RESEARCH ARTICLE **Open Access**

Comparison of models for the physiological estimation of internal mechanical power in cycling

Hedda P Brooks [1, 2]✉, Mark H Andrews[1], Adrian J Gray[3], Mark A Osborne[1, 2]

Abstract

Physiological models for estimating internal mechanical power (IP) generally share a common basis: the sum of IP and the external mechanical power (EP) is equal to the total mechanical power, as represented by net VO_2. While a biomechanical perspective of IP argues against this simple summation, physiological models serve a valuable purpose because they account for the total flow of energy through the system. Despite their common foundation, estimates of IP using various physiological models have not been consistent. Two pre-existing models that use a physiological approach, as well as variations of them and a simple body mass-cadence relationship, were applied to submaximal data from eight well-trained male cyclists. Three incremental cycling tests were performed at cadences of 50-55, 80-85 and 110-115 rev · min^{-1}. Differences in the mean and limits of agreement were used to show that values of IP calculated using two previously described models were not similar at any of the cadences tested. It was also shown that using relevant energy equivalents for converting VO_2 into metabolic power produced smaller values for IP than when using a generic energy equivalent. Differences in values for IP in the published literature, therefore, might not necessarily be caused by differences in participant characteristics, but rather differences in the accuracy of the variables that are input into the IP models.

Keywords: external mechanical power, delta efficiency, metabolic power

✉ **Contact email:** *hedda.brooks@uqconnect.edu.au*
(HP. Brooks)

[1] Queensland Academy of Sport, Australia

[2] School of Human Movement Studies, The University of Queensland, Australia

[3] Exercise & Sports Science, School of Science & Technology, University of New England

Received: 1 March 2013. Accepted:15 May 2013.

Introduction

In cycling, external mechanical power (EP) is the rate that energy flows from the cyclist to the pedals to overcome the forces that oppose forward motion. Internal mechanical power (IP) is the rate of mechanical work done to move the limbs against gravitational and inertial forces. If it were possible to decrease IP, it has been suggested that more metabolic energy would be available to contribute to the performance of EP (Hull et al. 1991). An increase in EP would, in turn, improve gross efficiency (the ratio of EP to total metabolic power). With more energy expended against those external forces resisting motion (i.e., EP), performance ought to be enhanced.

Normally, IP is calculated using either a physiological or biomechanical approach. A number of physiological models have been proposed to estimate IP during cycling (Francescato et al. 1995; Hansen et al. 2004; Martin et al. 2002; Tokui and Hirakoba 2008). Generally, these models are based on the assumption that IP and EP can be summed to equal the total mechanical power (TMP). It is worth noting here that such a simple relationship between IP and EP has been denounced in the biomechanical literature (Aleshinsky 1986; Broker and Gregor 1994; Kautz and Neptune 2002; van Ingen Schenau et al. 1990), where it has been argued that despite separate mechanical destinations (i.e., EP and IP), the metabolic energy is indeed shared. While the biomechanical perspective is not without its own limitations, further discussion is beyond the scope of this investigation. Furthermore, the validity of subtracting the metabolic cost of rest from the total metabolic cost, which is inherent in the physiological models for IP calculation, has also been argued (Chavarren and Calbet 1999; Gaesser and Brooks 1975; Stainbsy et al. 1980). This argument is in view of uncertainties related to consistent metabolic baselines across workloads and cadences (Stainbsy et al. 1980), as well as whether or not the physiological processes at rest can be considered independent of those during exercise (Chavarren and Calbet 1999). Despite these arguments, the physiological models for estimating IP provide useful information about the change in whole-body metabolic cost, inclusive of possible changes in the costs of gastrointestinal and ventilatory activity, body temperature regulation and splanchnic metabolism (Stainbsy et al. 1980).

Despite the common summation of EP and IP, the estimates for IP calculated from various physiological models, over a range of cycling cadences, are not consistent (e.g., ~1.9 W at 60 rev · min^{-1} (Francescato et al. 1995) compared to 15.1 W at 61 rev · min^{-1} (Hansen et al. 2004)). Differences in the values for IP at the same cadence may be explained by differences in participant characteristics and/or the cycling posture that is adopted, as well as differences in the values used for IP calculation. The purpose of this paper, therefore, was to apply two existing physiological models (Francescato et al. 1995; Hansen et al. 2004) for calculating IP, as well as modified versions of these models, to data from the same well-trained participant group, over a range of cadences and workloads. The model by Minetti et al. (2001) was also included in the current analysis because of its simplicity and inclusion in previous physiological IP investigations as it only requires information about cadence and body mass (Hansen et al. 2004; Tokui and Hirakoba 2007). It was expected that there would be more similarity between the estimates for IP in the models that used measured, rather than predicted, variables. In particular, the models that included an estimate of resting VO_2 based on participants' own body mass, rather than a constant value for all participants, would have similar results. Likewise, those models that used oxygen energy equivalents for the respective measured respiratory exchange ratios (RER) would provide similar estimates for IP. Finally, it was expected that the Minetti et al. (2001) model would calculate lower values for IP at all cadences. Such an expectation is due to the biomechanical foundation of this model, which, for example, assumes the body consists of rigid links that do not have the capacity to absorb or generate energy, and where joint powers represent the net power of the muscles surrounding each joint rather than the total power those muscles produced (Zatsiorsky 1994).

