantly between individuals and may be influenced by the velocities/power outputs at which they habitually train. Bell et al.[93] found that concurrent strength and endurance training resulted in training adaptations that differed from those that occurred after either strength or endurance training alone, emphasizing the importance of considering exercise mode when assessing training-induced adaptations.

Avalos et al.[94] found that they could separate swimmers into groups that reacted well to either a long-term, mid-term or short-term training period, emphasizing the need to individualize the distribution of training loads throughout a season in order to facilitate optimal adaptation in each athlete. Skinner et al.[22] found that individual changes in power output and $\dot{V}O_{2max}$ in response to 20 weeks of cycling training varied significantly among people who trained at heart rates associated with the same $\%\dot{V}O_{2max}$. Al-Ani et al.[95] reported an increase in heart rate variability, measured as the difference between maximum and minimum R-R interval in a respiratory cycle, in nine of 11 people after 6 weeks of endurance training. However, with the heart rate variability of the two other subjects having had the opposite response after training, the individuality of the training response is again highlighted.

Bouchard and Rankinen[96] and Rice et al.[97] found that initial training status and familial factors contributed significantly to the inter-individual variation in 'trainability' of individuals (measured as changes in heart rate, blood pressure and $\dot{V}O_{2max}$). Wilmore et al.[98] found that sex, race and age contributed to this heterogeneity. Conversely, Skinner et al.[99] found that age, sex, race and initial fitness had little influence on how $\dot{V}O_{2max}$ changed after a standardized endurance training programme. The authors showed that there were low, medium and high responders in both sexes, at all ages and at all fitness levels, and suggested that genetics may be responsible for the wide variation in individual responses. This individuality in training response thus requires further investigation.

4. Variability in the Relationship between Training Adaptations and Performance

Genetic traits may contribute substantially not only to the way in which athletes adapt to training but also to the observed heterogeneity in performance ability between athletes.[100,101] This variance may depend on different numbers and types of genes being activated in response to different intensities, durations and frequencies of exercise. Further variation may occur during DNA sequencing, gene transcription and protein translation. The potential for improvement in performance or optimal adaptation may also be influenced by a genetic predisposition for performance in a specific mode of exercise.[92] For example, the type of predominant muscle fibre in a muscle may predispose a person to better performance in either an endurance- or resistance-type sport.[92,102] Recently, interest in the identification of specific genes that may be associated with performance has increased.[101] One potential gene is *ACTN3*, which encodes the protein α-actinin-3 that forms part of the sarcomeres of fast twitch muscle fibres. There is evidence that the 577R allele and 577RR genotype may be associated with sprinting and/or power performance, whereas no association has been found

between the R577X polymorphism and endurance performance.[101,103,104]

In a study examining the effect of training on swimming performance, Mujika et al.[105] noted significant variance among elite swimmers in a number of systems model variables, including the time constants of decay of the fitness and fatigue impulses, and the fitness and fatigue multiplying factors, amongst others. The model explained between 45% and 85% of the variations in real performance. This emphasizes a limitation in the design of the performance models, which do not take into account the fact that individuals respond and adapt differently to training. However, a limitation in the study was the assumption that the model parameters were constant throughout the study (44 weeks), which ignores the possibility that adaptations to training may alter how an athlete responds to exercise.[105] Indeed, Banister et al.[48] suggested that the period within which the model parameters may be assumed to be constant is 60–90 days, after which the parameters need to be reset by comparing predicted performance to real performance.

