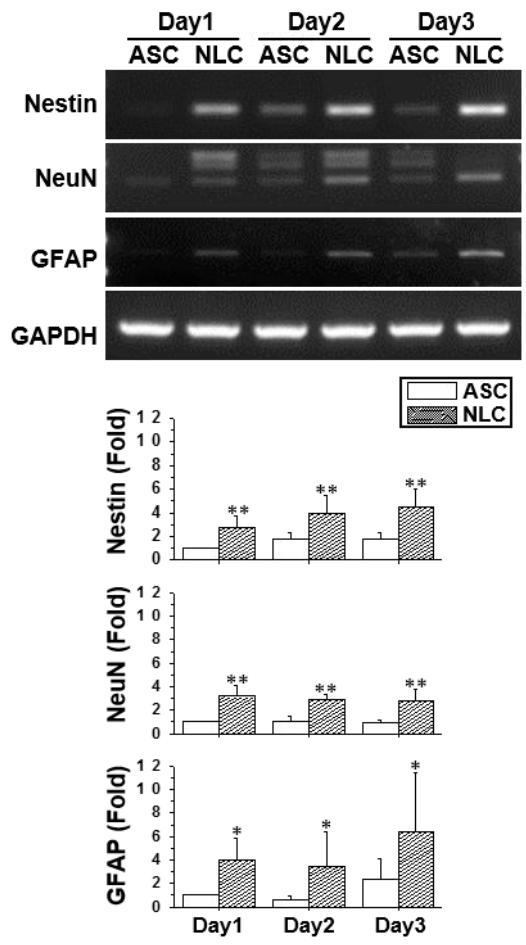


1 **Figure S1**

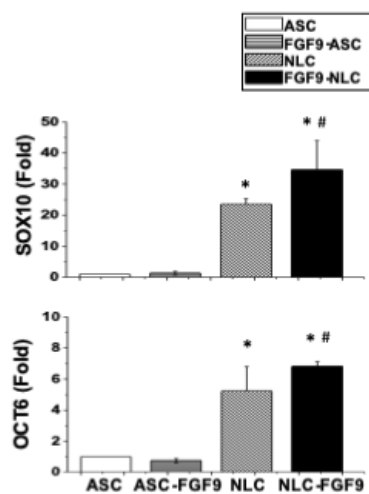


2
3
4
5
6
7
8
9
10
11
12

Figure S1. The gene expression of neural markers were observed by RT-PCR on day1 to day3 during the NLCs induction. Data are represented as mean \pm SD. N = 3 independent biological repeats. *p < 0.05, **p < 0.01 compared to undifferentiated ASCs.

13
14

Figure S2

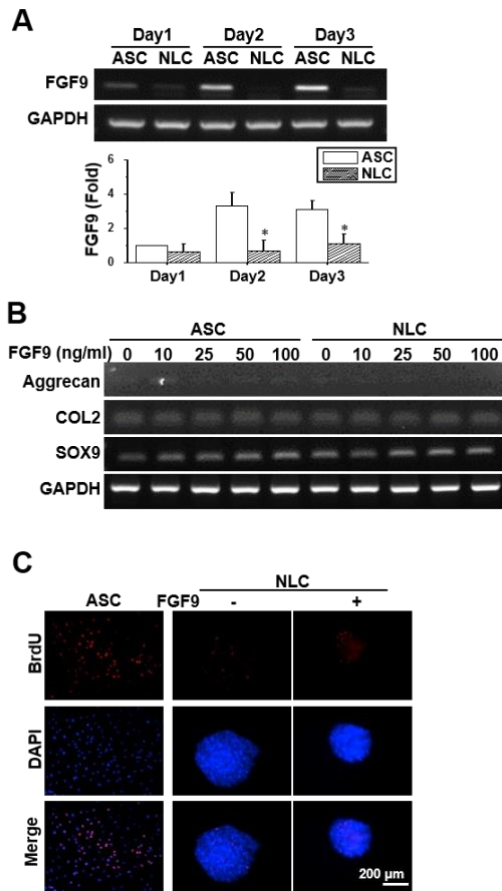


15
16
17
18
19
20

Figure S2. The gene expression of immature SC markers Sox10 and Oct6 were increased in NLCs. The administration of FGF9 further enhanced the expression of Sox10 and Oct6. Data are represented as mean \pm SD. N = 3 independent biological repeats. *p < 0.05 compared to undifferentiated ASCs. # p < 0.05 compared to NLCs without FGF9.