Methods

Participants

Eight healthy, Elite A division male road cyclists (mean ± SD: age 31.7 ± 6.6 years, mass 74.7 ± 5.2 kg, height 1.80 ± 0.04 m, VO_{2peak} 65.3 ± 5.1 ml · kg^{-1} · min^{-1}) participated in the study. Participants trained at least 14 hours per week, completing 487 ± 95 km per week, throughout the year. They averaged 9.7 ± 5.0 years of racing experience. The study was approved by an ethics committee at The University of Queensland. Participants provided informed consent prior to participating and completed a medical screening questionnaire before being accepted into the study.

Experimental Protocol

Participants completed three incremental cycling tests to exhaustion, within three discrete cadence ranges: 50-55, 80-85 and 110-115 rev · min^{-1}. All testing sessions were held at the same time of day to account for changes in circadian rhythm, and a minimum of five days separated each session. All trials were completed at the Queensland Academy of Sport, and were conducted under controlled environmental conditions (19.7 ± 1.1 ˚C; 53.2 ± 9.7 % RH; 756.9 ± 3.1 mmHg). Participants were cooled throughout the tests with a pedestal fan.

All tests were performed on the same AXIS cycle ergometer (Swift Performance Equipment, Carole Park, Australia), although participants used their own shoes and pedals. Measurements of seat height, seat fore-aft position, forward reach, seat-handlebar height difference and crank length were recorded from the participants' own bicycles in order to replicate their usual cycling position during testing and to ensure consistency across trials. The left and right AXIS Cranks (Swift Performance Equipment, Carole Park, Australia) have two full-bridge 350 Ω strain gauge configurations. One configuration measured strain on the crank to provide radial force, and the other measured shear on the crank to provide tangential torque. Data from each channel is preconditioned ten times oversampled data that is reported at a rate of 100 Hz. The tangential torque was multiplied by the crank angular velocity to determine EP. The cranks were calibrated using a dynamic calibration rig, and zeroed daily by storing offsets at 0°, 90°, 180° and 270°.

Participants were asked to refrain from intense physical activity in the 24 hours prior to each testing session and advised to schedule a rest or active recovery day on the day before each laboratory visit. They were provided with dietary guidance to consume 7 g CHO · $kg\ BW^{-1}$ within the 24 hours before testing and a pre-test meal consisting of 2 g CHO · $kg\ BW^{-1}$ two hours prior to arrival at the laboratory.

Incremental Tests

The test protocol required participants to first cycle for five minutes at an external power output (EP) of 100 W, after which the EP was increased by 50 W · 5 min^{-1} until volitional fatigue. Participants completed the three incremental tests in random order on separate days.

Standard open-circuit spirometry techniques were used throughout trials to determine respiratory gas exchange measures (Moxus Modular V,˙O_2 System, AEI Technologies, Pittsburgh, USA). Prior to each testing session, the metabolic cart was calibrated using alpha gases of known concentration (Coregas Pty Ltd, Yennora, Australia) and the turbine ventilometer was calibrated using a 3-L syringe at various flow rates. Expired gas was collected and averaged over 30-s sampling periods. Samples collected within the final two minutes of each 5-min bout were averaged for inclusion in the analysis.

Oxygen consumption (L · min^{-1}) was converted to metabolic power (MP; the rate of metabolic energy consumption, measured in J · s^{-1}) using the associated Respiratory Exchange Ratio (RER) and Zuntz's (1901) Thermal Equivalents of Oxygen for the Nonprotein Respiratory Quotient (OE) (Whipp and Wasserman 1969). Delta Efficiency (DE) was calculated either of two ways, depending on the model. First, DE was equal to the gradient of the linear regression between EP (J · s^{-1}, i.e., Watts) (on the y-axis) and MP. This was termed DE_1. DE was also calculated as the ratio of

a change in EP and the related change in MP for all combinations of EP and MP between stages 1 and 5, i.e., DE_2. The mean of all of these combinations was used for the respective models, as described below.

