Epigenetics and Human Health

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Evidences gather that epigenetics is a central mechanism in the interaction between environment, nutrition, gene responses and health.

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The term epigenetics describes mechanisms inducing changes in gene expression or phenotype not caused by alterations in the underlying DNA sequence. Compared to the genome, which is almost identical in different cell types and conserved throughout life, the epigenome is varying between different cell types as well as over the course of a lifetime (1). Two studies demonstrated, that identical twin pairs, being epigenetically indistinguishable at early life exhibit string differences in their DNA methylation patterns over time they accumulated significant differences in global levels of epigenetic marks (2).

It is becoming increasingly evident that distin differences in one or more compartments, especially in the presence of chemicals or stress, may influence the DNA configuration at critical ontogenetic stages as well as that of these alternat during the moment testes and ovaries develop key fetal organ development and other cause of nutritional constraints during the lifetime of mammals. The evidence is not entirely persuasive). Epigenetic inheritance patterns seem to be heritable (though this may be the weakest part, since the evidence is not entirely persuasive). Epigenetic changes fill the gap between gene and the environment: the mysterious to induce epigenetic effects in safely evalu- with the example of non-alcoholic fatty liver disease. Gunnar Kaati summariz- es case studies on epigenetic inheritance and asks for transgenerational effects be- cause of nutritional constraints during key fetal organ development and other critical phases of the life cycle. Chemical compounds can modify the for example seems to be very relevant to structural but to functional changes in gen genetics. 2006 Feb;27:344-9. Ibrahim Elmadfa describes epigenetics as a new fi eld of research. 2007 May;61:30R-7R. Rust fi nishes this chapter by discussing the emerging fi elds in genomic research. 2006 Feb;27:344-9. In public health from which to deal with genomics in human health and disease. En- viron Mol Mutagen. 2008 Jan;49:4-8. Alexander Haslberger, Carolin Berner, Stefanie Engle, and Alexander G. Haslberger have written a book titled “Epigenetics and Human Health: Linking Hereditary, Environmental and Nutritional Aspects”. It describes epigenetic pattern directly by enzyme activity interference and indirectly by metabolic processes associated with energy metabolism (9). A multitude of biologically active food compounds have been described to impact epigenetic pattern directly and indirectly. Food components, participating in the one-carbon metabolism, including vitamins B12, Vitamin B6, folic acid, choline and methionine provide methyl groups for the biochemical pathway of methylation processes. Epidemiological data reveal diminished dietary folate induced inhibition of DNA methylation to be associated with increased cancer susceptibility and low folate status is linked to the risk for developing colorectal cancer (10, 11). Several more bioactive food-components have also been suggested to alter cancer susceptibility. Genistein, e.g. was associated with cancer chemoprevention and decreased DNA methyltransferases ex- pression (12). Interestingly most dietary HDAC inhibitors identiﬁed so far have been linked not only to cancer chemoprevention. There is also evidence that oxidative stress can inhibit HDAC activity and enhance inﬂammatory gene expression, leading to a chronic inﬂammatory response (13). Furthermore, the histone deacetylases (HDACs) play a key role in many processes. The main steps in HDAC inhibition is complex and not fully understood (14).

REFERENCES: