Medical Xpress

Alternative DNA repair mechanism could provide better treatment for neuroblastoma in kids

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Researchers at the University of Michigan's C.S. Mott Children's Hospital have identified a promising new target for developing new therapies for kids with high-risk neuroblastoma, according to a new study published in *Molecular Cancer Research*.

The research, led by Erika Newman, M.D. of C.S. Mott Children's Hospital, found for the first time that components of an alternative DNA repair pathway are highly expressed in neuroblastoma tumors.

"We discovered that high-risk neuroblastoma cells preferentially use an efficient but erroneous DNA repair pathway that gives these cells survival advantage. Importantly, children with neuroblastoma tumors harboring these alternative repair factors have worse overall survival than children with tumors that have low expression," says Newman, who is assistant professor of pediatric surgery at the University of Michigan Medical School and surgical director of the Mott Solid Tumor Oncology Program (MSTOP).

Newman says this information could provide a promising treatment option for neuroblastoma patients, by developing new therapies that disrupt the ability of cancer cells to repair DNA damage.

"There is an urgent need to develop new therapies for children with high-risk neuroblastoma," Newman says.

"Nearly half of patients present with tumors that have already spread. Despite current treatment, most with high-risk neuroblastoma don't survive. The primary focus of our lab is to develop new treatment approaches for children with high-risk disease."

Neuroblastoma is the most common cancer infants and the most common solid tumor outside of the brain in all children, in which malignant cancer cells form in primitive nerve tissue called "ganglions" or in the adrenal glands.

"We are very excited that these findings have provided insight into the mechanism by which neuroblastoma tumors overcome DNA damage. This study provides evidence that an alternative repair mechanism is functional in neuroblastoma and offers experimental support for further preclinical investigation of DNA repair pathways as new therapeutic targets in high-risk neuroblastoma," says Newman.

More information: *Molecular Cancer Research*, DOI: 10.1158/1541-7786.MCR-14-0337

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