IP Models

The IP Models were founded on the assumption that the sum of EP and IP was equal to TMP, such that

$$MP = \frac{TMP}{DE_{1or2}} = \frac{(EP + IP)}{DE_{1or2}}$$

The gas exchange and crank power data for each participant at each cadence were input into all of the following models:

Model 1a – according to the following equation by Hansen et al. (2004):

$$EP + IP = (VO^2_{exercise} \cdot OE) - (VO^2_{rest} \cdot OE) \cdot DE$$

where the constant of 0.00417 $L \cdot s^{-1}$ was used for resting VO_2 (VO_{2rest}) across all participants (regardless of the cyclists' body mass) (Hansen et al. 2004). The net VO_2 ($VO_{2exercise}$ - VO_{2rest}) ($L \cdot min^{-1}$) was converted to MP ($J \cdot s^{-1}$) using the measured RER and the respective OE (as described above). DE was calculated for this model using the EP/MP ratio.

Model 1b – modified from the above equation by Hansen et al. (2004) as follows:

$$EP + IP = (VO^2_{exercise} - VO^2_{rest}) \cdot OE \cdot DE$$

The OE for $VO_{2exercise}$ was associated with the measured RER. The OE for VO_{2rest} was associated with an RER of 0.82 (McArdle et al. 1996). The values for VO_{2rest} and DE were as for Model 1a (0.00417 $L \cdot s^{-1}$ and DE_2).

Model 1c – the same as for Model 1b, except that VO_{2rest} was calculated using the ACSM guidelines for resting metabolic rate, i.e., 3.5 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$ (American College of Sports Medicine 2000).

An IP value was calculated for each of the five stages from 100 to 300 W when Models 1a, 1b and 1c were employed. The mean of the five values for each cadence range were used for comparative purposes.

Model 2a – according to the model of Francescato et al. (1995) in which, for each cadence range, the metabolic counterpart to IP was equal to the x-intercept of the relationship between EP and MP (when EP was plotted on the y-axis). MP was calculated for each EP as follows:

$$MP = (VO^2_{exercise} - 3.5 \text{ ml } O^2 \cdot kg_{body\ mass}^{-1}) \cdot 20.9 \text{ J} \cdot ml^{-1}$$

The x-intercept of the EP/MP relationship, IP, was converted to its mechanical equivalent by multiplying by DE1, i.e., the slope. Thus, a single IP value was derived for each cadence range.

Model 2b – modified from the model of Francescato et al. (1995) so that VO_{2rest} could be converted to MP with a relevant RER:

$$MP = (VO^2_{exercise} \cdot OE) - (3.5 \text{ ml } O^2 \cdot kg_{body\ mass}^{-1} \cdot OE_{RER=0.82})$$

where OEs for the respective measured RERs (Zuntz 1901) were used to convert the $VO_{2exercise}$ values ($L \cdot min^{-1}$) to MP ($J \cdot s^{-1}$) (Whipp and Wasserman 1969). VO_{2rest} was calculated as for Models 1c and 2a (American College of Sports Medicine 2000) and was then converted to MP using the O_2-equivalent for an RER of 0.82 (McArdle et al. 1996). As per Model 2a, the IP was equal to the mechanical equivalent of the x-intercept of the EP/MP relationship (x-intercept multiplied by DE1), yielding a single value for IP for each cadence.

Model 3 – according to the equation by Minetti et al. (2001) as follows:

$$IP = 0.153 \cdot BM \cdot cadence^3$$

where BM is body mass (kg) and cadence is measured in Hz. An IP value was calculated using Model 3 for each stage; the mean of these was used for comparisons.

Data Analysis

Data for all variables from stages one to five were included for analysis of the incremental test at 50-55 $rev \cdot min^{-1}$ since all participants were still exercising submaximally (RER $\leq$ 1.0) during the fifth stage (300 W). Four and three participants were still cycling submaximally at 300 W during the tests at 80-85 and 110-115 $rev \cdot min^{-1}$, respectively. Their metabolic and EP data at 300 W were included when calculating their DE values. Mean (± SD) IP was calculated for each model at each cadence range. Mean differences and limits of agreement (mean difference ± 2SD) were calculated between IP values from each model (Hopkins 2000), and Bland-Altman plots were used to illustrate agreement (Bland and Altman 2010). The threshold for similarity in IP was set to 5 W (i.e., a difference of less than 5 W indicated similarity between IP estimates), which was large enough to encompass the mean typical error (TE) in IP calculated for all stages of each incremental test by Models 1a-c and 3 (TE = 0.38 to 4.24, Hopkins 2011). Assuming a DE equal to 25% and an OE of 20.9 $J \cdot ml^{-1}$, 5 W is equivalent to a VO_2 of ~0.057 L $O_2 \cdot min^{-1}$ (VO_2 = IP ·

