The Epiphany of Epigenetics

It is common scientific knowledge that transcribing and translating the instructions encoded in the DNA of a gene manufactures proteins to contribute to an organism's genotype and phenotype. Astoundingly, a phenomenon coined 'epigenetics' transcends this entire process. Epigenetics refers to heritable alterations in gene expression that do not necessitate changes in the genetic code, however counterintuitive that might seem ("Epigenetics: Fundamentals," 2015). Although the genome dictates a cell's morphology, cells further differentiate by expressing or silencing certain genes under the guidance of an 'epigenome' of biochemical markers, taking the meaning of "birthmarks" to a whole new level. Epigenetics manifests itself through three processes that affect the genome: gene expression, epigenetic inheritance, and imprinting.

Gene expression is influenced by three different epigenetic mechanisms that allow cells to have different functions, although they are encrypted with the same DNA sequence (Simmons, 2008). The first mechanism is DNA methylation, in which methyl groups consisting of carbon and hydrogen are added to CpG sites on the double helix by enzymes called DNA methyltransferases. Methylation obstructs the chemical signals that would initiate transcription, thus suppressing the gene. DNA is tightly spooled around proteins called histones to form chromatin. The second mechanism of histone modification epigenetically loosens or condenses this packaging, dictating whether DNA will be transcribed or not. The third mechanism is modulated by antisense and noncoding RNAs, which can switch off genes by forming heterochromatin or by propagating DNA methylation. Endogenous and exogenous factors during prenatal and early postnatal life such as diet, pollutants, exercise, hormones, and toxins can influence epigenetic modifications to DNA and thus, gene expression ("The Epigenome Learns From," 2015). Environmental factors, like poor lifestyle choices, can supersede gene expression and results in severe health impacts that perpetuate through multiple generations and dramatically alter evolution. Errors in epigenetic modifications to DNA can cause atypical expression or silencing of genes that are linked to carcinogenesis, chromosomal abnormalities, and mental disorders.

During reprogramming in embryonic development, differentiated somatic cells evolve into pluripotent cells by the attenuation of epigenetic markings so that offspring inherit DNA without epigenetic markers (Cyranoski, 2014). However, some epigenetic markings avoid deletion and affect gene expression in progeny. This ability of epigenetic markers to circumvent reprogramming allows for 'transgenerational epigenetic inheritance' (Saini, 2014). Therefore, offspring do not inherit a 'tabula rasa' of the genome but rather; parental epigenetic markers influence its expression.

Imprinting is a gametogenic process that causes differential expression of genes based on parent (Nussbaum, McInnes, Willard, & Hamosh, 2007, p. [77]). Through epigenetic mechanisms, either the maternal or paternal allele of a gene is imprinted, or suppressed. This process continues into adulthood via cell division so that only the maternal or paternal copy of the gene is expressed. In the Prader-Willi and Angelman disorders, certain genes on chromosome 15 are subject to imprinting of either the maternal or paternal allele. When the other allele is missing, the gene is not expressed normally, resulting in anomalous phenotypes.

One glaring example of the phenomenon of epigenetics is the Dutch Famine. People with prenatal exposure to the Dutch Famine from 1944-1945 developed epigenomic changes that resulted in hypomethylation of the maternally imprinted insulin-like growth factor II gene, relative to their unexposed, same-sex siblings, which was associated with higher predisposition rates to schizophrenia and coronary disease later in life (Heijmans et al., 2008). This example of epigenetics singularly depicts that early embryonic development is a critical time for long-lasting epigenetic alterations and the environment has a blatant effect on epigenetic markers.

So is it nature or nurture? A trick question- epigenetics seems to combine the two. The scientific world formerly believed that DNA is the most fundamental structure that cements our destiny and our genetic code far precedes our identity. This, however, is disproved by the epigenome, which provides our genes with plasticity. Epigenetics flatly contradicts Darwinism, a well-accepted evolutionary theory stating pedigree is purely a function of chance events like natural selection and randomized mutation (Watters, 2006). The epigenome is adaptive to dynamic environmental conditions, shedding light on the overlap of gene regulation and the environment.

Sequencing the human genome was only half the battle, but mapping the epigenome is required for complete understanding of the human body. By being as stubborn as some epigenetic tags that have instigated a paradigm shift in genetics, we can redefine our thinking of inheritance and propel progeny towards healthy existences. If we play our cards right, we can correct past misconceptions, capitalize on present knowledge, and transform the future.

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