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 [1) 2) 3)]

1)
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.
2)
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.
3)
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.