Hellard et al.[89] reported that training variables explained only 30% of the variation in the performance of Olympic swimmers studied for 4 years. The authors suggest that performance may be affected by the phase of training in which the athlete is, as there were differing short-, intermediate- and long-term effects on performance. In addition, swimmers react differently to the same training load (interindividual differences) and between consecutive seasons (intraindividual differences). Bagger et al.[16] describe the magnitude of individual variation in a number of factors often used to assess training adaptations, because this is necessary to distinguish whether a change in the variable is the result of training or of random biological fluctuation. They found that performance and physiological measurements such as HR_{max}, HR_{10km}, RER_{submax}, HR_{submax}, and $\dot{V}O_{2max}$, amongst others, had the lowest total coefficient of variation (13%), whereas metabolic and hormonal variables had the highest coefficient of variation (37%). The variance as a result of within-subject variation in metabolic and hormonal variables was 53%, compared with only 13% in performance and physiological measurements.[16] Thus, training adaptations are a highly individual phenomenon, and the variation within and between athletes needs to be considered when assessing training-induced changes in performance.

5. Summary and Future Research

There is currently no accurate quantitative means with which to prescribe the pattern, duration and intensity of exercise required to produce specific physiological adaptations. Added to this is the fact that individuals adapt differently to the same exercise stimulus. Banister et al.[48] found that the constants used in the performance models need to be reset after a period of approximately 60–90 days. Mujika et al.[105] suggest that individual chronic training adaptation profiles could be developed by studying individual fatigue and fitness curves in order to better understand an individual's response to a training bout. In the development of the performance prediction model, Banister et al.[48] suggest that ''it may be assumed that all the constants of the model that are used to obtain the fit are exactly those that are peculiar to the individual being modeled''. Thus, studying individual fitness and fatigue curves may allow the quantification of individual response and adaptation to training. The accuracy of the mathematical models in predicting performance may also be improved when the physiological meaning of the modelled responses is better defined.[70,87] As such, the search continues to find easily measurable physiological markers of 'fitness' and 'fatigue' to improve the accuracy with which performance can be predicted.[87] Despite years of research, no single physiological marker has been identified that can quantify the fitness and fatigue responses to exercise or predict performance with accuracy. The correlation between training and the observed changes in these physiological variables is highly personal and depends on many factors that influence an individual's tolerance of an exercise load. Thus, more emphasis needs to be directed towards the measurement of markers that reflect an individual's global capacity to respond or adapt to training,

rather than an absolute measure of the changes in physiological variables in response to exercise.

Acknowledgements

The research undertaken in this study is funded in part by the University of Cape Town, Discovery Health, National Research Foundation, Ernst & Ethel Eriksen Foundation and Deutscher Akademischer Austausch Dienst. The authors have no conflicts of interest that are directly related to the content of this review.

