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A four compartment model on human exercise bioenergetics

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Abstract

Performance in endurance sports depends on the athlete's ability to generate power output through muscle contraction. The energy requirements of muscles are satisfied by the alactic and lactic bioenergetic pathways, working anaerobically, and the aerobic oxidative phosphorylation of fats and carbohydrates. The aim of this study was to apply further extensions to hydraulic bioenergetic modelling to better describe the regulation of oxidative fuel selection. For this reason, a four compartment bioenergetic model was introduced and regulation of fat and carbohydrate oxidation was implemented. Further regulation was applied to both oxidative fuel selection and anaerobic glycolysis to depend on the current carbohydrate store. The model was formulated mathematically as differential equations, which were solved numerically to perform simulations of human bioenergetics in exercise. Simulation results showed good consistency with experimental findings.

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1. Introduction

In endurance sports, performance is limited by the athlete's ability to produce power output through muscle contraction. The energy requirements of muscle are satisfied by the anaerobic and aerobic pathways of metabolism. The anaerobic alactic component refers to phosphagen splitting and the lactic component is the anaerobic breakdown of carbohydrates to lactate through glycolysis. The aerobic process refers to the oxidation of fats and carbohydrates, also called oxidative phosphorylation. These are all closely integrated processes that serve to keep the concentration of the energetic substrate adenosine triphosphate at an acceptable level for the working muscle.

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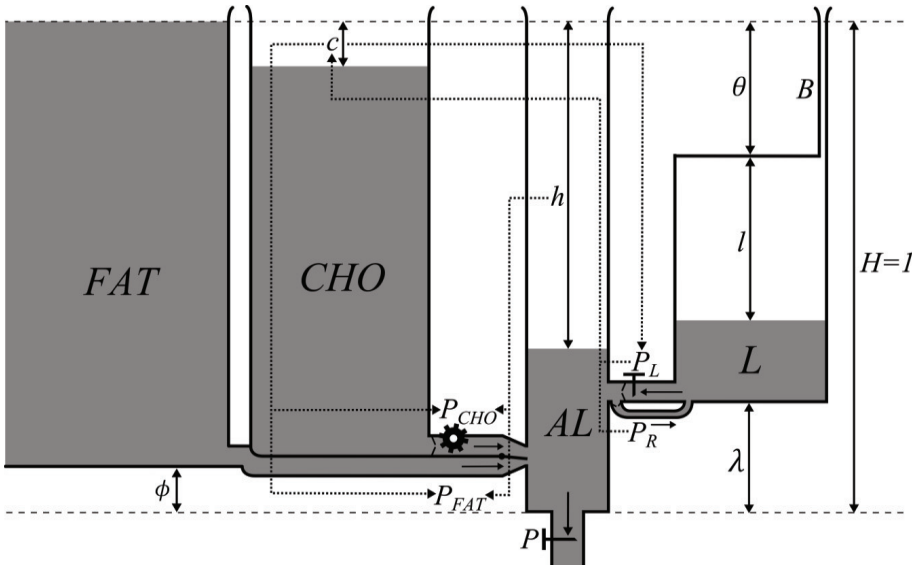

Fig. 1. The four compartment bioenergetic model. Solid arrows show directions of flow and the dashed arrows show which argument has an effect on the pointed out variable, except for hydraulic effects.

Mathematical models of human bioenergetics may be empirical models or theoretical constructs often represented as hydraulic compartment models. Hydraulic models include the simple critical power model and since its introduction many extensions and modifications have been carried out by Margaria [1], Morton [2, 3], and Behncke [4] to develop more realistic models of the human bioenergetics. The aim of the present study is to apply further extensions to the hydraulic bioenergetic model to provide a more circumstantial model.

2. The bioenergetic model

The bioenergetic model considered in this study is formulated with the intention to better than previous hydraulic models, describe the regulation of oxidative fuel selection. Previous hydraulic models have presumed that there is an unlimited source of carbohydrates. However, carbohydrate stores are finite and the regulation of fuel selection affects the time lapse of glycogen depletion. Therefore, this model consists of four compartments instead of the usual three compartments, namely: The fat energy store (FAT), the carbohydrate energy store (CHO), the alactic energy store of phosphagens (AL), and the lactic energy store of carbohydrates associated with the glycolytic production of lactate (L). Fig. 1 gives a diagrammatical representation of the considered hydraulic model. In this study, we use mechanical power as a proxy for metabolic energy expenditure rate, presupposing a constant mechanical efficiency. The energy stores are represented by the fluid in each compartment and the rate of energy transformation (or power) is represented by the fluid flow between compartments. All compartments except FAT has a variable fluid level denoted by c, h, and l for the CHO, AL, and L compartments, respectively. This also means that the FAT compartment has infinite capacity and the remaining compartments are all finite.

