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Effect of E-cadherin inhibition on mouse embryonic stem cells transcriptome: Relevance to oral squamous cell carcinoma

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Objectives: Oral Squamous Cell Carcinoma (OSCC) is a very invasive multistage process malignancy. It affects the stratified squamous epithelial cells of the oral mucosa and associated with high death rates worldwide. E-cadherin protein is an important marker of epithelial tissue and it is one of the key regulators of tissue integrity and polarity. The effect of E-cadherin protein down regulation with tumour progression is well known to be associated with poor prognosis. However, the way that this protein affects cell transcripts is still not well understood.

Methods: mouse Embryonic Stem Cells (mESCs) were used as a model for epithelial tissue alongside E-cadherin knockout mESCs (Ecad-/-mESCs). Microarray analysis and methylation sequencing were done for both cell lines. The results were assessed using Venn 2.1.1 bioinformatic, Bismark v0.17, GRAETAnnotation web tool mouse UCSC mm10 genome browser. The shared data between microarray and methylation sequencing were tested on OSCC tissue sections using immunofluorescence staining.

Results: The microarray has shown several thousand transcripts alterations which govern a number of biological processes. Some of these transcripts were up-regulated while others were down-regulated. The E-cadherin inhibition has shown bias toward hypermethylation of 10861annotated regions from Transcription Start Site (TSS) compared to hypomethylation (972 annotated regions±TSS). About 1417 of hypermethylated regions were in CpG islands. Venn bioinformatics has identified 10% of data shared between microarray down-regulated transcripts and hypermethylated genes. On the other hand, microarray up-regulated genes and hypomethylated genes have shown 4% of shared data. The immunofluorescence evaluation of some shared genes on OSCC tissue sections has shown significant effect of E-cadherin inhibition on their expression.

Conclusion: The inhibition of E-cadherin protein has a role on genes expression and methylation in mESC compared to Ecad-/-mESC, with bias toward increase genes methylation. The study highlighted some genes that were regulated by E-cadherin inhibition during oral squamous cell carcinoma formation.

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