Epigenetics

The term epigenetics seemingly has gained some traction in the nutritional field. Epigenetics is different and distinct from epigenesis, which describes incremental differentiation in the embryo leading to evolving cellular complexity. Strictly speaking epigenetics, as an umbrella concept, this term connotes epigenetic inheritance, reversible changes in the gene function without affecting the hereditary repertoire. These changes may occur spontaneously or in response to environmental stimuli, such as diet. Epigenetics is most commonly evoked with reference to the effect of the diet on human genetic endowment and its potential modulation by dietary factors.

Epigenetics has come to a head lately because of the possible role of diet in health and disease. Briefly, the increase in the incidence of chronic diseases can be attributed to changes in the dietary staple throughout the world over the past two to three hundred years. Due to the fact that the human genetic composite is not equipped to take these changes in stride. A measure of debate revolves around the so-called "Paleolithic" diet, the deviations from which are attributed to have caused almost alarming rise in diabetes, heart disease and cancer, among others chronic illnesses.

The constancy—indeed, even the immutability—of the human gene pool vis a vis the environmental stimuli is almost an article of faith, at least in some circles. The evidence, however, of any genetic changes adapted to the specific dietary ingredients awaits rigorous corroboration. This has not deterred some of the more vocal exponents of such a change from coining the term "epigenetic medicine." While some lines of investigations point to the possibility that some transient changes in gene expression might well be conceivable, to tout epigenetic medicine as an almost accomplished fact is premature to say the least.

The most common example given of nutrients to affect epigenetic changes has been the methylation agents, such as niacin, folic acid, and vitamin B12. The results of the usually cited studies are tenuous at best. Whereas dietary deficiency of, say, folic acid causes neural tube defects in children, and replenishment of this deficiency prevents it, this does not necessarily mean that a genetic change has taken place. It should be emphasized that, to the extent that a nutritive has an effect of gene expression, it has to be, first and foremost, inducible change, which is likely to be transient. As such, it is too early to postulate that nutritives change the patterns of gene expression. Equally importantly, the singular lesson of modern molecular biology is that changes in a given set of genes had best be interpreted in the chromosomal context, and not in isolation. That is, the era of epigenetic medicine is yet to usher, if it is at all likely to happen.

This contention rests on the premise that chronic diseases are a consequence of the post-Paleolithic diet. Quite to the contrary, archeological excavations have shown that many of the ills afflicting modern humans were prevalent in more ancient times when Paleolithic diet was the norm. This is not to suggest, however, that a nutritive regimen cannot exercise indirect effect on gene expression. For example, Gingko biloba extract can induce gene expression in the hippocampus, the area of the brain critical in memory formation. Likewise, pro-inflammatory cytokines levels may be regulated by the inhibition of NF-KB, a transcription factor involved in the inflammatory cascade, by sundry proteases used systemically. It should be noted, however, that such changes are not a direct result of gene modulation; rather, such changes may well be secondary manifestations of intracellular signaling not necessarily triggered by certain nutritives. This distinction, along with the issues of inducibility and transiency, is crucial in the debate on "epigenetic medicine."

The issue arguably is more of an "ideal" diet for humans. In and of itself, it may not be a goal easy to define, let alone achieve. This is poignantly driven home by a study on longevity published in September 2006. The study showed that mortality is a function of diet for humans. In and of itself, it may not be a goal easy to define, let alone achieve. This is poignantly driven home by a study on longevity published in September 2006. The study showed that mortality is a function of diet for humans. In and of itself, it may not be a goal easy to define, let alone achieve. This is poignantly driven home by a study on longevity published in September 2006. The study showed that mortality is a function of diet for humans. In and of itself, it may not be a goal easy to define, let alone achieve. This is poignantly driven home by a study on longevity published in September 2006. The study showed that mortality is a function of...
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