21
22
23

Figure S3

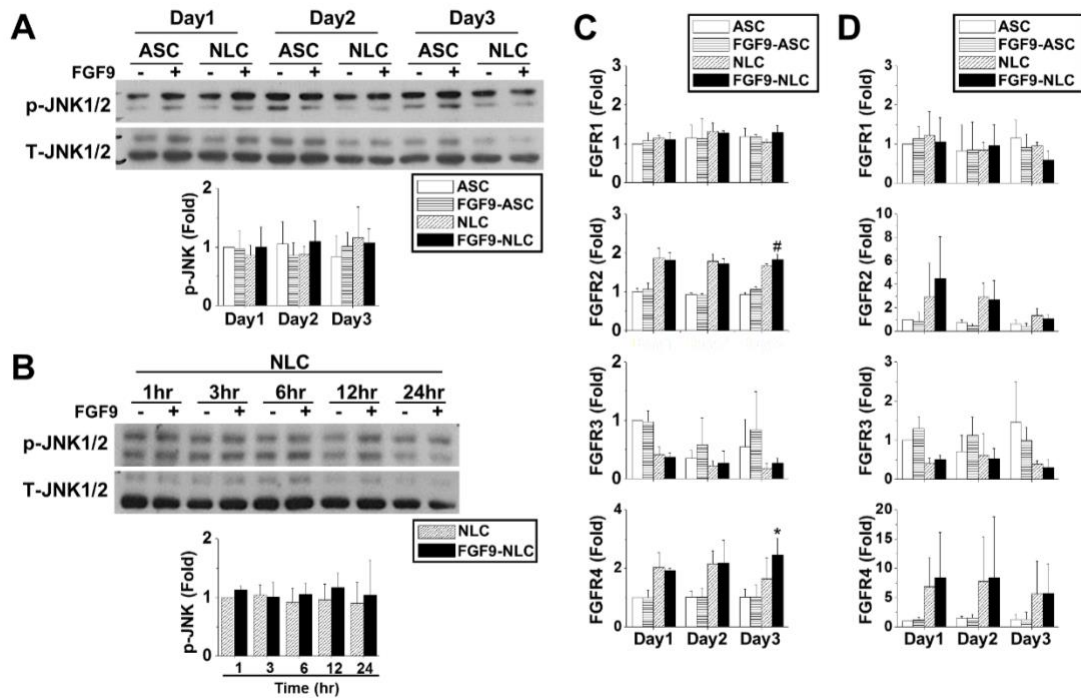


24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40

Figure S3. (A) The gene expression of FGF9 were observed by RT-PCR on day1 to day3 during the NLCs induction. (B) The undetectable change of aggrecan, collagen II (COL2), and Sox9 in ASCs and NLCs indicated no chondrogenic differentiation with different dosage of FGF9 treatments. (C) The BrdU was added into the medium during sphere formation to label the proliferating cells (Red color) and demonstrated very few NLCs were positive to BrdU as compared to the adherent ASCs. Furthermore, the addition of FGF9 had no considerable effect on increasing proliferation in NLCs. Data are represented as mean \pm SD. N = 3 independent biological repeats. * $p < 0.05$ compared to undifferentiated ASCs.

