Nutritional Epigenetics: A New Level of Complexity About Impact of Diet on Health

Laura Bordoni*

School of Advanced Studies, University of Camerino, Via Gentile III da Varano, Camerino, MC, Italy

*Corresponding Author: Laura Bordoni, School of Advanced Studies, University of Camerino, Via Gentile III da Varano, Camerino, MC, Italy.

Received: February 21, 2017; Published: March 11, 2017

Among the wide range of activities for which many nutrients are known in their role on prevention and mitigation of various diseases, epigenetic effects recently acquired an emerging importance [1]. The term "epigenetics" refers to potentially reversible and tissue specific modifications in gene expression caused by DNA methylation, histone tail methylation and acetylation, small non-coding miRNAs [2].

Whether it is still debated if the epigenetic states of histones can be inherited during cell division or not, it has been demonstrated that DNA methylation constitutes a firm epigenetic mark that can be transmitted through DNA replication. With the exception of parentally imprinted genes, the genome is demethylated in early embryos and it is generally thought that patterns of DNA methylation induced during early development are stable throughout the life course [3]. Even if these marks are erased and set-up during gametogenesis and development, they can lead to a memorization of early environment and induce long-term alteration of cell and tissue functions, which will influence the predisposition to future diseases [4,5].

Given that, there is substantial evidence to support the hypothesis that different epigenetic mechanisms could, at least in part, explain the way through which environmental factors, including nutrition, particularly in specific periods of the life (such as maternal and neonatal diet), are able to affect the susceptibility to develop diseases in adulthood [5-7]. Moreover, epigenetic plasticity seems to be extended also beyond early development, including life course stages associated with rapid physiological change such as puberty and aging. Thus, if the epigenome can be altered by nutritional exposures leading to changes in cell function, a modulation of these epigenotypes through diet or specific nutrients may also be able to prevent diseases and maintain health [1,6].

This dual nature of epigenetics between stability and plasticity renders nutrigenomics research particularly fascinating and the development of epigenetic markers of disease risk should represent, in the future years, a hot topic in nutrition for its substantial potential for public health benefits. With respect to this last point, histone modifications are considered more dynamic than DNA methylation which is stable and relatively easier to measure; thus, to date, most studies on the interaction between epigenetic regulation of genes and nutrition have focused on DNA methylation, as it seems to be the best candidate as epigenetic marker [6].

Nutrition influences patterns of DNA methylation in several different possible ways and these multiple mechanisms are mutually compatible and may operate together in time, enriching the complexity of this regulative pathway [8]. In particular, nutrition provides substrates necessary for proper DNA methylation, cofactors that modulate enzymatic activity of DNA methyltransferases (DNMTs) and can regulate activity of the enzymes involved in the one-carbon cycle.

Recent researches indicate that secondary plant metabolites, such us polyphenols, can exert beneficial effects via modulation of gene expression, chromatin remodeling through modulation of histone deacetylases (HDAC) and DNMTs activity. Epigallocatechin-3-gallate (EGCG), polyphenol in green tea, or the genistein present in soybean showed a competitive inhibition of DNMTs activity, while myricetin has been shown to be able to decrease DNA methylation by inhibiting SssI DNMT [8]. Epigenetic effects have been shown also for other dietary components such as butyrate, sulforaphane and curcumin [1]. Moreover, all the precursors of S-Adenosylmethionine (SAM), the

Citation: Laura Bordoni. "Nutritional Epigenetics: A New Level of Complexity About Impact of Diet on Health". *EC Nutrition* 7.5 (2017): 185-186.

universal methyl-donor for DNA methyl- transferases, such as methionine, folate, choline, betaine and vitamins B₂, B₆ and B₁₂ can contribute to the net synthesis of SAM influencing DNA methylation patterns [8].

These are just few examples about the growing body of literature that underline the important role that a diet containing epigenetically active food compounds can play in numerous aspects of health. Moreover not just natural food components but also several classes of pesticides (including endocrine disruptors, persistent organic pollutants, arsenic, several herbicides and insecticides) have been shown to modify epigenetic marks [9].

Thus, it is almost established that nutrition, in several different ways, can have long lasting effects on human health [5] and despite of the early stage of knowledge of these topics and the technological and intellectual challenges, epigenetics represents an emerging and promising research field for advances in nutrition.

Bibliography

- 1. Remely M., *et al.* "Therapeutic perspectives of epigenetically active nutrients". *British Journal of Pharmacology* 172.11 (2015): 2756-2768.
- Clarissa G. "Cancer Chemoprevention and Nutri-Epigenetics: State of the Art and Future Challenges". *Topics in Current Chemistry* 329 (2013): 73-132.
- 3. Burdge GC and Lillycrop KA. "Bridging the gap between epigenetics research and nutritional public health interventions". *Genome Medicine* 2.11 (2010): 80.
- 4. Panchenko PE, *et al.* "Epigenetics and Nutrition: maternal nutrition impacts on placental development and health of offspring". *Biologie Aujourd'hui* 209.2 (2015): 175-187.
- 5. Mochizuki K., *et al.* "Relationship between epigenetic regulation, dietary habits, and the Developmental Origins of Health and Disease theory". *Congenital Anomalies (Kyoto)* (2017).
- 6. Burdge GC., *et al.* "Epigenetics: are there implications for personalised nutrition?" Current Opinion in Clinical *Nutrition and Metabolic Care* 15.5 (2012): 442-447.
- 7. Canani RB., et al. "Epigenetic mechanisms elicited by nutrition in early life". Nutrition Research Reviews 24.2 (2011): 198-205.
- 8. Zhang N. "Epigenetic modulation of DNA methylation by nutrition and its mechanisms in animals". *Animal Nutrition* 1 (2015): 144-151.
- 9. Collotta M., et al. "Epigenetics and pesticides". Toxicology 307 (2013): 35-41.

Volume 7 Issue 5 March 2017 © All rights reserved by Laura Bordoni. 186