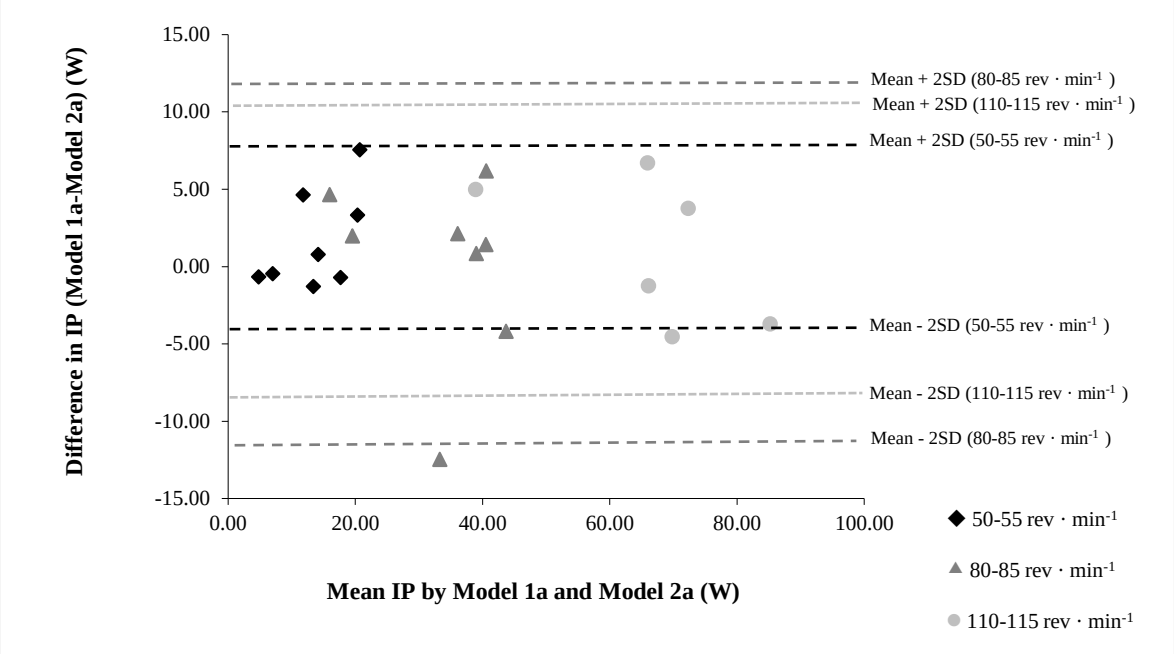

Figure 1 Bland-Altman plot of the difference in IP values calculated by Model 1a and Model 2a at low, moderate and high cadences.

DE^{-1} · 20.9 J · ml O_2^{-1} · 60 sec), which is less than 5% of the mean VO_2 (1.6 L) measured in this study at 100 W and 50-55 rev · min^{-1}. Furthermore, the smallest worthwhile change in mechanical power output has previously been reported to be between 0.5 and 1% (Lamberts et al. 2009; Paton and Hopkins 2001).

Results

Mean (±SD) IP calculated by each model, at each cadence range, are included in Table 1.

Tables 2, 3 and 4 show mean differences (W) and limits of agreement for IP calculated by Models 1a-c, 2a-b and 3 for each of the cadence ranges. Differences exceeding the threshold for similarity between models (i.e., 5 W) are denoted by an asterisk. A Bland-Altman plot of the mean difference in IP calculated by Models 1a and 2a, the models described by Hansen et al. (2004) and Francescato et al. (1995) respectively, for each participant at each cadence is illustrated in Figure 1.

