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Development of VO_2 Prediction Equation for Elliptical Crosstrainer Exercise

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Purpose: The purpose of this study was to develop an accurate metabolic prediction equation for elliptical cross-trainer (ECT) exercise.

Methods: Forty male and female subjects (mean SD, age: 30 7 y; height: 173 11 cm; weight: 72.3 13.8 kg; body composition: 18.3 6.9 %) completed two randomized testing sessions. Steady-state oxygen uptake (VO_2) was measured while subjects exercised on the ECT at nine separate workloads each testing session. Steady-state VO_2 measurements from the last 2 min of each workload were used to develop a metabolic prediction equation for ECT exercise.

Results: Multiple regression analysis to predict steady-state VO_2 from ECT resistance, ETC cadence, and subject body mass resulted in the following model (R^2 = 0.783):

Steady-state VO_2 = 3.82 + 1.5(Cadence) + 1.22(Resistance) − 0.11(Weight)

Both the standard error of the estimate (SEE) and total error (TE) for the prediction of steady-state VO_2under all ECT workload conditions combined was 2.8 $mLkg^{-1}min^{-1}$. The correlation coefficient between predicted and measured steady-state VO_2 values was r = 0.89. A dependent t-test revealed significant mean differences ($P < 0.05$) between predicted (21.2 $mLkg^{-1}min^{-1}$) and measured (21.6 $mLkg^{-1}min^{-1}$) VO_2 measurements.

Conclusion: SEE and TE values for the developed ECT metabolic equation are similar to those reported in previous studies investigating the accuracy of metabolic equations for other exercise modalities. These findings support the use of the equation developed in the present study to predict steady-state VO_2 for ECT exercise.

Key Words: Oxygen consumption, prediction of VO_2, ellitpical crosstrainer

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Roles of DYRK1A in the Pathogenesis of Down Syndrome and Alzheimer's Disease

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Individuals with Down Syndrome (DS) show an early-onset of Alzheimer's disease (AD), resulting from an extra copy of chromosome 21. Located on this chromosome are genes that encode b-amyloid precursor protein (APP), and dual-specificity tyrosine-(Y)-phosphorylation regulated kinase 1A (DYRK1A) which phosphorylates Tau at Thr212 *in vitro*. Here, we describe a potential mechanism for the development of AD pathology in DS. First, we show that Dyrk1A phosphorylates APP at Thr668 and Tau at Ser202 and Ser404 *in vitro*. We then demonstrate that the amounts of phospho-Thr668-APP, phospho-Ser202-Tau, phospho-Thr212-Tau, and phospho-Ser404-Tau are increased in the brains of transgenic mice that overexpress human DYRK1A. Furthermore DYRK1A transgenic mouse brains also contain elevated amounts of b-amyloid. Taken together, these results provide the first *in vivo* evidence of a physiological role for DYRK1A in APP processing and Tau hyperphosphorylation, suggesting that DYRK1A might represent a therapeutic target for early-onset of AD in DS patients. This work was supported by IBST Grant 2006 and by a Korea Research Foundation Grant funded by the Korean Government (MOEHRD) (KRF-2006-331-C00189).