Aerobic metabolism

Aerobic metabolism is the creation of energy through the combustion of carbohydrates, amino acids and fats in the presence of oxygen. The body burns sugars, fats, and proteins. The only byproducts of aerobic metabolism of carbohydrates are carbon dioxide and water, which your body disposes of by breathing, sweating and urinating.

In the aerobic metabolic process, the human body uses a molecule of glucose to produce 36 ATP molecules.

ATP is what fuels the muscles. Anaerobic metabolism, which is used for vigorous muscle contraction, only produces 2 ATP molecules per glucose molecule, so it is much less efficient.

Acute brain injury is associated with depressed aerobic metabolism. Below a critical mitochondrial pO2 cytochrome c oxidase, the terminal electron acceptor in the mitochondrial respiratory chain, fails to sustain oxidative phosphorylation. After acute brain injury, this ischaemic threshold might be shifted into apparently normal levels of tissue oxygenation. We investigated the oxygen dependency of aerobic metabolism in 16 acutely brain-injured patients using a 120-min normobaric hyperoxia challenge in the acute phase (24-72 h) post-injury and multimodal neuromonitoring, including transcranial Doppler ultrasound-measured cerebral blood flow velocity, cerebral microdialysis-derived lactate-pyruvate ratio (LPR), brain tissue pO2 (pbrO2), and tissue oxygenation index and cytochrome c oxidase oxidation state (oxCCO) measured using broadband spectroscopy. Increased inspired oxygen resulted in increased pbrO2 [ΔpbrO2 30.9 mmHg $p < 0.001$], reduced LPR [ΔLPR -3.07 $p = 0.015$], and increased cytochrome c oxidase (CCO) oxidation (Δ[oxCCO] + 0.32 µM $p < 0.001$) which persisted on return-to-baseline (Δ[oxCCO] + 0.22 µM, $p < 0.01$), accompanied by a 7.5% increase in estimated cerebral metabolic rate for oxygen ($p = 0.038$). Our results are consistent with an improvement in cellular redox state, suggesting oxygen-limited metabolism above recognised ischaemic pbrO2 thresholds. Diffusion limitation or mitochondrial inhibition might explain these findings. Further investigation is warranted to establish optimal oxygenation to sustain aerobic metabolism after acute brain injury [1)].

1)

Ghosh A, Highton D, Kolyva C, Tachtsidis I, Elwell CE, Smith M. Hyperoxia results in increased aerobic metabolism following acute brain injury. J Cereb Blood Flow Metab. 2016 Nov 11. pii: 0271678×16679171. [Epub ahead of print] PubMed PMID: 27837190.