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Editorial

DNA Methylation Alterations in Down Syndrome Toddlers' Blood Cells

Kristine Warner*

Editorial office, Journal of Medical Diagnostic Methods, Barcelona, Spain

EDITORIAL

Trisomy 21 causes genome-wide abnormalities in DNA methylation patterns, which have been observed in multiple organs of people with Down Syndrome (DS) at various developmental stages, according to new research. New data on systematic genome-wide DNA methylation alterations in blood cells of people with DS from hitherto understudied age group-young children-is presented here. We show that the findings of the study are very similar to those found in the preceding literature. We further follow a quasi-longitudinal trend in the DS-associated DNA methylation patterns as systematic epigenomic instability with age, using relevant published data from two other developmental stages, neonatal and adult. One of the most frequent chromosome abnormalities is Down Syndrome (DS). According to the World Health Organization, the global incidence of Down syndrome is between one and 1000 live births. The presence of extra genetic material on chromosome 21 causes the syndrome, which is linked to physical growth delays, intellectual incapacity, and other developmental and physical impairments. More than 80 clinical DS characteristics have been identified, none of which can be explained solely by chromosome 21 gene triplication. Furthermore, despite the expected effect of "gene dosage," the expression of these genes may stay unchanged in DS. To date, most genomic studies have linked this diversity of DS clinical symptoms to genome destabilisation caused by additive effects of the trisomy on adjacent genes within a network or pathway, resulting in changes in gene expression and the mechanisms that govern it, such as DNA methylation. Tran's epigenetic effects, where the existence of an extra chromosome 21 influences methylation on other chromosomes, are frequently reported in an increasing body of research on DNA methylation in DS. Chromosome 21 contains epigenetic modifier genes involved in de novo methylation, such as DNA methyltransferase DNMT3L, whose extra-activity due to a "dosage effect" may lead to genome-wide eugenomic changes. Furthermore, an excess of transcription factors on chromosome 21, such as RUNX1, binding to the core element of numerous enhancers and promoters, may disrupt the genome-wide chromatin structure and DNA methylation pattern. In numerous cells and tissues, such as placenta tissue, blood cells, buccal epithelial cells and brain tissue, significant genome-wide alterations in DNA methylation in DS have been identified in comparison to matched controls. Immune system problems in DS have been linked to anomalies in blood cell form and function, as well as the altered prevalence of distinct blood cell subpopulations, according to haematological study. Because DNA methylation is tissue and cell specific, changes in cell-type proportions could skew the results of the differential methylation analysis when comparing DNA methylation in whole blood from children with and without Down syndrome. We report on genome-wide DNA methylation abnormalities in blood cells of children with DS during a critical time of development—infancy and toddlerhood, from 0.5 years to 4.5 years of age-to address a vacuum in the data on epigenetic perturbations in persons with DS of various age groups. We examine the findings of this work in the context of the existing literature on DS-related epigenetic changes, highlighting the findings' high level of consistency and reproducibility. Furthermore, we monitor a quasi-longitudinal trend in the age dynamics of DS-associated DNA methylation modifications in blood cells using relevant empirical data for babies and people with DS.

Correspondence to: Kristine Warner, Editorial office, Journal of Medical Diagnostic Methods, Barcelona, Spain, E-mail: editor.jmdm@journalres.com

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