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Nestin mediates hedgehog pathway tumorigenesis by regulating Gli3 processing

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The transcriptional activation by sonic hedgehog signaling (SHH) depends on the Gli family of transcription factors. Among the Gli proteins, Gli3 regulates shh signaling by either serving as transcriptional activators or being phosphorylated and process into truncated transcriptional repressors. In this study, we show that nestin, a type VI intermediate filament protein, plays a regulatory role in Gli3 processing in medulloblastoma tumorigenesis. We found that overexpression of nestin resulted in minimal Gli3 processing, conversely, Gli3 processing is dramatically increased with the loss of nestin. Our results demonstrate that binding of nestin to Gli3 blocks Gli3 phosphorylation and its subsequent proteolytic processing. Furthermore, deletion of nestin in medulloblastoma cells inhibited cell proliferation. These data suggest that nestin serves as a regulator by overcoming negative regulation of shh pathway, a pathway that plays important roles in both development and cancer.

Biography

Hing Leung Eric Lee has completed his PhD in 2013 from Clemson University. He is currently a Post-doctoral Associate in Fox Chase Cancer Center. His research focuses on different regulatory partners in the sonic hedgehog pathway. Currently, he is working on the regulatory and functional role of Nestin and different factors in the stem cell niche.

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