<Further Development 3.6>

DNA Methylation and Genomic Imprinting

DNA methylation has explained at least one very puzzling phenomenon, that of genomic imprinting (Ferguson-Smith 2011). It is usually assumed that the genes one inherits from one's father and from one's mother are equivalent. In fact, the basis for Mendelian ratios (and the Punnett square analyses used to teach them) is that it does not matter whether the genes came from the sperm or from the egg. But if genes in the eggs and sperm are methylated differently, it can matter. In mammals, there are about 100 genes for which it matters (International Human Epigenome Consortium).[1] In these cases, the chromosomes from the male and the female are not equivalent; only the sperm-derived or only the egg-derived allele of the gene is expressed. Thus, a severe or lethal condition may arise if a mutant allele is derived from one parent, but that same mutant allele will have no deleterious effects if inherited from the other parent. In some of these cases, the nonfunctioning gene has been rendered inactive by DNA methylation. The methyl groups are placed on the DNA during spermatogenesis and oogenesis by a series of enzymes that first take the existing methyl groups off the chromatin and then place new sex-specific ones on the DNA (Ciccone et al. 2009; Gu et al. 2011). (See Further Development 3.5, Mechanisms of DNA Methylation during Genomic Imprinting, Further Development 3.7, Poised Chromatin, Further Development 3.8, Chromatin Diminution, and Further Development 3.9, The Nuclear Envelope's Role in Gene Regulation, all online.)

---

[1] A list of imprinted mouse genes is maintained at www.mousebook.org/all-chromosomes-imprinting-chromosome-map.