References

1. Williams JG, Eston RG. Determination of the intensity dimension in vigorous exercise programmes with particular reference to the use of the rating of perceived exertion. Sports Med 1989; 8 (3): 177-89
2. Budgett R. Fatigue and underperformance in athletes: the overtraining syndrome. Br J Sports Med 1998; 32 (2): 107-10
3. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. Sports Med 2004; 34 (14): 967-81
4. Urhausen A, Kindermann W. Diagnosis of overtraining: what tools do we have? Sports Med 2002; 32 (2): 95-102
5. Budgett R, Newsholme E, Lehmann M, et al. Redefining the overtraining syndrome as the unexplained underperformance syndrome. Br J Sports Med 2000 Feb 1; 34 (1): 67-8
6. Hopkins WG. Quantification of training in competitive sports: methods and applications. Sports Med 1991; 12 (3): 161-83
7. Lambert MI, Bryer L, Hampson DB, et al. Accelerated decline in running performance in a masters runner with a history of a large volume of training and racing. J Aging Phys Activ 2002; 10: 314-21
8. Borresen J, Lambert MI. Validity of self-reported training duration. Int J Sports Sci Coach 2006; 1 (4): 353-9
9. Shephard RJ. Limits to the measurement of habitual physical activity by questionnaires. Br J Sports Med 2003; 37 (3): 197-206
10. Buchheit M, Gindre C. Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. Am J Physiol Heart Circ Physiol 2006; 291 (1): H451-8
11. Achten J, Jeukendrup AE. Heart rate monitoring: applications and limitations. Sports Med 2003; 33 (7): 517-38
12. Arts FJ, Kuipers H. The relation between power output, oxygen uptake and heart rate in male athletes. Int J Sports Med 1994; 15 (5): 228-31
13. Robinson DM, Robinson SM, Hume PA, et al. Training intensity of elite male distance runners. Med Sci Sports Exerc 1991; 23 (9): 1078-82
14. Karvonen J, Vuorimaa T. Heart rate and exercise intensity during sports activities: practical application. Sports Med 1988; 5 (5): 303-11
15. Lambert MI, Mbamba ZH, St Clair Gibson A. Heart rate during training and competition for long-distance running. J Sports Sci 1998; 16: S85-90
16. Bagger M, Petersen PH, Pedersen PK. Biological variation in variables associated with exercise training. Int J Sports Med 2003; 24 (6): 433-40
17. Xu F, Rhodes EC. Oxygen uptake kinetics during exercise. Sports Med 1999; 27 (5): 313-27
18. Kohrt WM, Morgan DW, Bates B, et al. Physiological responses of triathletes to maximal swimming, cycling, and running. Med Sci Sports Exerc 1987; 19 (1): 51-5
19. Swain DP, Leutholtz BC, King ME, et al. Relationship between % heart rate reserve and % $\dot{V}O_2$ reserve in treadmill exercise. Med Sci Sports Exerc 1998; 30 (2): 318-21
20. Swain DP, Leutholtz BC. Heart rate reserve is equivalent to %$\dot{V}O_2$ reserve, not to % $\dot{V}O_2$max. Med Sci Sports Exerc 1997; 29 (3): 410-4
21. Baldwin J, Snow RJ, Febbraio MA. Effect of training status and relative exercise intensity on physiological responses in men. Med Sci Sports Exerc 2000; 32 (9): 1648-54
22. Skinner JS, Wilmore KM, Krasnoff JB, et al. Adaptation to a standardized training program and changes in fitness in a large, heterogeneous population: the HERITAGE Family Study. Med Sci Sports Exerc 2000; 32 (1): 157-61
23. Pyne DB, Lee H, Swanwick KM. Monitoring the lactate threshold in world-ranked swimmers. Med Sci Sports Exerc 2001; 33 (2): 291-7
24. Jacobs I. Blood lactate: implications for training and sports performance. Sports Med 1986; 3 (1): 10-25
25. Weltman A, Seip RL, Snead D, et al. Exercise training at and above the lactate threshold in previously untrained women. Int J Sports Med 1992; 13 (3): 257-63
26. Stegmann H, Kindermann W, Schnabel A. Lactate kinetics and individual anaerobic threshold. Int J Sports Med 1981; 2 (3): 160-5
27. Swart J, Jennings CL. Use of blood lactate concentration as a marker of training status. S Afr J Sports Med 2004; 16: 3-7
28. Jeukendrup AE, Hesselink MK. Overtraining: what do lactate curves tell us? J Sports Med 1994; 28 (4): 239-40
29. Urhausen A, Gabriel HH, Weiler B, et al. Ergometric and psychological findings during overtraining: a long-term follow-up study in endurance athletes. Int J Sports Med 1998; 19 (2): 114-20
30. Kirwan JP, Costill DL, Flynn MG, et al. Physiological responses to successive days of intense training in competitive swimmers. Med Sci Sports Exerc 1988; 20 (3): 255-9
31. Green JM, McLester JR, Crews TR, et al. RPE association with lactate and heart rate during high-intensity interval cycling. Med Sci Sports Exerc 2006; 38 (1): 167-72
32. Little T, Williams AG. Measures of exercise intensity during soccer training drills with professional soccer players. J Strength Cond Res 2007; 21 (2): 367-71
33. Ozkan A, Kin-Isler A. The reliability and validity of regulating exercise intensity by ratings of perceived exertion in step dance sessions. J Strength Cond Res 2007; 21 (1): 296-300
34. Chen MJ, Fan X, Moe ST. Criterion-related validity of the Borg ratings of perceived exertion scale in healthy