41
42
43

Figure S4

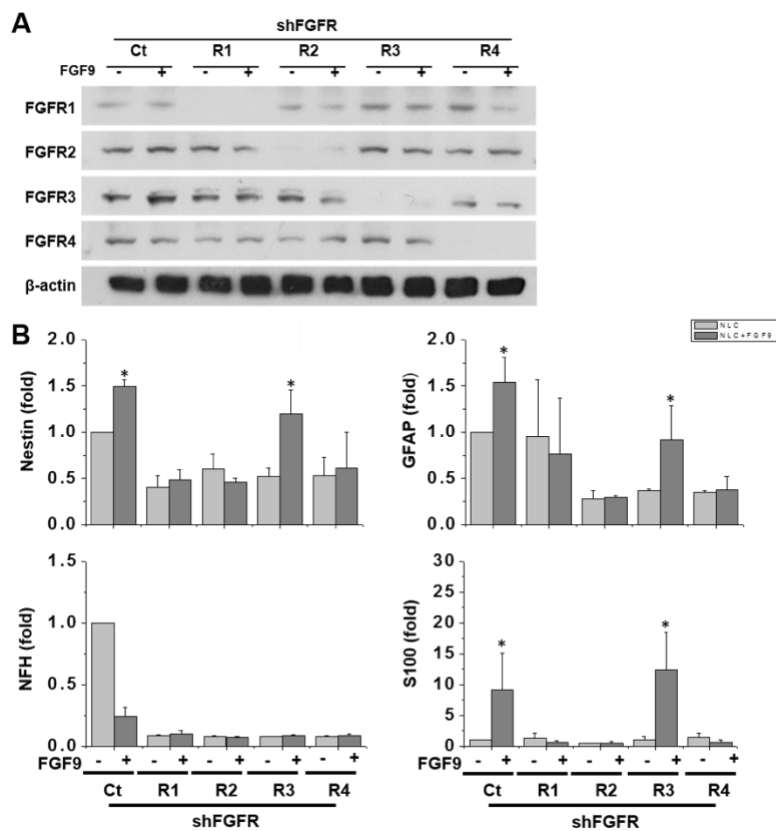


44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60
61
62
63
64

Figure S4. (A) The phosphorylation of JNK was investigated with/without FGF9 treatments between Day1 to Day3 in ASCs and NLCs. (B) The effect of FGF9 on p-JNK were also measured in NLCs at shorter time points within 24 hr. The phosphorylation of JNK was not affected by sphere formation as well as by FGF9 treatment. (C & D) The quantification result of Figure 3E & 3F. Data are represented as mean \pm SD. N = 3 independent biological repeats. For C, *p < 0.05 compared to undifferentiated ASCs, # p < 0.05 compared to day1 undifferentiated ASCs.

65
66
67

Figure S5

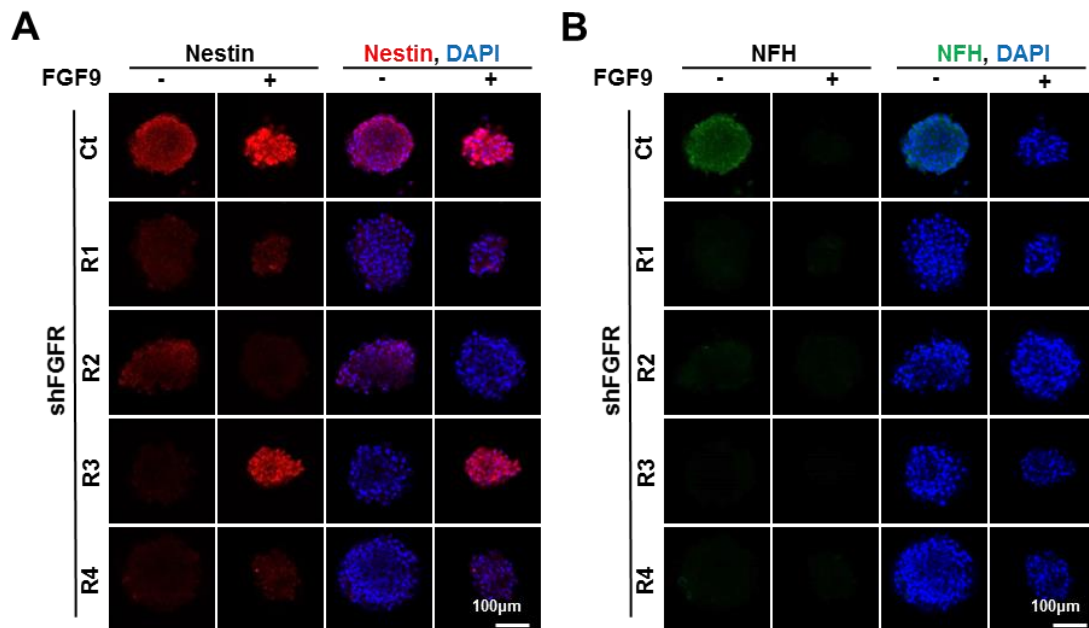


68
69
70
71
72
73
74
75
76
77
78
79
80
81
82
83
84
85

Figure S5. (A) The ASCs were transfected with specific shFGFR1-4 before NLCs induction. The specificity and efficiency of each shFGFR was confirmed by western blotting with specific antibody against FGFR1-4. (B) The quantification result of Figure 4B. Data are represented as mean \pm SD. N = 3 independent biological repeats. For B, * $p < 0.05$ compared to undifferentiated ASCs.

