Study identifies mechanisms underlying osteochondroma formation

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Osteochondromas are cartilage-capped benign bone tumors that form on the surface of bones near growth plates. These growths typically occur in adolescents and can result in growth impairment, pain, and fractures. In hereditary forms of the disease, 80-90% of patients have mutations in the genes exostosin 1 and 2; however, these mutations are not found in all patients.

In this issue of *JCI Insight*, Antonios Aliprantis and colleagues at Brigham and Women's Hospital and Harvard Medical School in Boston demonstrate that loss of the genes encoding nuclear factor of activated T cells c1 and c2 (known as NFATs) induce osteochondroma formation in mice. Moroever, Aliprantis and colleagues identified the specific population of cells that give rise to these growths, known as aggrecan-expressing cells.

Although mutations in the NFATs have yet to be identified in human osteochondromas, these studies indicate that therapeutic strategies targeting NFATs could potentially be used to treat skeletal diseases characterized by excessive growth of this cell population.

More information: Xianpeng Ge et al. NFAT restricts osteochondroma formation from entheseal progenitors, *JCI Insight* (2016). DOI: 10.1172/jci.insight.86254