individuals: a meta-analysis. J Sports Sci 2002; 20 (11): 873-99

35. Dekerle J, Baron B, Dupont L, et al. Maximal lactate steady state, respiratory compensation threshold and critical power. Eur J Appl Physiol 2003; 89 (3): 281-8
36. Pringle J, Jones A. Maximal lactate steady state, critical power and EMG during cycling. Eur J Appl Physiol 2002; 88 (3): 214-26
37. Brickley G, Doust J, Williams CA. Physiological responses during exercise to exhaustion at critical power. Eur J Appl Physiol 2002; 88 (1): 146-51
38. Vanhatalo A, Doust JH, Burnley M. Determination of critical power using a 3-min all-out cycling test. Med Sci Sports Exerc 2007; 39 (3): 548-55
39. Jeukendrup A, van Diemen A. Heart rate monitoring during training and competition in cyclists. J Sports Sci 1998; 16: S91-9
40. Foster C, Heimann KM, Esten PL, et al. Differences in perceptions of training by coaches and athletes. S Afr J Sports Med 2001; 8 (2): 3-7
41. Larsson P. Global positioning system and sport-specific testing. Sports Med 2003; 33 (15): 1093-101
42. Townshend AD, Worringham CJ, Stewart IB. Assessment of speed and position during human locomotion using nondifferential GPS. Med Sci Sports Exerc 2008; 40 (1): 124-32
43. Rodriguez DA, Brown AL, Troped PJ. Portable global positioning units to complement accelerometry-based physical activity monitors. Med Sci Sports Exerc 2005; 37 (11 Suppl.): S572-81
44. Larsson P, Henriksson-Larsen K. The use of dGPS and simultaneous metabolic measurements during orienteering. Med Sci Sports Exerc 2001; 33 (11): 1919-24
45. Schutz Y, Herren R. Assessment of speed of human locomotion using a differential satellite global positioning system. Med Sci Sports Exerc 2000; 32 (3): 642-6
46. Vermeulen AD, Evans DL. Measurements of fitness in thoroughbred racehorses using field studies of heart rate and velocity with a global positioning system. Equine Vet J Suppl 2006; (36): 113-7
47. Kingston JK, Soppet GM, Rogers CW, et al. Use of a global positioning and heart rate monitoring system to assess training load in a group of thoroughbred racehorses. Equine Vet J Suppl 2006; (36): 106-9
48. Banister EW, MacDougall JD, Wenger HA, et al. Modeling elite athletic performance: physiological testing of the high-performance athlete. Campaign (IL): Human Kinetics Books; 1991: 403-25
49. Morton RH, Fitz-Clarke JR, Banister EW. Modeling human performance in running. J Appl Physiol 1990; 69 (3): 1171-7
50. Busso T, Häkkinen 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): 48-54
51. Sweet TW, Foster C, McGuigan MR, et al. Quantitation of resistance training using the session rating of perceived exertion method. J Strength Cond Res 2004; 18 (4): 796-802
52. McGuigan MR, Egan AD, Foster C. Salivary cortisol responses and perceived exertion during high intensity and low intensity bouts of resistance exercise. J Sports Sci Med 2004; 3: 8-15