The flow out of the AL compartment represents the power output (P) and the flows between compartments are decided by their differences in fluid level. The flow out of the bottom of the AL compartment induces a drop in the fluid level that results in flows from the FAT and CHO compartments. The flow from the FAT and CHO compartments to the AL compartment are representatives of the oxidative phosphorylation of fatty acids (P_{FAT}) and carbohydrates (P_{CHO}), respectively. These processes are aerobic and results in the production of adenosine triphosphate (ATP), the immediate fuel source of the muscle. The sum of P_{FAT} and P_{CHO}, the aerobic power ($\dot{V}O_2$), increases linearly with h, because of the hydraulics of the model. As exercise also induces an increase in c, a hydraulic pump has to be added on P_{CHO} to keep this linearity. This is indicated by the paddle wheel in Fig. 1. The regulation of oxidative phosphorylation substrate is decided by the hinged rudder at the outlet of P_{FAT} and P_{CHO}.

This regulation, which depends on both c and h, is decided by a modulated sinus-formed relationship for fatty acid oxidation, proposed by Chenevière et al. [5].

Anaerobic glycolysis is represented by the flow from the L compartment to the AL compartment. This flow is dependent on the difference between h and l. Glycolysis transforms carbohydrates into puruvic or lactic acid while producing ATP. Enzymes involved in glycolysis are inhibited by low pH and low carbohydrate stores because glucose is the primary substrate in this pathway. As sustained anaerobic glycolysis results in a fall in pH, P_L is directly influenced by l. For this reason, P_L decreases linearly with l while increasing with h, thereby buffering the alactic substrates at the onset of exercise. Furthermore, P_L is set to decrease with decreasing carbohydrate store (increasing c), indicated by a tap in Fig. 1. If exercise intensity and thereby power output declines, h may reach a lower value than $l + \theta$ and recovery of the L compartment sets in. In realty, this process, called gluconeogenesis, is much slower than glycolysis. Therefore, check valves force the return flow (P_R) to pass through the lower narrower tube (see Fig. 1) constrained to a lower maximal flow. Because glycolysis uses glucose as a substrate, the flows P_L and P_R are set to directly affect c. For any given amount of produced ATP, glycolysis needs 16 times the amount of glucose, compared to oxidative phosphorylation. The hydraulic bioenergetic model considered has a presumed height of $H = 1$ which is also the height of the AL compartment. The top of the FAT, CHO, and AL compartments are limited to this height and the top of the L compartment is located at a distance θ lower than the others to delay the onset of anaerobic glycolysis. According to Crowther and Carey [6] it may take several seconds until anaerobic glycolysis is activated. Furthermore, the bottom of the FAT and CHO compartments are located at a height ϕ and the bottom of the L compartment is at height λ. The L compartment is intended to simulate the energetic contribution of anaerobic glycolysis, however, does not represent a separate store of carbohydrates. From the top of the L compartment, a narrow tube, B is attached. This tube does not contain any substantial amount of lactic energy and therefore it is not included in the mathematical description in section 3. This hydraulic model will enable five different phases of compartment dynamics that are represented by five different systems of ordinary differential equations. For known power output, these equations may be solved to simulate the bioenergetics of exercise.

3. Differential equations

The bioenergetic model can be represented by some set of differential equations. The levels h and l along with θ, ϕ, and λ determines the current phase and therefore the active differential equations of the model. However, one differential equation is identical to all phases, namely

$$P = P_{FAT} + P_{CHO} + P_{AL} + P_L = P_{FAT} + P_{CHO} + A_{AL}\frac{dh}{dt} + A_L\frac{dl}{dt} \tag{1}$$

where P_{FAT} and P_{CHO} are the powers of oxidative phosphorylation for fats and carbohydrates, respectively, P_{AL} is the alactic power, and P_L is the lactic power of sole anaerobic glycolysis. If $h < l$, then P_L will be replaced by P_R which represents the gluconeogenesis. A_{AL} and A_L are the cross-sectional areas of the AL and L compartments, respectively, and t is time. The following five phases are possible for the model.

