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The Effect of DNA Methylation on TP73 Expression in Tumorgenesis

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Background

TP73 is a member of the TP53 family of proteins that acts as a transcription factor to help regulate cellular distress. This tumor protein may play a dual role as a tumor suppressor and tumor promoter. The TP73 gene is mapped to chromosome 1p36, a frequently deleted region in neuroblastoma and other types of tumors. Neuroblastoma is typically a childhood cancer that begins in the nerve tissue atop the adrenal gland of kidneys. Cancer is characterized by uncontrollable cell proliferation stemming from the accumulation of many mutations that develop over a long period of time.

Objective

To examine the role of TP73 in the formation of neuroblastoma

Methods

Normal fibroblasts and tumor kidney cells were grown at 37°C in 5% CO₂ in their respective growth media. DNA and RNA of both cell types were extracted using standard procedures. RNA expression was analyzed by RT-PCR and DNA methylation was analyzed by bisulfite sequencing. The expressions of TP73 gene and its protein were analyzed by gel electrophoresis and immunohistochemistry respectively.

Results

The TP73 gene was expressed in all of the neuroblastoma cells but not in the normal cell lines. This was due to the methylation of TP73 promoter in the normal cell lines.

Conclusions

Our studies revealed that TP73 was expressed in neuroblastoma cells but not in non-cancerous cells as a result of silencing by DNA methylation. In conclusion, the absence of TP73 in normal cells is in part due to DNA methylation implying that TP73 plays a role in the promotion and/or progression of carcinogenesis.











Figure 2. mRNA levels in Neuroblastoma cell lines (IMR32 and SK-N-SH) compared to normal fibroblast cell lines (HS27).

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Neuroblastoma

Figure 1. Immunohistochemical analyses of TP73 protein expression in healthy and neuroblastoma tissues.









References

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