

Genome and cell size as factors determining metabolic rate scaling

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Explanations of allometric scaling of metabolic rate (MR) are subject of hot debate, which generally centers around two alternative hypotheses: 1) ubiquity of the $\frac{3}{4}$ scaling exponent, derived from the presumed, fractal-like structure of supplying systems. It also predicts size-invariance of metabolically active cells (e.g. erythrocytes) and 2) dependence of MR scaling on the cell size (CS), mediated by nucleus/genome size variation. To test both hypotheses, we measured CS and MR in two animal models: polyploid fish of the *Cobitis taenia* hybrid complex and laboratory mice subject to artificial, divergent selection on basal metabolic rate (BMR). In both models, BMR/SMR scaled to body mass with exponents higher than the $\frac{3}{4}$ value, while cell sizes scaled with body mass with an allometric exponents significantly different from 0. In *C. taenia* we demonstrated a positive correlation of cell size with genome/nucleus size and an inverse relation between those traits and SMR. In mice, we found a negative correlation between BMR and erythrocyte and skin cell size, and a positive correlation between BMR and hepatocytes, duodenum epithelium and kidney cells. Our results therefore provide partial empirical support for CS and genome/nucleus size being important determinants of MR variation and consequently, its allometric scaling.