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Overexpression of Kinectin Isforms Containing the D2 Variant Splicing Region enhance Cell Proliferation in Hepatocellular Carcinoma

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Kinectin, an endoplasmic reticulum binding protein, was identified as a receptor of kinesin firstly. In our previous study, we found that Kinectin was one of the tumor associated antigens of hepatocellular carcinoma (HCC) by SEREX method, and Kinectin mRNA expression levels concluding D2 variant splicing regions were significant higher in HCC tissues than both cancer paired adjacent tissues and normal liver tissues. Here, we found Kinectin containing D2 variant splicing regions isoforms protein and Kinectin conserved region protein were higher expressed in HCC tissues than in paired adjacent tissues. And Kinectin containing D2 variant splicing regions isoforms can significantly enhance cell proliferation and colony forming efficiency, promote cell from G1 phase into S phase, and reduce cell apoptosis, but had no effect on cell migration in HepG2 cells. And Kinectin containing D2 variant splicing regions isoforms can significantly promote the tumorigenicity and development of tumors in nude mice *in vivo*. Our results shed new light.

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Identification of Transcription factor GCF2 target genes involved in Hepatocellular carcinoma metastasis and invasion

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GC-binding factor 2 (GCF2) is a transcriptional repressor that represses target genes transcriptional activity, and GCF2 is high expressed in most HCC tissues and cell lines including HepG2. Here we found that specific down-regulation of GCF2 expression in HepG2 repressed cell migration and invasion by the wound healing and transwell assay. To explore the target genes involved in metastasis and invasion in HepG2, chromatin immunoprecipitation (ChIP) combined with DNA microarray analysis (ChIP-chip) was used in this experiment. We got 62 migration related target genes and 6 invasion related target genes. Real-time fluorescent quantitative RT-PCR were performed on 18 target gens, and we found that MAPK1 mRNA expression was low-regulated, and LGALS1 up-regulated in HepG2 cell when GCF2 gene was knockdown by siRNA. Our results suggest that GCF2 participate in metastasis and invasion through the key signal transduction pathway involving MAPK1 and LGALS1.