Discussion

The purpose of this study was to apply different models for calculating internal mechanical power (IP) using data from the same group of well-trained male cyclists to compare the results across a range of cadences. It was hypothesised that the models that required better resolution in their contributing values, i.e., measured rather than predicted values, would produce more similar results. Thus, according to the hypothesis, Models 1c and 2b should have calculated results more alike in comparison to the other models for two reasons: i) they both required an estimation of resting VO_2 based upon the participants' body mass; and ii) both of these models used oxygen energy equivalents based upon the actual measured exercising RERs and an estimated resting RER. The results of this study showed that the IP estimates for Models 1c and 2b were indeed similar at 50-55 and 80-85 rev · min^{-1} (Tables 2 and 3). The lower limit of agreement between the models was just outside of the accepted threshold for agreement (5 W) at 110-115 rev · min^{-1} (Table 4). These models were no closer in agreement, however, than the differences in IP values calculated by Models 1a, 1b and 1c, which were based on a method described by Hansen et al. (2004).

Physiological Approach to IP: Similar Basis, Different Models

The novelty of this study was the comparison of IP calculated using pre-existing physiological models (Francescato et al. 1995; Hansen et al. 2004) and various modified versions of them. The basis of these models was the same, i.e., that the metabolic cost of EP

Table 1. Internal mechanical power (IP) calculated using each of the models at each of three cadences.

	Internal Mechanical Power[1] (W)		
	50-55 rev·min^{-1}	80-85 rev·min^{-1}	110-115 rev·min^{-1}
Model 1a	12.9 ± 5.1	33.4 ± 10.9	60.7 ± 19.5
Model 1b	13.5 ± 5.0	34.0 ± 11.1	61.3 ± 19.7
Model 1c	12.7 ± 5.5	33.2 ± 10.6	60.7 ± 18.9
Model 2a	14.6 ± 6.8	33.6 ± 10.1	64.5 ± 14.3
Model 2b	10.7 ± 5.2	32.6 ± 10.8	70.2 ± 22.0
Model 3	7.5 ± 1.1	29.4 ± 3.2	72.1 ± 6.2

[1]The mean IP calculated for the five stages of each incremental test is presented here for Models 1a-c and Model 3; a single value was calculated for the entire test using Models 2a and 2b

(EP/DE) can be subtracted from MP, the energetic counterpart of the net oxygen consumption ($VO_{2exercise}$ - VO_{2rest}), to leave the metabolic cost of IP, which can be converted to its mechanical equivalent (W) by multiplying by DE. Despite these similarities and their intention to calculate the same value, the results from models of previous studies (Francescato et al. 1995; Hansen et al. 2004; Martin et al. 2002; Tokui and Hirakoba 2008), have not provided a consistent value for IP at the same, or similar, cadence. In this study, the models proposed by Hansen et al. (2004) and Francescato et al. (1995) (Models 1a and 2a, respectively) were applied to data from the same participant group (Figure 1). Model 1a tended to predict a lower value for IP across the cadence range (0.1 to 1.6 W), with limits of agreement (-11.5 W to 11.6 W) that exceeded the imposed threshold of 5 W to indicate a meaningful difference (Tables 2-4).

Variations in the Resting Metabolism Term

Modifications in this study to the model described by Hansen et al. (2004) (Model 1a in this study) were intended to highlight the possible error in using an energy equivalent associated with an exercising RER for converting a resting oxygen consumption value to metabolic power. Model 1b required an OE for VO_{2rest} that was associated with a resting RER, so that exercising and resting metabolic powers could be calculated prior to baseline subtraction, and in effort to determine a more accurate value for IP. Analysing the difference in the mean IP values calculated by these models did not indicate any

Table 2. Mean differences and limits of agreement of IP (W) calculated during an incremental test at 50-55 rev · min^{-1}.

	Model 1a (W)	Model 1b (W)	Model 1c (W)	Model 2a (W)	Model 2b (W)	Model 3 (W)
Model 1a						
Model 1b	0.5 (0.2 to 0.8)					
Model 1c	-0.2 (-2.9 to 2.5)	-0.7 (-3.4 to 2.0)				
Model 2a	1.6* (-4.6 to 7.9)	1.1* (-5.2 to 7.5)	1.8* (-2.5 to 6.2)			
Model 2b	-2.2* (-5.3 to 0.9)	-2.7* (-5.8 to 0.4)	-2.0 (-4.9 to 0.9)	-3.3* (-8.1 to 0.4)		
Model 3	-5.4* (-16.5 to 5.7)	-5.9* (-16.9 to 5.1)	-5.2* (-17.3 to 6.9)	-7.0* (-21.9 to 7.9)	-3.2* (-14.8 to 8.4)	

* Mean difference and/or limits of agreement exceed threshold for similarity between models (i.e., 5 W). A positive value for the mean difference indicates a greater prediction of IP from the model in the left-hand column compared to the model in the top row.

Table 3. Mean differences and limits of agreement of IP (W) calculated during an incremental test at 80-85 rev · min^{-1}.