53. McGuigan MR, Foster C. A new approach to monitoring resistance training. Strength Cond J 2004; 26 (6): 42-7
54. Egan AD, Winchester JB, Foster C, et al. Using session RPE to monitor different methods of resistance exercise. J Sports Sci Med 2006; 5: 289-95
55. Foster C, Daines E, Hector L, et al. Athletic performance in relation to training load. Wis Med J 1996; 95 (6): 370-4
56. Foster C, Florhaug JA, Franklin J, et al. A new approach to monitoring exercise training. J Strength Cond Res 2001; 15 (1): 109-15
57. Edwards S. The heart rate monitor book. Sacramento (CA): Fleet Feet Press, 1993
58. Herman L, Foster C, Maher MA, et al. Validity and reliability of the session RPE method for monitoring exercise training intensity. S Afr J Sports Med 2006; 18 (1): 14-7
59. Foster C. Monitoring training in athletes with reference to overtraining syndrome. Med Sci Sports Exerc 1998; 30 (7): 1164-8
60. Impellizzeri FM, Rampinini E, Coutts AJ, et al. Use of RPE-based training load in soccer. Med Sci Sports Exerc 2004; 36 (6): 1042-7
61. Borresen J, Lambert MI. Quantifying training load: a comparison of subjective and objective methods. IJSPP 2008; 3: 16-30
62. Day ML, McGuigan MR, Brice G, et al. Monitoring exercise intensity during resistance training using the session RPE scale. J Strength Cond Res 2004; 18 (2): 353-8
63. Earnest CP, Jurca R, Church TS, et al. Relation between physical exertion and heart rate variability characteristics in professional cyclists during the Tour of Spain. Br J Sports Med 2004; 38 (5): 568-75
64. Lucia A, Hoyos J, Santalla A, et al. Tour de France versus Vuelta a Espana: which is harder? Med Sci Sports Exerc 2003; 35 (5): 872-8
65. Stagno KM, Thatcher R, van Someren KA. A modified TRIMP to quantify the in-season training load of team sport players. J Sports Sci 2007; 25 (6): 629-34
66. Kuipers H, Keizer HA. Overtraining in elite athletes: review and directions for the future. Sports Med 1988; 6 (2): 79-92
67. Kuipers H. Training and overtraining: an introduction. Med Sci Sports Exerc 1998; 30 (7): 1137-9
68. Uusitalo ALT. Overtraining: making a difficult diagnosis and implementing targeted treatment. Phys Sportsmed 2001; 29 (5): 35-50
69. Fitz-Clarke JR, Morton RH, Banister EW. Optimizing athletic performance by influence curves. J Appl Physiol 1991; 71 (3): 1151-8
70. 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
71. Wood RE, Hayter S, Rowbottom D, et al. Applying a mathematical model to training adaptation in a distance runner. Eur J Appl Physiol 2005; 94 (3): 310-6
72. Lambert MI, Borresen J. A theoretical basis of monitoring fatigue: a practical approach for coaches. Int J Sports Sci Coach 2006; 1 (4): 371-88

