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 extracellu-lar 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 HMGR to cholesterol [9-11]; GCN2 (kinase general control non-derepressible 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 ex- plored 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


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