	Model 1a (W)	Model 1b (W)	Model 1c (W)	Model 2a (W)	Model 2b (W)	Model 3 (W)
Model 1a						
Model 1b	0.6 (0.1 to 1.1)					
Model 1c	-0.2 (-2.9 to 2.5)	-0.8 (-3.5 to 1.9)				
Model 2a	0.1* (-11.5 to 11.6)	-0.5* (-12.4 to 11.4)	0.3* (-10.7 to 11.2)			
Model 2b	-1.0 (-4.4 to 2.4)	-1.6* (-5.0 to 1.9)	0.8 (-2.0 to 0.4)	-1.0* (-11.7 to 9.6)		
Model 3	-4.2* (-22.3 to 13.9)	-4.8* (-23.3 to 13.7)	-4.0* (-21.5 to 13.5)	-4.3* (-19.6 to 11.1)	-3.2* (-20.6 to 14.2)	

* Mean difference and/or limits of agreement exceed threshold for similarity between models (i.e., 5 W). A positive value for the mean difference indicates a greater prediction of IP from the model in the left-hand column.

Table 4. Mean differences and limits of agreement of IP (W) calculated during an incremental test at 110-115 rev · min^{-1}.

	Model 1a (W)	Model 1b (W)	Model 1c (W)	Model 2a (W)	Model 2b (W)	Model 3 (W)
Model 1a						
Model 1b	0.6 (-0.2 to 1.0)					
Model 1c	-0.04 (-2.77 to 2.68)	-0.6 (-3.5 to 2.3)				
Model 2a	1.0* (-8.4 to 10.3)	0.3* (-9.2 to 9.9)	0.9* (-7.1 to 8.9)			
Model 2b	-1.9 (-4.7 to 3.7)	-2.5* (-8.1 to 3.0)	-1.9* (-6.2 to 2.4)	-2.8* (-11.0 to 5.4)		
Model 3	4.6* (-20.6 to 29.8)	4.0* (-21.5 to 29.4)	4.6* (-18.3 to 27.4)	3.6* (-14.8 to 22.0)	6.5* (-17.5 to 30.2)	

* Mean difference and/or limits of agreement exceed threshold for similarity between models (i.e., 5 W). A positive value for the mean difference indicates a greater prediction of IP from the model in the left-hand column.

appreciable change in IP at any cadence despite these efforts (Tables 2, 3 and 4). Further, Model 1c was included to investigate the potential problem of using the same resting $\dot{V}O_2$ for all participants (i.e., 0.00417 $L \cdot s^{-1}$ in Models 1a and 1b). Although it would be preferable for such a comparison, resting VO_2 was not measured in this study. Employing a rate of 3.5 ml O_2 per kilogram, as recommended by guidelines from ACSM (American College of Sports Medicine 2000), it was hoped might at least have individualised the estimate somewhat. Again, there were no differences between IP calculated by Model 1c and Model 1b, which was identical except for the value for VO_{2rest}. It would appear, therefore, that the magnitudes of difference between an assumed resting VO_2 and the actual metabolic cost of rest, and between the energy equivalents for rest and exercise, were too small to have an effect on the IP estimate.

Participant Characteristics as a Cause of Inconsistencies in IP

One explanation for the lack of consistency in results between earlier studies may have been due to differences in participant characteristics. Differences between participants in limb segment characteristics can result in differences in segment kinetic and potential energies. For example, it is possible that a group of physically active but not highly trained participants (Francescato et al. 1995; Tokui and Hirakoba 2007, 2008) can have quite different segment masses and mass distributions, and even segment lengths, compared to a group of participants "highly trained in road cycling" (Martin et al. 2002). Such differences in these segment characteristics and potential and kinetic energies, in turn, affect kinematic patterns (Durkin and Dowling 2006; Ganley and Powers 2004), and hence change IP. These differences may also manifest in the metabolic cost. Furthermore, differences in training status probably infer differences in pedalling skill; trained cyclists might be more economical (a smaller metabolic cost per unit of work), particularly at higher cadences (Marsh and Martin 1993). By applying the models to the same group of participants in the present study, it was thought that potential variations in IP due differences in segment characteristics and pedalling economy, that may have occurred across previous studies, could have been avoided here. If it were possible to avoid these variations, the values for IP calculated by Model 1a and 2a, and indeed across all of the models in this study, would have been similar. This was not the case, however, in this study (Tables 2, 3 and 4).