73. Jones AM, Carter H. The effect of endurance training on parameters of aerobic fitness. Sports Med 2000; 29 (6): 373-86
74. Mayes R, Hardman AE, Williams C. The influence of training on endurance and blood lactate concentration during submaximal exercise. Br J Sports Med 1987; 21 (3): 119-24
75. Urhausen A, Gabriel H, Kindermann W. Blood hormones as markers of training stress and overtraining. Sports Med 1995; 20 (4): 251-76
76. Filaire E, Bernain X, Sagnol M, et al. Preliminary results on mood state, salivary testosterone: cortisol ratio and team performance in a professional soccer team. Eur J Appl Physiol 2001; 86 (2): 179-84
77. Smith DJ, Roberts D. Effects of high volume and/or intense exercise on selected blood chemistry parameters. Clin Biochem 1994; 27 (6): 435-40
78. Angeli A, Minetto M, Dovio A, et al. The overtraining syndrome in athletes: a stress-related disorder. J Endocrinol Invest 2004; 27 (6): 603-12
79. Beard J, Tobin B. Iron status and exercise. Am J Clin Nutr 2000; 72 (2 Suppl.): 594S-7S
80. Wilkinson JG, Martin DT, Adams AA, et al. Iron status in cyclists during high-intensity interval training and recovery. Int J Sports Med 2002; 23 (8): 544-8
81. Hawley JA, Stepto NK. Adaptations to training in endurance cyclists: implications for performance. Sports Med 2001; 31 (7): 511-20
82. Hawley JA. Adaptations of skeletal muscle to prolonged, intense endurance training. Clin Exp Pharmacol Physiol 2002; 29 (3): 218-22
83. Horowitz JF, Klein S. Lipid metabolism during endurance exercise. Am J Clin Nutr 2000; 72 (2 Suppl.): 558S-63S
84. Schmitt B, Fluck M, Decombaz J, et al. Transcriptional adaptations of lipid metabolism in tibialis anterior muscle of endurance-trained athletes. Physiol Genomics 2003; 15 (2): 148-57
85. 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
86. 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
87. Taha T, Thomas SG. Systems modelling of the relationship between training and performance. Sports Med 2003; 33 (14): 1061-73
88. Busso T. Variable dose-response relationship between exercise training and performance. MedSciSports Exerc 2003; 35 (7): 1188-95
89. Hellard P, Avalos M, Millet G, et al. Modeling the residual effects and threshold saturation of training: a case study of Olympic swimmers. J Strength Cond Res 2005; 19 (1): 67-75
90. Tremblay MS, Copeland JL, Van Helder W. Effect of training status and exercise mode on endogenous steroid hormones in men. J Appl Physiol 2004; 96 (2): 531-9
91. Lehmann M, Foster C, Keul J. Overtraining in endurance athletes: a brief review. Med Sci Sports Exerc 1993; 25 (7): 854-62
92. Heck AL, Barroso CS, Callie ME, et al. Gene-nutrition interaction in human performance and exercise response. Nutrition 2004; 20 (7-8): 598-602
93. Bell GJ, Syrotuik D, Martin TP, et al. Effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations in humans. Eur J Appl Physiol 2000; 81 (5): 418-27
94. Avalos M, Hellard P, Chatard JC. Modeling the training-performance relationship using a mixed model in elite swimmers. Med Sci Sports Exerc 2003; 35 (5): 838-46
95. Al Ani M, Munir SM, White M, et al. Changes in R-R variability before and after endurance training measured by power spectral analysis and by the effect of isometric muscle contraction. Eur J Appl Physiol Occup Physiol 1996; 74 (5): 397-403
96. Bouchard C, Rankinen T. Individual differences in response to regular physical activity. Med Sci Sports Exerc 2001; 33 (6 Suppl.): S446-51
97. Rice T, An P, Gagnon J, et al. Heritability of HR and BP response to exercise training in the HERITAGE Family Study. Med Sci Sports Exerc 2002; 34 (6): 972-9
98. Wilmore JH, Stanforth PR, Gagnon J, et al. Heart rate and blood pressure changes with endurance training: the HERITAGE Family Study. Med Sci Sports Exerc 2001; 33 (1): 107-16
99. Skinner JS, Jaskolski A, Jaskolska A, et al. Age, sex, race, initial fitness, and response to training: the HERITAGE Family Study. J Appl Physiol 2001; 90 (5): 1770-6
100. Macarthur DG, North KN. Genes and human elite athletic performance. Hum Genet 2005; 116 (5): 331-9
101. MacArthur DG, North KN. ACTN3: A genetic influence on muscle function and athletic performance. Exerc Sport Sci Rev 2007; 35 (1): 30-4
102. Brooks GA, Fahey TD, White TP, et al. Exercise physiology: human biogenetics and its applications. Columbus (OH): McGraw-Hill Companies, Inc., 2000
103. Yang N, MacAruthur J. ACTN3 genotype is associated with human elite athletic performance. Am J Hum Genet 2003; 73: 627-31
104. Saunders CJ, September AV, Xenophontos SL, et al. No association of the ACTN3 gene R577X polymorphism with endurance performance in Ironman triathlons. Ann Hum Genet 2007; 71 (6): 777-81
105. 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

Correspondence: Prof. *Mike Lambert*, MRC/UCT Research Unit for Exercise Science and Sports Medicine, Sports Science Institute of South Africa, Boundary Road, Newlands 7700, South Africa.
E-mail: mike.lambert@uct.ac.za