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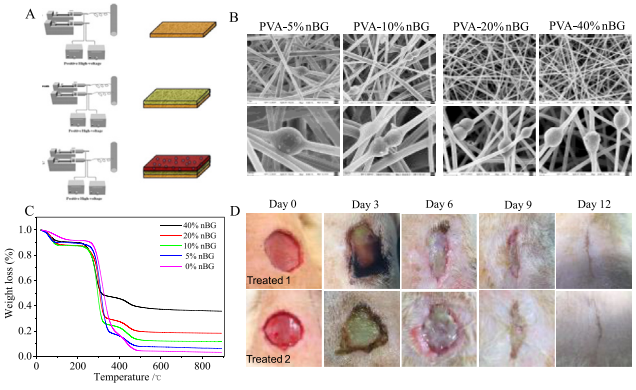

Figure 1 (A) Schematic diagram for the preparation of multilayer films. (B) Transmission electron microscope image of PVA fibers incorporating various contents of nBG (5, 10, 20, and 40%). (C) Thermo-gravimetric analysis of samples showing weight loss with temperature increase, revealing the remaining weight corresponding to the quantity of nBG initially incorporated. (D) Representative images of full-thickness skin defects in rodents (Treated 1: PVA/PVA-CS/PVA-nBG; Treated 2: CS/PVA-CS/PVA-nBG).

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ROLE OF FGFR2 IN CALVARIAL DEFECT HEALING

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Introduction: FGFR2 is an important members of the FGFRs family, many studies have shown that FGFR2 plays an important role in bone development. Calvarial defect healing is a complicated regeneration process which to some degree recapitulates the skull development. Many molecules involved in skull development also play important roles during calvarial defect healing such as Wnts, FGFs, BMPs, etc. Given that FGFR2 plays an important role in skull development, we speculate FGFR2 may participate in the regeneration process after calvarial defect, but its specific function and related mechanism are not clear. So, we induced gain-of-function mutation of FGFR2 (inducing expression P253R mutation) in adult mice, and through establishing of calvarial defect model , we observe the influence of FGFR2 on calvarial defect healing and discuss the possible mechanism.
Subjects and Methods: Calvarial defects were established in $FGFR2^{P253R/+}$ mice. Micro-CT was used to dynamic observation the healing process 2 weeks, 4 weeks, 8 weeks after calvarial defect and defect healing area were also quantitatively measured. H.E staining was used to observe new bone formation around defect edge 8 weeks after calvarial defect. Quantitative PCR was used to detect mRNA levels of Runx2, Coll, OP, OC 2 weeks after calvarial defect. Osteoblast cell culture was used to examine the role of FGFR2 on osteoblast proliferation and differentiation. The protein level of phosphorylated ERK1/2 was detected by western blot, the mRNA levels of canonical Wnt/β-catenin pathway genes Dvl2, β-catenin, Tcf1 in osteoblast cells were detected by quantitative PCR.
Results: Micro-CT and HE staining results show that $FGFR2^{P253R/+}$ mice have accelerated defect healing ability. The proliferation and differentiation of $FGFR2^{P53R/+}$ osteoblasts were accelerated. The expressions of bone formation markers such as Runx2, Coll and OP were increased, and the activity of ERK1/2 pathway was enhanced in $FGFR2^{P253R/+}$ osteoblast cells. mRNA levels of canonical Wnt/β-catenin pathway genes Dvl2, Tcf1 were increased in $FGFR2^{P253R/+}$ osteoblast cells.
Discussion and Conclusion: FGFR2 promotes calvarial defect healing. FGFR2 accelerates the proliferation of osteoblast cells, and promotes the differentiation of osteoblasts by up-regulating Runx2 and partially through ERK1/2 MAPK pathway. FGFR2 accelerates osteoblasts differentiation partly by activating the canonical Wnt/β- catenin signaling pathway.

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THE INDIVIDUALIZED CORRECTION OF THE SECONDARY DEFORMITIES POST-TRAUMATIC PHYSEAL BAR OF DISTAL TIBIA

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Objective: Distal tibia physis injury had a high incidence of physeal arrest. There was secondary deformity tends to have a disturbance on the development and function of the limb. The purpose of the study was to evaluate the treatment of the children patients with the secondary deformities post-traumatic physeal bar of distal tibia.
Methods: A retrospective study of distal tibia premature physeal arrest with deformity of 16 patients in our institution between January 2008 and October 2014. With analysis of the radiographic findings of physeal arrest, we try to classify the deformity into four Type. And those also was evaluated in two duration with shorten of the limb or not. We made the surgical plan based on the classification, and divided the patients into 2 group. Patients in group 1 were treated with physeal bar resection, filled the defect with fat and done osteotomy on the distal tibia furthermore. While patients in group 2 were treated with arthrodesis of ankle and limb lengthening. In group 1 there were 6 boys and 5 girls, aged 5—13 years (mean, 10.8 years), 8 left and 3 right. There were 4 boys and 1 girl in group 2, aged 10—15 (mean, 12.7 years), 3 left and 2 right. There was 3—6 months (mean, 4.8 months) spent on limb lengthening with external fixation, and the lengthening length was 3—6.5 cm (meas, 4.2 cm).
Results: A surgical procedure was successfully created in all the children, mean follow-up was 14.9 months (range, 6—24 months). All the children had an obvious improvement with the function and appearance in the last follow-up. There were 10 children had satisfactory results and 3 had minor loss of correction, however, 3 occurred deformity recurrence and 1 need revision. According to the Johner—Wruhs score scale in the last follow-up, there were 10 excellence, 2 good, 2 mid and 1 bad.
Conclusion: Autogenous fat graft had a good efficiency to fill into the defect after physeal bar resection. Currently, the premature partial closure of the physis mainly classified according to the size and position of physeal bar, and there are still insufficient to evaluate the clinical work. So we try to propose a new classification and staging method to evaluate the premature partial closure of the physis based on the secondary deformities. The growth arrest of lower limb caused by physeal bar were complex, a reliable and individualized method should be used.
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