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Classified as

New Finding

Different cell types have been proposed as potential cellular drivers for heterotopic ossification (HO) in human fibrodysplasia ossificans progressiva (FOP). By using a physiologically relevant conditional expression allele of *Acvr1R206H*, the authors of this work generated a mouse model of FOP that highlighted the role of fibro/adipogenic progenitors (FAPs) in the development of HO. Interestingly, in this mouse model, FAPs were identified as the common player in the full spectrum of major HO anatomical sites reported in FOP patients and in the different forms of the disease (spontaneous and injury-induced). Moreover, a cell-autonomous effect of the mutant receptor as well as its stimulatory activity on chondro-osseous signaling in response to activin ligands were also shown. These results do not exclude the potential contribution to HO of the different local or circulating cell populations reported in previous work {1,2}, as the authors themselves recognize. However, they provide a novel perspective and a powerful experimental system for further studies on the pathogenesis as well as on the prevention/reversion of HO in human FOP.

References

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Disclosures

None declared

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