NEWS

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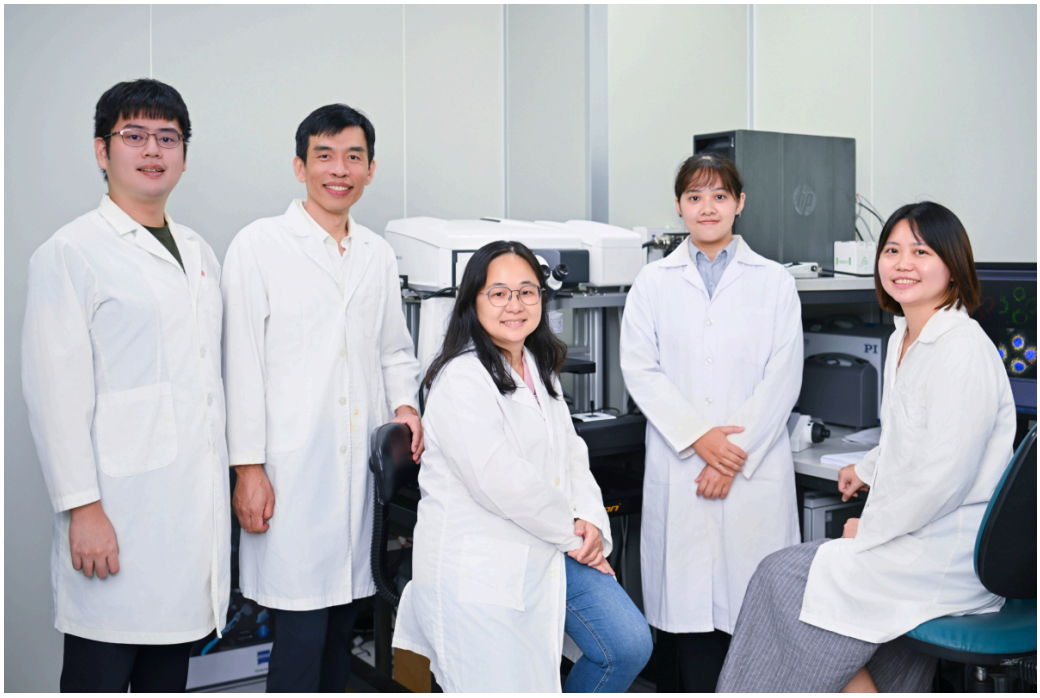
NYCU Identifies New Target for Pediatric Brain Cancer Treatment — Uncovering the Role of Cellular “Antennas” in Tumor Growth



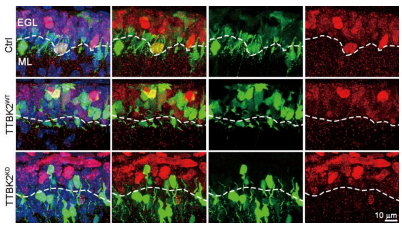
The research team, from left to right: Yue-Ru Li, Prof. Jin-Wu Tsai, Prof. Won-Jing Wang, Yu-Wen Cheng, and I-Hsuan Lin.

Edited by Chance Lai

This dynamic balance

In a breakthrough that could change the future of pediatric brain cancer therapy, researchers at National Yang Ming Chiao Tung University (NYCU) have identified a critical molecular mechanism that drives the development of medulloblastoma—the most common malignant brain tumor in children.

The findings, published in *Cell Death & Differentiation*, pave the way for new precision therapies that may spare young patients from the severe side effects of current treatments.



The discovery that TTBK2 activity promotes the proliferation of cerebellar GNPs highlights its critical role in brain development and disease.

Understanding the Roots of Brain Tumors

between TTBK2 and HUWE1 is essential for healthy brain development. But in medulloblastoma, the system breaks down. TTBK2 fails to degrade, leading to persistent cilia, unchecked GNP proliferation, and ultimately tumor formation.

A Promising Therapeutic Target

Crucially, the researchers demonstrated that suppressing TTBK2 not only eliminates the cilia on tumor cells—reducing their ability to receive growth signals—but also significantly curbs tumor growth. These results identify TTBK2 as a promising new therapeutic target for medulloblastoma.

“Brain cancer remains one of the most challenging diseases in medicine,” said Prof. Jin-Wu Tsai. “While current therapies such as surgery, radiation, and

Medulloblastoma originates in the cerebellum—the brain region that coordinates movement and balance—and is closely linked to developmental errors. A key player in this process is a population of cells called granule neuron progenitors (GNPs), which must proliferate and differentiate with high precision during early brain development. These cells rely on tiny, antenna-like structures on their surface—primary cilia—to receive growth signals from their environment.

A research team led by Prof. Won-Jing Wang (Institute of Biochemistry and Molecular Biology) and Prof. Jin-Wu Tsai (Institute of Neuroscience) at NYCU has now uncovered how two key genes—TTBK2 and HUWE1—work together to regulate this ciliary signaling process. Using both mouse and zebrafish models, the study is the

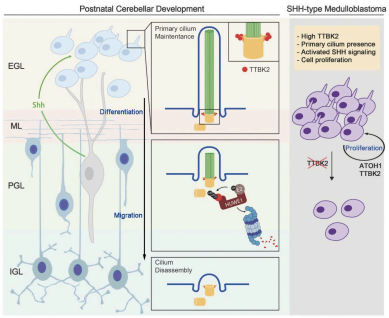
chemotherapy can prolong survival, they often come with serious long-term consequences like cognitive impairments or secondary cancers. Our study shows that precise disruption of tumor growth mechanisms could lead to safer, more effective treatments.”

Prof. Won-Jing Wang added, “Scientists once considered primary cilia to be evolutionary remnants without real function. But it turns out they act like true antennas—critical for how cells interpret their environment. Our findings highlight not only the importance of cilia in brain development, but also their potential role in cancer and drug resistance. This opens up an entirely new direction for brain tumor precision medicine.”

first to establish their central role in both normal cerebellar development and tumor formation.

The Antenna Keepers: TTBK2 and HUWE1

The team found that TTBK2 acts as a “ciliary guardian”, maintaining the structure and function of primary cilia in GNPs to ensure they continue receiving signals that promote proliferation. Once these cells complete their growth phase, HUWE1 acts as a molecular switch, degrading TTBK2 and dismantling the cilia—thereby prompting the cells to differentiate into mature neurons.



The research team discovered that SHH signaling protects a protein called TTBK2, allowing it to remain on the cell’s primary cilium and promote neuronal growth. However, in brain tumors, this mechanism is hijacked to accelerate tumor progression. The study suggests that inhibiting TTBK2 could lead to new therapeutic strategies for SHH-subtype medulloblastoma.

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