

# Craniosynostosis Pathogenesis

Becker LE, Hinton DR. Pathogenesis of craniosynostosis. *Pediatr Neurosurg*. 1995;22(2):104-7. doi: 10.1159/000120885. PMID: 7710971.

The convergence of multi-signals on the [Erk1/2](#) signaling pathway indicated the vital role of Erk1/2 in the pathogenic processes of [craniosynostosis](#). Over the past years, researchers tried to interfere the processes of [suture fusion](#) via molecule mechanisms, especially FGFs and related signaling <sup>1) 2) 3)</sup>

<sup>1)</sup>

Shukla V, Coumoul X, Wang RH. et al. RNA interference and inhibition of MEK-ERK signaling prevent abnormal skeletal phenotypes in a mouse model of craniosynostosis. *Nat Genet*. 2007;39:1145-50.

<sup>2)</sup>

Morita J, Nakamura M, Kobayashi Y. et al. Soluble form of FGFR2 with S252W partially prevents craniosynostosis of the apert mouse model. *Dev Dynam*. 2014;243:560-7.

<sup>3)</sup>

Yin L, Du X, Li CL. et al. A Pro253Arg mutation in fibroblast growth factor receptor 2 (Fgfr2) causes skeleton malformation mimicking human Apert syndrome by affecting both chondrogenesis and osteogenesis. *Bone*. 2008;42:631-43.

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