

A link has been hypothesised to occur between suboptimal maternal nutrition and impaired foetal development leading to a predisposition to a range of adult pathologies. As a clear connection between dietary intake of methyl group donors and epigenetic defects has been demonstrated both *in vivo* and *in vitro*, this project had the purpose of generating a disruption into the methyl/folate cycle to investigate DNA methylation alterations during human preimplantation embryo development, using human embryonic stem cells (hESCs) as an *in vitro* model. In particular, HUES-7 stem cells were employed and cultured using either standard or methyl deficient media to test this hypothesis. After the treatments, that included an inhibitor of a key enzyme of the cycle, Differentially Methylated Regions (DMRs) of six imprinted genes were analysed and assessed for their methylation status at Cytosine-phospho-Guanosine (CpG) sites. As a consistent decrease of methylation was observed for the gene *H19* in treated cultures, its allelic expression was then investigated and an initial process of Loss Of Imprinting (LOI) was found. Additionally, global DNA MethylTransferase (DNMT) activity was examined and a statistically significant decrease in treated samples was detected. Finally, hESCs were differentiated into Embryoid Bodies (hEBs), which were compared and stained for pluripotency and germ-layer specific markers. Consistently different expression of *OCT-4* and *NANOG* was noticed for treated-culture derived hEBs.