

# Food or nutrient restriction offers insight into cancer prevention and metabolic disease

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Could limiting food intake be a valid treatment strategy for certain types of cancers? New research in *The FASEB Journal* suggests "maybe." In a report appearing online in *The FASEB Journal*, scientists show that food restriction increases levels of a tumor-suppressing molecule, called p53, in both hepatocytes grown in the laboratory and in mouse liver. In addition, this research gives evidence that p53-induction is required for the fasting-induced adaptation of nutrient metabolism.

"Metabolic diseases and cancer are major health burdens in our societies," said Andreas Prokesch, Ph.D., study author and Associate Professor at the Institute of Cell Biology, Histology, and Embryology at the Medical University of Graz in Graz, Austria. "We hope that our study will yield new therapeutic concepts to fight these diseases."

To make their discovery, Prokesch and colleagues first determined the abundance of [p53 protein](#) in the livers of mice with free access to food compared to mice that had food withheld for 24 hours. They found that there was a strong induction of p53 protein in the livers of the mice that had undergone food withdrawal. This p53 accumulation was also observed in cultured mouse or human hepatocytes upon the removal of nutrients from their culture media. The researchers then compared blood glucose and amino acid utilization in the livers of [mice](#) with or without acute inactivation of the [p53 gene](#). Mice that lacked p53 in the liver showed reduced [blood glucose levels](#) and altered hepatic amino acid metabolism during starvation.

"Connections between nutrients and tumor suppression have long been known, but this study adds a sharp focal point," said Thoru Pederson, Ph.D., Editor-in-Chief of *The FASEB Journal*. "The altered amino acid metabolism observed is especially provocative, as that is a rapidly emerging area of interest in tumor biochemistry."

**More information:** A. Prokesch et al, Liver p53 is stabilized upon starvation and required for amino acid catabolism and gluconeogenesis, *The FASEB Journal* (2016). [DOI: 10.1096/fj.201600845R](https://doi.org/10.1096/fj.201600845R)

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