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Editorial

Nutrition Sensing: How Nutrients Influence Cellular Functioning

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From saving hunger, preventing diseases, to focusing on how substances inside human body are transformed (metabolic pathways and biochemical steps), the science of Nutrition has come a long way as technology in molecular biology, biochemistry, and genetics getting more advanced.

The human body is known to be a well-designed, energy saving and recycling, and tightly controlled machine. For example, when people try to lose weight by decreasing foods intake, eventually, the weight loss will slow down. And the human body will try to conserve energy that body takes in by transforming it into "Fat". So, how human body knows when, what, and how to adjust itself after food is taken in, this is truly a big question. Taking advantage of an opportunity of advanced science and technology, the science of the effect of genetic variation on dietary response and the role of nutrients and bioactive food compounds in gene expression is formed. It's called "Nutrigenetics and Nutrigenomics" [1-5].

Nutrition sensing is the ability of all organisms to sense and respond the nutrients required to generate energy and the building blocks of cells in their environment (coordinate growth and development). Different metabolic pathways can detect intracellular and extracellular levels of macronutrients and substitute metabolites. During food abundance, via hormonal signals, nutrient sensing pathways engage anabolism and storage; and in food insufficiency, nutrient sensing pathways activate homeostatic mechanisms to mobilize the internal store.

The sensing of a particular nutrient may involve the direct binding of the sensed molecule to the sensor, such as GPR40, GPR120, and CD36 to fatty acids [6-9]; SCAP (SREBP1 cleavage activating protein) and HMGCR to cholesterol [9-11]; GCN2 (kinase general control nonderepresible 2), mTORC1, and T1R1+T1R3 (oral taste receptors) to amino acids [9,12-15]; and glucokinase (GCK), glucose transporter GLUT2 (SLC2A2), insulin, and T1R2+T1R3 (oral taste receptors) to glucose [9,16-18]; AMPK and mTORC1 to autophagy as an internal source of stored nutrients under conditions of nutrient limitation [9,19-21].

Furthermore, in times of excess oxidative stress, ER stress, and mitochondrial dysfunction all lead to metabolic activities stress and cell damage. Over time, the nutrition sensing process will become damaged and incapable to function properly. The deregulated nutrient sensing pathways can dramatically alter metabolism and drastic changes in metabolism often result in obesity, diabetes, other metabolic diseases, and aging [9,22,23]. This is an important topic to the prevention of metabolic diseases associated with aging. It could be explored to see how this ability to sense nutrients becomes "dysregulated" with aging and how nutrition/food science and perhaps exercise (particularly with respect to antioxidant defenses) might help prevent deregulated nutrient sensing. Regardless of intense research, our understanding of nutrient sensing mechanisms is still a long way to go.

Bibliography

1. Fenech Michael., *et al*. "Nutrigenetics and nutrigenomics: viewpoints on the current status and applications in nutrition research and practice". *Journal of Nutrigenetics and Nutrigenomics* 4.2 (2011): 69-89.

2. Simopoulos Artemis P. "Nutrigenetics/nutrigenomics". *Annual Review of Public Health* 31 (2010): 53-68.

3. Corella Dolores and Jose M Ordovas. "Nutrigenomics in cardiovascular medicine". *Circulation: Cardiovascular Genetics* 2.6 (2009): 637-651.

4. Ferguson Lynnette R. "Nutrigenomics approaches to functional foods". *Journal of the American Dietetic Association* 109.3 (2009): 452-458.

5. Kaput Jim. "Nutrigenomics research for personalized nutrition and medicine". *Current Opinion in Biotechnology* 19.2 (2008): 110-120.

6. Itoh Yasuaki., *et al*. "Free fatty acids regulate insulin secretion from pancreatic β cells through GPR40". *Nature* 422.6928 (2003): 173-176.

7. Talukdar Saswata., *et al*. "GPR120 is an omega-3 fatty acid receptor mediating potent anti-inflammatory and insulin-sensitizing effects". *Cell* 142.5 (2010): 687-698.

8. Ichimura Atsuhiko., *et al*. "Dysfunction of lipid sensor GPR120 leads to obesity in both mouse and human". *Nature* 483.7389 (2012): 350-354.

9. Efeyan Alejo., *et al*. "Nutrient-sensing mechanisms and pathways". *Nature* 517.7534 (2015): 302-310.

10. Feramisco Jamison D., *et al*. "Intramembrane aspartic acid in SCAP protein governs cholesterol-induced conformational change". *Proceedings of the National Academy of Sciences of the United States of America* 102.9 (2005): 3242-3247.

11. Sever, Navdar., *et al*. "Accelerated degradation of HMG CoA reductase mediated by binding of insig-1 to its sterol-sensing domain". *Molecular Cell* 11.1 (2003): 25-33.

12. Dong Jinsheng., *et al*. "Uncharged tRNA activates GCN2 by displacing the protein kinase moiety from a bipartite tRNA-binding domain". *Molecular Cell* 6.2 (2000): 269-279.

13. Laplante Mathieu and David M Sabatini. "mTOR signaling in growth control and disease". *Cell* 149.2 (2012): 274-293.

14. Bachmanov Alexander A and Gary K Beauchamp. "Taste receptor genes". *Annual Review of Nutrition* 27 (2007): 389-414.

15. Damak Sami., *et al*. "Detection of sweet and umami taste in the absence of taste receptor T1r3". *Science* 301.5634 (2003): 850-853.

16. Thorens Bernard and Mike Mueckler. "Glucose transporters in the 21st Century". *American Journal of Physiology-Endocrinology and Metabolism* 298.2 (2010): E141-E145.

17. Zhang Feng., *et al*. "Molecular mechanism for the umami taste synergism". *Proceedings of the National Academy of Sciences* 105.52 (2008): 20930-20934.

18. Nelson Greg., *et al*. "Mammalian sweet taste receptors". *Cell* 106.3 (2001): 381-390.

19. Egan Daniel F., *et al*. "Phosphorylation of ULK1 (hATG1) by AMP-activated protein kinase connects energy sensing to mitophagy". *Science* 331.6016 (2011): 456-461.

20. Kim Joungmok., *et al*. "AMPK and mTOR regulate autophagy through direct phosphorylation of Ulk1". *Nature Cell Biology* 13.2 (2011): 132-141.

21. Naito Takako., *et al*. "Differential contribution of insulin and amino acids to the mTORC1-autophagy pathway in the liver and muscle". *Journal of Biological Chemistry* 288.29 (2013): 21074-21081.

22. López-Otín Carlos., *et al*. "The hallmarks of aging". *Cell* 153.6 (2013): 1194-1217.

23. Ortega-Molina Ana., *et al*. "Pten positively regulates brown adipose function, energy expenditure, and longevity". *Cell Metabolism* 15.3 (2012): 382-394.

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