

# Temporal gene expression analysis of hypoxia in human muscle cell

Masaki S. Morioka<sup>1,\*</sup>, Ryosuke R. Ishiwata<sup>2</sup>, Soichi Ogishima<sup>2</sup>, Satoru Oshiro<sup>3</sup>  
and Hiroshi Tanaka<sup>1,2</sup>

1. Dept. of Computational Biology, School of Biomedical Science, Tokyo Medical and Dental University, Tokyo, Japan
  2. Dept. of Bioinformatics, Medical Research Institute, Tokyo Medical and Dental University, Tokyo, Japan
  3. Dept. of Health Science, Faculty of Sports and Health Sciences, Daito Bunka university, Saitama, Japan
- \*email: morioka@bioinfo.tmd.ac.jp

Hypoxic stress affects various biological processes such as glycolysis, angiogenesis, cancer proliferation and apoptosis. Hypoxia inducible factors (HIFs) are key factors regulating gene expressions in those processes, which respond to cellular and tissue hypoxia. However, it has not been revealed how HIFs and other hypoxia responsible factors cooperatively coordinate various processes which comprise hypoxic response by gene regulation during hypoxia.

Here, we examined time-course gene expression responded to hypoxia in human muscle cell (SJCRH30, RC13) by using microarray analysis. Cultured human muscle cell were exposed for 0, 6, 12 and 24 hours to 1% oxygen pressure, followed by processing for analysis on human genome DNA microarray.

By using Jonckheere-Terpstra trend test, 199 and eight genes showed upregulated and downregulated trends, respectively. To assess functional roles of these genes in biological pathways, we conducted GO analysis and KEGG pathway mapping. Most of up/down-regulated genes were classified into metabolism, biosynthesis, chemotaxis, and external stimulus response. These results showed hypoxia induced both glycolytic genes and negative regulatory genes for glycogenesis.

We will show representation of temporal gene expression on these glycolysis pathways responded to hypoxia in order to elucidate the transcriptional regulation by 3D hierarchical and dynamical network visualization tool.