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Celebrating metabolism from cells to organisms

This month, we host a free, virtual Nature Conference with *Nature Metabolism* and *Nature Reviews Molecular Cell Biology*, 'Metabolic Communication Across Biological Scales', and highlight recent articles that enrich our understanding of cellular metabolism in health and disease.

Metabolic reactions provide the foundation to understand how cells function and communicate. So much of cell biology relies on metabolism — within a cell, the metabolic regulation and modification of organic compounds affects their molecular roles and defines the composition and function of subcellular compartments. Beyond the cell, metabolites contribute to cell behaviour and cell–cell communication in tissues, all converging to ensure the metabolic homeostasis of an organism.

To recognize the importance of metabolism in cell biology, we are delighted to present a free, virtual Nature Conference together with *Nature Metabolism* and *Nature Reviews Molecular Cell Biology*. The 'Metabolic Communication Across Biological Scales' meeting bridges perspectives from cellular metabolism to systems biology. Experts from diverse fields discuss how metabolic functions are regulated and influence cells in health and disease. Session topics include lipid and organelle metabolism, metabolic crosstalk within tissues and organismal metabolism. To accompany the meeting, we invite you to browse a Collection of recent articles published in *Nature Cell Biology*, *Nature Metabolism* and *Nature Reviews Molecular Cell Biology* that cover cellular to systemic metabolic regulation.

Cell biologists are increasingly profiling the metabolome of cells with high-throughput, quantitative analyses. These snapshots of dynamic metabolic processes that occur across compartments can be exploited by transparent reporting of analyses and tools that enable data re-use. The community is currently developing and systematically adopting standards for the deposition and reporting of such data. In their Comment, Ethan Stancliffe and Gary J. Patti offer quick tips for re-using metabolomics data, which we hope are helpful for the growing number of cell biologists interested in this data type.

Metabolic communication lies at the intersection of many fields, including immunology and cancer cell biology. An interesting example of this intersection occurs in the tumour microenvironment (TME), where the metabolic milieu is shaped by and influences both immune and tumour cells. In a Review in this issue, Chin-Hsien Tsai, Ping-Chih Ho and colleagues describe recent insights in the metabolic crosstalk between cancer and immune cells and how this affects immune surveillance and anti-tumour immune responses. They cover mechanisms for metabolism-mediated immune escape and the influence of nutrient limitation in the TME. The authors further explore potential strategies to target tumour-associated metabolic pathways to elicit immune surveillance responses and prevent cancer progression.

In a new Article, Daqian Xu, Zhimin Lu and colleagues examine how metabolic enzymes contribute to cancer progression. The enzyme fructose-1,6-bisphosphatase 1 (FBP1) contributes to gluconeogenesis through hydrolysis of fructose-1,6-bisphosphate to fructose-6-phosphate. The authors describe a surprising moonlighting function for FBP1 in the nucleus. They find that a change in the protein binding partners of FBP1 is key to explaining the subcellular localization of FBP1 and its differential effect in tumour and non-tumour cells. After glucose deprivation in hepatocytes, FBP1 is phosphorylated and translocates to the nucleus, where it interacts with the transcriptional regulator PPARα. FBP1 dephosphorylates histone H3 at Thr11, dampening PPARα-mediated expression of genes related to β-oxidation. In hepatocellular carcinoma cells, FBP1 instead interacts with *O*-linked *N*-acetylglucosamine (GlcNAc) transferase. With enhanced FBP1 *O*-GlcNAcylation, FBP1 phosphorylation and nuclear translocation are decreased. The authors show that in the absence of the restraining role of FBP1 in transcription, enhanced β-oxidation and increased energy production support tumour growth in mice. These findings are discussed in a News & Views article by Scott A. Berger and Arminja N. Kettenbach.

Cellular metabolic pathways can shape the behaviour of cells, and in turn, cell populations can contribute to an organism's metabolic regulation. Immune cells provide an example for both these properties. Cellular metabolism is important during immune cell specialization and has emerging roles in thymocyte development. Although phosphatase and tensin homolog (*PTEN*) is a tumour suppressor gene frequently mutated in cancer, its role in lymphopoiesis and tissue homeostasis is less well understood. In an Article published in this issue, Kai Yang, Hongbo Chi and co-authors show that loss of PTEN results in accumulation of type-17 innate-like T cells in the thymus. IL-23 is required for the accumulation of these cells in the context of PTEN deficiency. The authors find that the effects of PTEN loss depend upon mTOR, FOXO1 and IL-23–STAT3 signalling to shape type-17 innate-like cell programming. These studies reveal the importance of PTEN regulation in the development of IL-17-producing thymocytes through the control of metabolic programs. In their News & Views article, Nikolaos Patsoukis and Vassiliki A. Boussiotis highlight this work and its potential implications in the treatment of autoimmune and inflammatory diseases.

We are thrilled to celebrate metabolic research throughout this month and across our content. We'll continue to support studies of metabolic communication across organelles, cells and tissues and look forward to future discoveries. We thank our readers, authors and reviewers for their contributions to advancing our knowledge of cellular and organismal metabolism. Thank you also to our Nature Conference speakers and co-organisers for creating an exciting forum for the field to learn about and discuss metabolic signalling across biological scales.

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