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Overexpression of cofactor of BRCA1 in HepG2 cells: A step towards understanding the role of COBRA1 in hepatocellular carcinoma

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Cofactor of *BRCA1 (COBRA1)* is a *BRCA1* interacting protein that represents one of the four subunits of the negative elongation factor (NELF) complex. NELF is known by its ability to stall RNA Polymerase II during the early phase of transcription elongation, resulting in repressed transcription of several genes including ones associated with tumorigenesis of different cancer types. While it was found to be down-regulated in breast cancer, *COBRA1* was found to be up-regulated in the upper gastrointestinal carcinoma. Up to date, the role of *COBRA1* in hepatocellular carcinoma (HCC) is unclear. We have previously demonstrated that silencing of *COBRA1* in the HCC cell line HepG2, significantly inhibited the proliferation and migration potentials of the cells. Here, we investigated the effect of ectopic expression of *COBRA1* on HepG2 cells proliferation and migration. Lipofectamine 3000 was used to transfect HepG2 cells with a pCMV5-HCOBRA1 plasmid. The transfection efficiency was determined by the percentage of EGFP positive cells (pEGFP-N1+) via fluorescent microscope, semi-quantitative RT-PCR as well as western blot analysis. The cells proliferation and migration following *COBRA1* overexpression were assessed using the Trypan blue dye exclusion method and the wound-healing assays respectively. The semi-quantitative RT-PCR was used to analyze the mRNA expressions of the other NELF subunits, *TFF1* and *TFF3* genes, which are known to be regulated by the NELF complex, as well as other tumorigenesis related genes. Our results revealed that *COBRA1* transfected cells exhibited a comparable proliferation and migration rates to non-transfected cells. These results were accompanied by an insignificant effect of *COBRA1* over-expression on the levels of the proliferation marker-Ki-67 and the anti-apoptotic gene-Survivin. Also, the mRNA levels of the other NELF subunits, *TFF1* and *TFF3* were found to be comparable among all the tested groups. Collectively, our results suggest that the proposed involvement of *COBRA1* in HCC is supported by and dependent on the assembly of the active NELF complex, which requires the expression of all four NELF subunits. Moreover, *COBRA1* mediated role in HCC tumorigenesis might be due to mechanisms and regulatory pathways other than the ones examined here. However, further studies are required to confirm these notions.

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