Measured vs. Predicted Metabolic Variables as a Cause of Inconsistent IP Estimations

An alternate explanation for the differences in IP is that they could be attributed to discrepancies in the values entered into the models: net versus gross $\dot{V}O_2$ values; predicted versus measured resting metabolic rate; a single, general oxygen energy equivalent versus an intensity-specific OE. For example, Francescato et al. (1995) calculated IP using the method of Model 2a and reported values equivalent to 1.9 and 78.4 W at 60 and 100 $rev \cdot min^{-1}$ (when multiplied by the mean lower limb mass of 23.76 kg). On the other hand, Martin et al. (2002) used the same method except that they plotted gross $\dot{V}O_2$ against EP and then subtracted a generic resting MP from the intercept. They calculated metabolic equivalents for IP of 98 ± 38 and 144 ± 58 W [rather than reporting the mechanical IP] in adults who were cycling at 60 and 90 $rev \cdot min^{-1}$. This would translate to mechanical equivalents in the order of 25 and 40 W, respectively, had a DE of approximately 25% been employed for the metabolic to mechanical energy conversion. The values for IP in these studies were inconsistent (Francescato et al. 1995; Martin et al. 2002), which is why the results of Models 2a and 2b were compared in the present investigation. That is, Models 2a and 2b were selected to discern the effect of subtracting resting VO_2 compared to resting MP from the exercising counterparts. The results suggested that Model 2a, which subtracted a predicted resting $\dot{V}O_2$ from the measured $VO_{2exercise}$, typically calculated a larger value for IP at all cadences, with the greatest difference occurring at 80-85 $rev \cdot min^{-1}$. Model 2b allows for more relevant energy equivalents to be used for converting oxygen consumption to metabolic power. For example, the OE for the mean RER measured at 200 W was used to convert VO_2 to MP, rather than the single OE value of 20.9 $J \cdot ml^{-1}$ that was used in Model 2a for converting $\dot{V}O_2$ recorded at all EPs between 100 W and 300 W to metabolic power. Francescato et al. (1995) maintained that using this constant conversion factor of 20.9 $J \cdot ml^{-1}$ would cause less than 4% error in the calculations. The results of current study showed, however, that the values estimated for IP were not the same when Models 2a and 2b were compared, i.e., that using OEs relative to the exercise intensity when converting VO_2 to MP typically resulted in smaller values to when the 20.9 $J \cdot ml^{-1}$ conversion factor was used for all intensities (Tables 2, 3 and 4). Therefore, when accuracy is the goal of the IP estimation, using Model 2b may be favourable.

Limitations to Model 3: A Biomechanical Model

Previously, the estimate of IP using the model by Minetti et al. (2001) has been used to compare against physiological estimates (Hansen et al. 2004; Tokui and Hirakoba 2007) because of its ease of application, since only information about cadence and body mass is needed (Minetti et al. 2001). In agreement with the hypothesis and consistent with earlier studies, where it was concluded that the model by Minetti et al. (2001) underestimates IP (Hansen et al. 2004; Tokui and Hirakoba 2007), the values for IP from Model 3 in this study were considerably smaller at all cadences than for any of the other models (Tables 2, 3 and 4). The limits of agreement across the cadences ranged from -23.3 W to 30.2 W – six times the threshold for similarity. Such a result was expected, not only because of the results of previous comparisons (Hansen et al. 2004; Tokui and Hirakoba 2007), but also because the model by Minetti

et al. (2001) was derived from a kinetic and kinematic analysis of cycling. Such a biomechanical analysis presents a 'net' view of the flow of energy in the system, i.e., it can describe the flow of energy within and between limb segments, but cannot determine how much and from which muscles the energy was sourced (Zatsiorsky 1994), nor can it account for the energy that is used for ventilation, circulation and digestion and that which is lost as heat. A physiological approach, in contrast, can describe the magnitude of the energy flow in a 'gross' sense, that is, it does account for these demands for energy as well as the demands from the exercising muscles, although which muscles and how much energy they require cannot be identified. The Minetti et al. (2001) model represents the line of fit of data from the biomechanical analysis, and so its simplicity is traded off against more accurate information about the energy flow of the system. This model is further limited in its application to elite cyclists because the data for the line of fit was sourced from recreational cyclists on mountain bikes (Minetti et al. 2001). Their cycling posture may have produced an alternate kinematic pattern to that which might have been derived from the cyclists of the present study. It is possible that these features of the Minetti et al. (2001) model (i.e., Model 3) effected the substantial difference in the present data, where IP was consistently predicted to be lower than that calculated by any of the physiological models (Models 1a-c and Models 2a and 2b).

