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Absence of specific enzyme in cartilage can lead to benign tumors in mice

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Rhode Island Hospital researchers have found that the absence of the Shp-2 enzyme near specialized cartilage cells can lead to the development of multiple benign cartilage tumors in mice, a model that recapitulates the rare human tumor syndrome metachondromatosis. Shp2 is an enzyme in the cell that regulates the activity of other proteins and signaling pathways. Mice lacking Shp2 formed two types of tumors: enchondromas and osteochondromas, and also developed deformed joints. The study is published online in advance of print in the journal *Nature*.

"Transgenic mouse models of human disease are powerful tools to gain a better understanding of disease processes and to test novel treatments," said lead author, Wentian Yang, Ph.D., of the department of orthopedics at Rhode Island Hospital. "In humans, some of these benign tumors will progress to malignant cartilage tumors, called chondrosarcomas, which are even more serious since they spread and can be fatal."

He continued, "Knowing the cell of origin for tumor formation can be helpful in developing new treatments since each type of cell has its weak points. Our research led to the discovery of a novel cartilage stem/progenitor cell population that is the cell of origin for the tumors in our model, and to the identification of a potential therapy for this rare disease. In the future, our model may also be useful for developing novel treatments for some forms of chondrosarcoma, a type of bone cancer."

Injury to the growth plate has been the assumed reason for the formation

of osteochondromas, which look similar to a growth plate that has taken off in the wrong direction, causing bone and cartilage to grow out of the bone. This research demonstrates that one cause of osteochondromas is a molecular abnormality in an enzyme in a tiny group of cells near the growth plate, resulting in often large tumors growing out of the bone. These tumors are a very common in children.

"These findings put us one step closer to understanding how these tumors form," Yang said. "The next step will be to develop a way to stop the tumors from growing at an early stage so that they do not have to be surgically removed."

Provided by Lifespan

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