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SHORT COMMUNICATION

Differential expression of a subset of ribosomal protein genes in cell lines derived from human nasopharyngeal epithelium

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Extraribosomal functions of human ribosomal proteins (RPs) include the regulation of cellular growth and differentiation, and are inferred from studies that linked congenital disorders and cancer to the deregulated expression of RP genes. We have previously shown the upregulation and downregulation of RP genes in tumors of colorectal and nasopharyngeal carcinomas (NPCs), respectively. Herein, we show that a subset of RP genes for the large ribosomal subunit is differentially expressed among cell lines derived from the human nasopharyngeal epithelium. Three such genes (*RPL27*, *RPL37a* and *RPL41*) were found to be significantly downregulated in all cell lines derived from NPC tissues compared with a nonmalignant nasopharyngeal epithelial cell line. The expression of *RPL37a* and *RPL41* genes in human nasopharyngeal tissues has not been reported previously. Our findings support earlier suspicions on the existence of NPC-associated RP genes, and indicate their importance in human nasopharyngeal organogenesis.
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INTRODUCTION

Products of ribosomal protein (RP) genes are essential for cellular protein biosynthesis. Besides this, studies have also linked them to human congenital disorders and cancers. For instance, *RPS4* has been implicated in Turner's syndrome,[1] and the mutant *RPS19* was found in individuals with Diamond–Blackfan anemia.[2] In colorectal carcinoma, the overexpressions of *RPS3*,[3] *RPS19*[4] and *RPL7a*[5] have been reported. *RPL23*, a tumor metastasis-related gene, was found to induce high invasiveness of a human lung adenocarcinoma cell line.[6]

In our previous study, 33 RP genes were overexpressed in tumors of colorectal carcinoma relative to their normal controls.[7] We have also recently identified two RP genes (namely *RPS27* and *RPS26*) to be downregulated in nasopharyngeal carcinoma (NPC) tumors compared with a normal control.[8]

Despite the increasing number of cancer-associated RP genes identified thus far, the full repertoire of RP genes linked to human cancers remains unclear. In this study, 18 RP genes encoding proteins for large ribosomal subunits were tested on cell lines derived from NPC tissues and the normal nasopharyngeal epithelium. This effort was aimed at identifying nasopharyngeal-associated RP genes.

MATERIALS AND METHODS

Cell lines and RT-PCR

Total RNAs were from the nonmalignant nasopharyngeal epithelial (NPE) cell line, NP69;[9] and from the NPC cell lines of TW01, HONE1 and SUNE1. Complementary DNAs (cDNAs) were constructed from the RNA, and RT-PCR was carried out using specific 20-mer primers designed for RP genes *MRPL3*, *RPL3*, *RPL7a/surf3*, *RPL9*, *RPL10*, *RPL12*, *RPL18*, *RPL21*, *RPL23a*, *RPL26*, *RPL27*, *RPL27a*, *RPL29*, *RPL30*, *RPL35a*, *RPL37a*, *RPL38* and *RPL41*. The RT-PCR products ranged in size from 105–433 bp.

Differential expression analysis

Expressions of all 18 RP genes and the internal control, glyceraldehyde-3-phosphate dehydrogenase (*GAPDH*), were assessed through band intensity measurement using the AlphaEaseFC (Alpha Innotech, San Leandro, CA, USA) software. Intensity values of genes for each cell line are normalized to the *GAPDH* value of the respective cell line. For calibration of band intensity measurement, bands from a DNA size reference marker (100-bp ladder) was evaluated for delimits in the range of measurable intensities. A replicate test was conducted for all genes in all cell lines. The mean normalized value of band intensities was then plotted in a bar (with s.d.) and line charts for descriptive statistical analysis. Analysis of the *MRPL3* gene was omitted because of undetectable expression. For quantitative statistical analysis, expression data

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