3.1. Phase O

This phase is valid on the interval $h < \theta$, $l = 0$ and consists of a system of two differential equations, including equation (1) and

$$A_{CHO}\frac{dc}{dt} = P_{CHO}, \tag{2}$$

$$\text{where } P_{CHO} = M_o\frac{h}{1-\phi} - P_{FAT}, \tag{3}$$

$$\text{and } P_{FAT} = \frac{1-\phi-c}{1-\phi}M_{FAT1}\sin\left[\left(h\frac{\pi^{\frac{1}{\sigma_1}}}{\pi+2\delta_1}\frac{\pi}{1-\phi} + \delta_1 + \tau_1\right)^{\sigma_1}\right] + \frac{c}{1-\phi}M_{FAT2}\sin\left[\left(h\frac{\pi^{\frac{1}{\sigma_2}}}{\pi+2\delta_2}\frac{\pi}{1-\phi} + \delta_2 + \tau_2\right)^{\sigma_2}\right]. \tag{4}$$

However, if $P_{FAT} > M_o \frac{h}{1-\phi}$, then $A_{CHO} \frac{dc}{dt} = 0$ and $P_{FAT} = M_o \frac{h}{1-\phi}$, (5)

else if $P_{FAT} < 0$, then $A_{CHO} \frac{dc}{dt} = M_o \frac{h}{1-\phi}$ and $P_{FAT} = 0$. (6)

In these expressions, A_{CHO} is the cross-sectional area of the CHO compartment, M_O is the maximal rate of oxidative phosphorylation, M_{FAT1} is the maximal fat oxidation rate at full carbohydrate stores and M_{FAT2} is the maximal rate at empty carbohydrate stores. Furthermore, σ_1, δ_1, and τ_1 is the symmetry, dilatation, and translation parameters respectively, for the fat oxidation rate at full carbohydrate stores and σ_2, δ_2, and τ_2 is the symmetry, dilatation, and translation parameters, respectively, at empty carbohydrate stores [5]. The transformation between these fatty acid oxidation kinetics is linearly dependent on the remaining store of carbohydrates, c.

3.2. Phase OL1

This phase is valid on the interval $\theta + l < h < 1 - \lambda$ and consists of the same system of differential equations as phase O, however, complemented with equation (7) and with equations (2), (5), and (6) replaced by equations (8), (9), and (10), respectively.

$$A_L \frac{dl}{dt} = P_L = M_L \frac{1-\phi-c}{1-\phi} \frac{h-\theta-l}{1-\theta-\lambda}, \quad (7)$$

$$A_{CHO} \frac{dc}{dt} = P_{CHO} + 16 A_L \frac{dl}{dt}. \quad (8)$$

If $P_{FAT} > M_o \frac{h}{1-\phi}$, then $A_{CHO} \frac{dc}{dt} = 16 A_L \frac{dl}{dt}$ and $P_{FAT} = M_o \frac{h}{1-\phi}$, (9)

else if $P_{FAT} < 0$, then $A_{CHO} \frac{dc}{dt} = M_o \frac{h}{1-\phi} + 16 A_L \frac{dl}{dt}$ and $P_{FAT} = 0$. (10)

In these expressions, M_L is the maximal rate of sole anaerobic glycolysis and the integer 16 stands for the ratio (32/2) of ATP produced per glucose molecule through complete oxidative phosphorylation (including Krebs's cycle and electron transport chain) divided by the ATP produced for sole glycolysis. Therefore, sole glycolysis drains the carbohydrate store at a 16-fold faster rate than complete oxidative phosphorylation.

3.3. Phase OR

This phase is valid on the interval: $h < \theta + l < 1 - \lambda$, $l > 0$ and consists of the same system of differential equations as phase OL1 but with equation (7) replaced by

$$A_L \frac{dl}{dt} = P_R = M_R \frac{h-\theta-l}{1-\lambda} \quad (11)$$

where M_R is the maximal rate of gluconeogenesis.

3.4. Phase OL2

This phase is valid on the interval: $1 - \lambda < h < 1 - \phi$ and has a lactic power that is not dependent on h. This phase consists of the same system of differential equations as phase OL1 except for equation (7) which is replaced by

$$A_L \frac{dl}{dt} = P_L = M_L \frac{1-\phi-c}{1-\phi} \frac{1-\theta-\lambda-l}{1-\theta-\lambda}. \quad (12)$$

3.5. Phase MOL

This phase is valid on the interval: $1 - \phi < h$ and consists of the same system of differential equations as phase OL2 but with equation (3), (5), and (6) replaced by (13), (14), and (15), respectively.

$$P_{CHO} = M_o - P_{FAT}. \tag{13}$$

If $P_{FAT} > M_o$, then $A_{CHO}\frac{dc}{dt} = 16A_L\frac{dl}{dt}$ and $P_{FAT} = M_o$, (14)

else if $P_{FAT} < 0$, then $A_{CHO}\frac{dc}{dt} = M_o + 16A_L\frac{dl}{dt}$ and $P_{FAT} = 0$. (15)