86
87
88

Figure S6

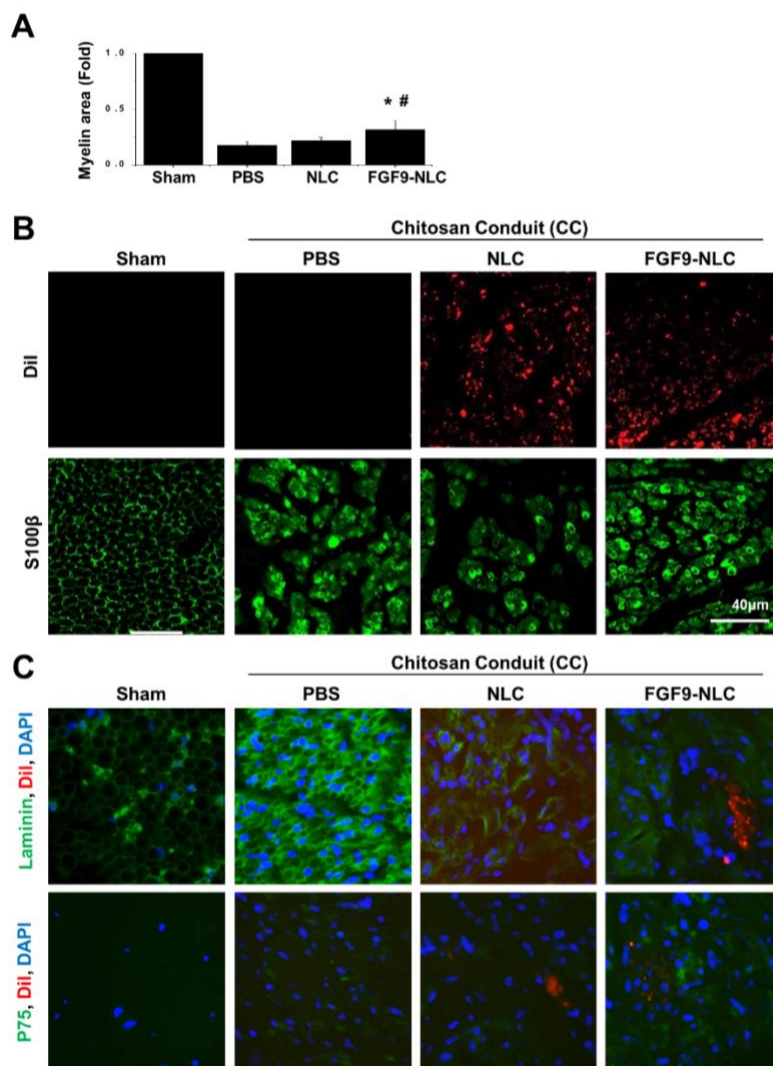


89
90
91
92
93
94
95
96
97
98
99
100
101
102
103
104
105
106
107
108
109
110

Figure S6. (A) The immunofluorescent staining of Nestin showed the blockage of FGFR2 (shFGFR2 with or without FGF9) inhibited the increase of Nestin expression with FGF9 treatment (Ct with FGF9). Other shFGFR for R1 and R2 also prohibited the Nestin expressions in NLCs, but not shFGFR3. (B) Silencing of any FGFR decreased the NFH expression in NLCs without FGF9 treatment. Scale bar in fluorescent image: 100 μm.

111
112
113

Figure S7



114
115
116
117
118
119
120

Figure S7. (A) The quantification of myelin sheath area showed improvement in FGF9-NLCs treatment. (B) The immunofluorescent staining of S100 β (green) on nerve section. The DiI-labeled cells were tracked by red fluorescent image in the same section. (C) The fibrotic scar occurred by laminin staining in PBS group was decreased in both NLC and FGF9-NLC treatment. Scale bar in fluorescent image: 40 μ m. * $p < 0.05$ compared to PBS. # $p < 0.05$ compared to NLC.