The Sum of EP and IP

Calculating IP has been deemed important because if it were possible to decrease its magnitude, it has been suggested that more metabolic energy would be available to perform the EP and thus gross efficiency (external power/metabolic power) would improve and performance would be enhanced (Hull et al. 1991). However, it is important to note that while the $\dot{V}O_2$ presents a gross measure of the energy flow of the system, the basis of the above physiological models, that VO_{2net} is simply equal to the sum of the metabolic equivalents of EP and IP, has been rejected by biomechanists (Broker and Gregor 1994; Kautz and Neptune 2002; van Ingen Schenau 1998; van Ingen Schenau et al. 1990; Zatsiorsky 1998). It has instead been argued that there is some degree of transfer of energy between EP and IP. In other words, it has been argued that the same metabolic energy is used to perform EP and IP – there are not two independent sources of metabolic energy for the different mechanical energy destinations (Broker and Gregor 1994; van Ingen Schenau et al. 1990). Thus, the physiological approach to calculating IP provides an over-estimate of IP because it considers two separate metabolic costs. Further discussion of this is outside the scope of this manuscript, although suffice to say that due to limitations of the biomechanical models, the extent of this transfer is not known.

Conclusion

The present study showed that IP, calculated by two previously described physiological models (Francescato et al. 1995; Hansen et al. 2004) applied to the same group of elite cyclists, was more than 5 W different at 50-55, 80-85 and 110-115 $rev \cdot min^{-1}$. Variations to the model by Francescato et al. (1995) suggested that using relevant energy equivalents for O_2 conversion may have an effect on the prediction of IP, compared to using the same OE for all intensities of activity. On the other hand, modifications to the model by Hansen et al. (2004) did not indicate such an effect, nor did they demonstrate an effect of using an individualised resting oxygen consumption value in place of a generic one. It was also shown that IP estimated using a biomechanically-derived model of cycling (Minetti et al. 2001) was low across all of the cadences tested in the present study, consistent with previous findings (Hansen et al. 2004; Tokui and Hirakoba 2007). Therefore, one must consider the accuracy of the values that are required for the estimation of IP using physiological models when making assessments of IP during cycling. In order to make the most accurate estimation of IP, it would be best to measure resting metabolic rate, use a relevant OEs for the intensity of activity (i.e., an OE associated with the resting RER for converting to MP at rest, and an OE associated with the exercising RER for converting to exercising MP).

Practical applications

Even if it were not possible to reduce IP in cycling so that more metabolic energy may be used to increase EP, knowledge of the metabolic cost of IP is of particular importance to exercise and sport scientists and to coaches. For example, a scientist or coach may want to consider two investigations of the efficacy of a particular supplement, drug or training intervention that used cycling performance tests at two different cadences. Equipped with an understanding of IP, that scientist or coach could make an allowance for the additional metabolic cost at the higher cadence when interpreting and comparing the results of the two studies. In the numerous investigations where the cadence is not reported, the scientist or coach would know to interpret the results cautiously. Moreover, with the knowledge of their athlete's MP-cadence (IP) relationship, a coach or scientist can be more informed when planning to use a particular gearing combination or developing a pacing schedule for racing. In order to determine the IP and its metabolic cost, the scientist or coach must decide which model to use for its estimation. The results of current investigation would help the scientist or coach to make the most appropriate decision for the situation: using Model 3 might not be suitable for a pursuit cyclist because the predicted values for IP are too large due to its basis on upright cycling by novices; using Model 2a rather than Model 2b when deciding on a pacing strategy for an elite road time triallist

might mean that the cyclist crosses the finish line with too much energy to spare, i.e., that they could have gone faster. Rather than using only one of the models of the current study, and bearing in mind the assumptions and limitations of both the physiological and biomechanical models, it may be more appropriate, particularly in a practical setting, to define a range within which the real value of the energetic cost of IP lies. At the upper limit of this range is the overestimation of IP, that is, IP calculated using a physiological model in which IP and EP are considered independent and without the capacity to transfer energy between one another. Ensuring accuracy in the contributing values, as in, for example, Models 1c and 2b in the present study, may assist in moving closer to the real value of IP. At the lower limit of this range is the estimate of IP using a biomechanical approach, since the limitations and assumptions of biomechanical models would underestimate IP. Future research will continue to refine this estimate of the lower limit by improving upon biomechanical models. Ultimately, this range of IP values would allow for the description of energy during cycling that considers a combination of metabolic and mechanical parameters.

Acknowledgment

The authors thank the Centre of Excellence for Applied Sport Science Research at the Queensland Academy of Sport for the grant to fund this research.

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