4. Numerical solution

The differential equations were solved sequentially using the *ode45* function in the Matlab software where the problem was formulated as a system of first order ordinary differential equations. For the assessment of the model we chose model parameters according to the experimental results in study 1 of Gastin et al. [7] to be able to compare simulations to empirical findings. Therefore, constant power outputs of $P = 1.07M_O$ and $P = 1.25M_O$ were simulated for 186 and 94 s duration, respectively. For those variables not available from Gastin et al. [7], we chose feasible values for trained male individuals. Consequently, initial AL, CHO, and L stores were set to 4.18, 542, and 16.0 kJ, respectively. This corresponds to about 40 g ATP, 120 g phosphocreatine, and 500 g carbohydrates for oxidative phosphorylation as reported by MacLaren and Morton [8]. The available carbohydrates to anaerobic glycolysis corresponded to 236 g which is close to the previously used value of Sundström et al. [9]. As a result, the cross-sectional areas for the compartments were $A_{CHO} = 542000/(1-\phi)$ J$^{2/3}$, $A_L = 16000/(1-\theta-\lambda)$ J$^{2/3}$, and $A_{AL} = 4180$ J$^{2/3}$. Furthermore, maximal powers of the different bioenergetic pathways were set to $M_O = 350$ W, $M_{FAT1} = 75$ W, $M_{FAT2} = 2M_{FAT1} = 150$ W, $M_L = 750$ W, and $M_R = M_L/10 = 75$ W. Fat metabolism parameters were set to $\sigma_1 = 0.97$, $\sigma_2 = 1.3$, $\delta_1 = \delta_2 = -0.1$, and $\tau_1 = \tau_2 = 0.08$, representing moderately trained athletes doing cycling exercise [10]. However, σ_2 was chosen to offset M_{FAT2} from M_{FAT1} so it occurred at higher aerobic power, because it represents the fat metabolism at low carbohydrate stores. The model geometrics were set to $\phi = 0.05$ J$^{1/3}$ and $\lambda = 0.1$ J$^{1/3}$ as proposed by Morton [3]. However, the top of L was set at $\theta = 0.2$ J$^{1/3}$ to account for a ~2.2 s delay in the onset of anaerobic glycolysis.

The results of these model simulations can be seen in Fig. 2. Both simulations started in phase O and consecutively continued to the OL1, OL2, and MOL phases. Because the constant power simulations did not involve any recovery, phase OR was never activated in neither of the simulations.

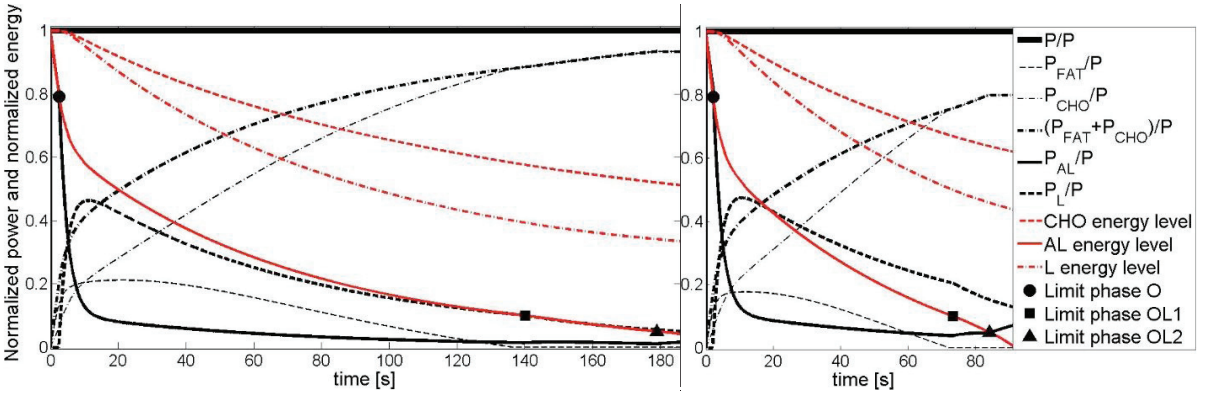

Fig. 2. Simulations of the bioenergetic model with constant power output of $P = 1.07M_O$ (left) and $P = 1.25M_O$ (right). Power and compartment energy are presented in normalized values. Limits between different phases are indicated with geometrical symbols.

