The suture provides a niche for mesenchymal stem cells of craniofacial bones.

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Contributions
H.Z. and Y.C. designed the study. H.Z. carried out most of the experiments and analyzed the data. J.F. participated in the suture cell culture experiments. T.H. and W.G. participated in the microCT analysis. M.U. provided comments. H.Z. and Y.C. co-wrote the paper. Y.C. supervised the research.

Competing financial interests
The authors declare no competing financial interests

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Summary

The suture mesenchyme connects craniofacial bones and has previously been believed not to possess significant functions. In our study, we identified *Gli1*+ cells within the suture mesenchyme as mesenchymal stem cells (MSCs). They give rise to the periosteum, dura and craniofacial bones during homeostasis and injury repair. Ablation of *Gli1*+ cells from adult mice leads to fusion of all craniofacial sutures, arrest of skull growth, and severe osteoporosis. *Gli1*+ cells are typical MSCs *in vitro* and are regulated by IHH secreted from the osteogenic front. Blockage of this hedgehog signaling leads to osteoporosis. *Twist1*$^{+/-}$ mice with craniosynostosis show reduced numbers of *Gli1*+ cells in sutures, suggesting the synostosis may result from a loss of suture MSCs. Our study reveals a novel function for craniofacial suture mesenchyme, providing a new perspective for understanding the onset of craniosynostosis and other suture deformities, as well as a potential new therapy for craniofacial disorders.