Journal of Clinical Epigenetics
ISSN: 2472-1158

Open access Commentary

Growth Restriction and Genomic Imprinted Phenotypes Support the Concept of Imprinted Genetics

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DESCRIPTION

In human Glioblastoma, the presence of a small population of cells with stem cell properties, termed Glioma Stem Cells (GSCs), has been described. These cells may be GBM and are involved in tumor development. However, it remains to be determined whether GSCs derive from normal Neural Stem Cells (NSCs) as a result of genetic and epigenetic alterations and/or somatic dedifferentiation. Genomic imprinting is an epigenetic marking process that directs gene expression according to parental origin. Dysregulation of imprinting patterns or loss of genomic imprinting has been described in a variety of tumors, including GBM, and is one of the earliest and most common events occurring in human cancer. Here, we collected current knowledge on the role of imprinted genes in the normal function of NPCs and how the imprinting process is altered in human GBM. We also examine alterations in specific imprinted loci that may be involved in tumor development. Understanding the mechanistic similarities in the regulation of genomic imprinting between normal her NSCs and her GBM cells will help identify possible molecular actors involved in the development of human GBM. This process is initiated in the germline, where parental epigenetic memory is maintained after fertilization and further induces allele-specific transcription and chromatin modifications of single or multiple neighboring genes known as imprinted genes increase. To date, more than 260 imprinted genes have been identified in the mouse genome, most of which are thought to be primary imprinted, imprinted germline Differentially Methylated Regions (DMR). Recent studies have shown that a subset of placenta-specific imprinted genes without DMRs is regulated by maternal histone modifications. To better understand DNA methylation-dependent (canonical) and non-canonical (non-canonical) imprints, this review article summarizes the loci under the control of each imprint type in mice and compares them to others compared to the corresponding homologues in rodents. Understanding the epigenetic systems that differ across loci or species can provide new models for studying genetic regulation and evolutionary divergence. Seed dormancy allows plant seeds to time germination until environmental conditions are suitable for seedling survival. This trait has high adaptive value and is of great agricultural importance. Endosperm is the reproductive tissue that forms after fertilization and plays an important role in establishing seed dormancy, in addition to supporting embryonic development. Many genes adopt specific expression patterns for the endosperm-derived parent. This is a phenomenon called genome imprinting. Imprinted genes are attacked by epigenetic mechanisms that act before and after fertilization. Recent studies have shown that imprinted genes are involved in seed dormancy formation, highlighting novel mechanisms of parental control over this adaptive trait. Intrauterine and postnatal developmental disorders are important clinical features of imprinted disorders, a molecularly defined group of congenital syndromes caused by molecular alterations affecting parental imprinted genes. These genes, like their parents, are expressed as mono alleles and influence human growth and development. Indeed, some genes exclusively expressed by the paternal allele have been shown to promote fatal growth, whereas maternally expressed genes repress it.

ACKNOWLEDGEMENT

None.

CONFLICTS OF INTERESTS

The authors declare that they have no conflict of interest.

Received:	03-October-2022	**Manuscript No:**	ipce-22-15147
Editor assigned:	05-October-2022	**PreQC No:**	ipce-22-15147 (PQ)
Reviewed:	19-October-2022	**QC No:**	ipce-22-15147
Revised:	24-October-2022	**Manuscript No:**	ipce-22-15147 (R)
Published:	31-October-2022	**DOI:**	10.21767/2472-1158-22.8.46

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Citation Abawi A (2022) Growth Restriction and Genomic Imprinted Phenotypes Support the Concept of Imprinted Genetics. J Clin Epigen. 8:46.

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