The relative contributions to the total work for the $P = 1.07M_O$ simulation was 9.1% FAT, 65.5% CHO, 19.6% L, and 5.8% AL. Furthermore, the corresponding contributions for the $P = 1.25M_O$ simulation was 8.7% FAT, 52.5% CHO, 28.5% L, and 10.3% AL. As a result of these distributions, the $P = 1.07M_O$ simulation gave an aerobic work portion of 75% while Gastin et al. [7] reported the same value to be 76%. Moreover, the $P = 1.25M_O$ simulation gave an aerobic work portion of 61% while experiments show an average portion of 59% [7].

5. Discussion

The main objective of the present study was to introduce further extensions to the hydraulic bioenergetic models developed mainly by Margaria [1] and Morton [2, 3]. More exactly, the intention was to extend the model to include the regulation between fat and carbohydrate oxidation. To perform a complete validation of this model is beyond the scope of this study but some conclusions can be drawn only by comparing the constant power simulations in Fig. 2 with empirical results from constant power exercise testing. In a review paper, Gastin [11] presents previous data [7] on contributions of the three energy supplying systems: Aerobic, alactic, and glycolysis. This data describes the bioenergetics of constant power exercise at 107% of maximal aerobic power and the outline of the included components showed great resemblance with the left graph in Fig. 2. Furthermore, the relative aerobic and anaerobic contributions to work resulting from the proposed model were in line with the same experimental results [7]. This comparison does not tell anything about the relative contributions of fat and carbohydrates, however, the model of Chenevière et al. [5] is validated to experimental data, relating the fat oxidation rate to the total aerobic power. This also provides the corresponding relationship for carbohydrate oxidation since the aerobic power is the sum of fat and carbohydrate oxidation. Furthermore, Heigenhauser et al. [12] showed that glycogen depletion increases the relative contribution of fat metabolism at both 50% and 80% of maximal aerobic power. This ratifies the use of the carbohydrate store (c) as a factor influencing the regulation of oxidative fuel selection in equation (4), which also influences equations (1), (3), (5), (6), (9), (10), (13), (14), and (15). The distinction of fat and carbohydrate energy stores in bioenergetics modelling is most important in prolonged exercise, where the limited carbohydrate store is sufficiently depleted, resulting in impaired performance. Proper bioenergetic modeling along with mechanical modelling enable optimized pacing strategies to be calculated for athletes to improve performance. A pacing scheme may then be communicated by audio or visual interfaces to the athlete.

In this model, neither the basal metabolic rate nor power output constraints connected to any muscle fatigue mechanisms were taken into consideration. However, future modeling efforts may result in the integration of such modifications to further the development in hydraulic bioenergetic models.

References

[1] Margaria, R. *Biomechanics and energetics of muscular exercise*: Clarendon Press Oxford, 1976.
[2] Morton, RH. A three component model of human bioenergetics. *J. Math. Biol.* 1986;24:451-66.
[3] Morton, RH. Modelling human power and endurance. *J. Math. Biol.* 1990;28:49-64.
[4] Behncke, H. A mathematical model for the force and energetics in competitive running. *J. Math. Biol.* 1993;31:853-78.
[5] Cheneviere, X, Malatesta, D, Peters, EM,Borrani, F. A mathematical model to describe fat oxidation kinetics during graded exercise. *Med Sci Sport Exer* 2009;41:1615-25.
[6] Crowther, GJ, Carey, MF, Kemper, WF,Conley, KE. *Control of glycolysis in contracting skeletal muscle. I. Turning it on*, 2002.
[7] Gastin, PB, Costill, DL, Lawson, DL, Krzeminski, K,McConell, GK. Accumulated oxygen deficit during supramaximal all-out and constant intensity exercise. *Med Sci Sports Exerc* 1995;27:255-63.
[8] MacLaren, D,Morton, J. *Biochemistry for sport and exercise metabolism* 1ed: Wiley-Blackwell, 2011.
[9] Sundström, D, Carlsson, P,Tinnsten, M. Comparing bioenergetic models for the optimisation of pacing strategy in road cycling. *Sports eng* 2014;17:207-15.
[10] Cheneviere, X, Malatesta, D, Gojanovic, B,Borrani, F. Differences in whole-body fat oxidation kinetics between cycling and running. *Eur J Appl Physiol* 2010;109:1037-45.
[11] Gastin, PB. Energy system interaction and relative contribution during maximal exercise. *Sports Med* 2001;31:725-41.
[12] Heigenhauser, GJF, Sutton, JR,Jones, NL. Effect of Glycogen Depletion on the Ventilatory Response to Exercise. *J Appl Physiol* 1983;54:470-4.