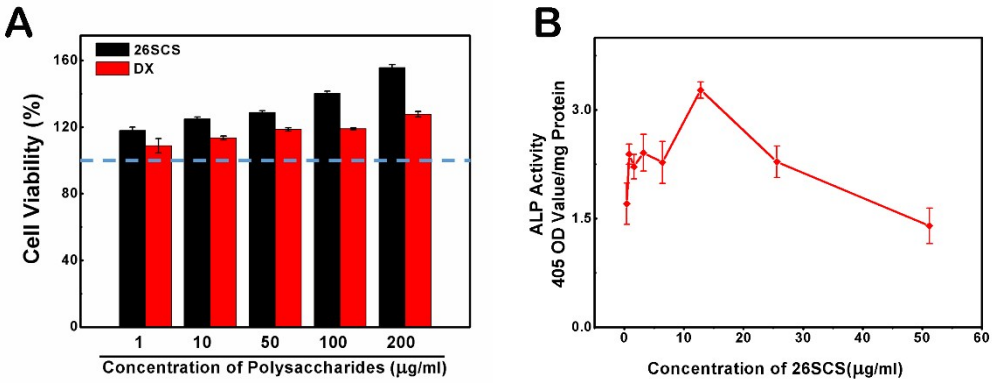

Fig.S1. (A) Cell viability of BMSCs: BMSCs were cultured for 3 days with 1, 10, 50, 100 and 200 μg/mL polysaccharides, respectively. (n=3). The results show that 26SCS and DX are not cytotoxic to BMSCs. (B) Concentration screening of 26SCS used for *in vitro* osteogenic differentiation. ALP relative activity assay (n=3) of BMSCs was proceeded after co-culturing with PM with various concentrations of 26SCS and 400 ng/mL rhBMP-2 for 7 days, respectively. 26SCS can improve ALP activity of BMSCs even with low concentration. In 26SCS concentration of 12.8μg/mL, the relative activity of value is significantly higher than other concentrations. Thus, the optimal concentration ratio 26SCS and rhBMP-2 is set as 32:1.

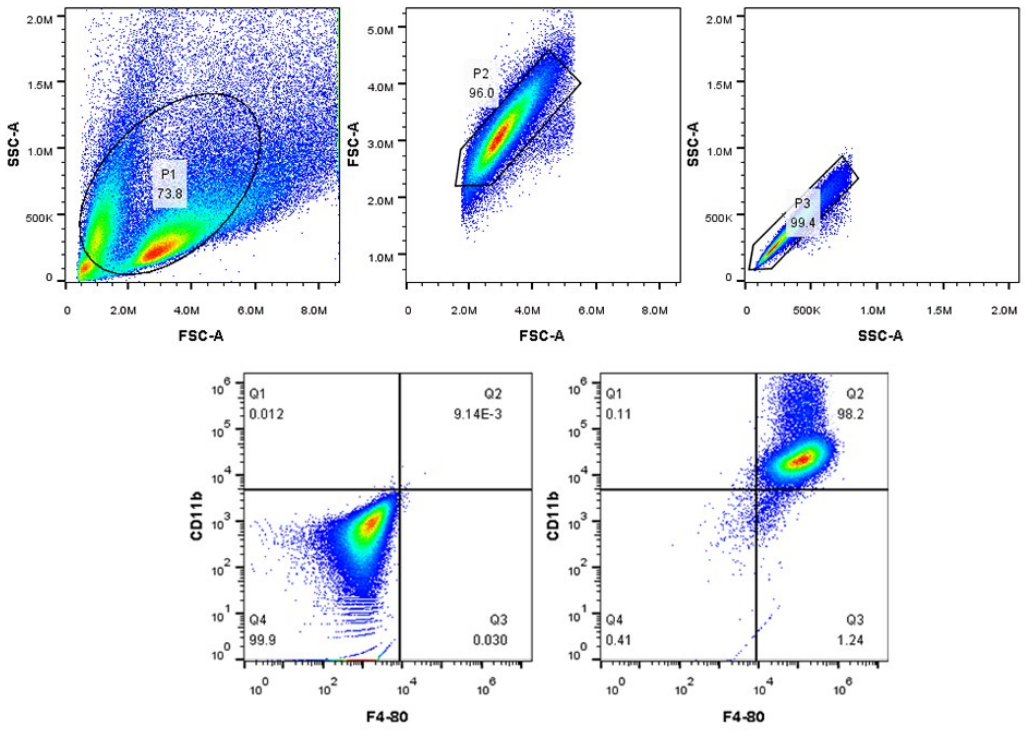

Fig. S2. Identification of Macrophages.

F4/80 and CD11b was used to identify the isolated macrophages by Flow cytometry analysis. 98% of the peritoneal cell surface expressed both CD11b and F4-80 antibodies, so it can be considered that the cells cultured by this method are macrophages and can be used for subsequent experimental studies.

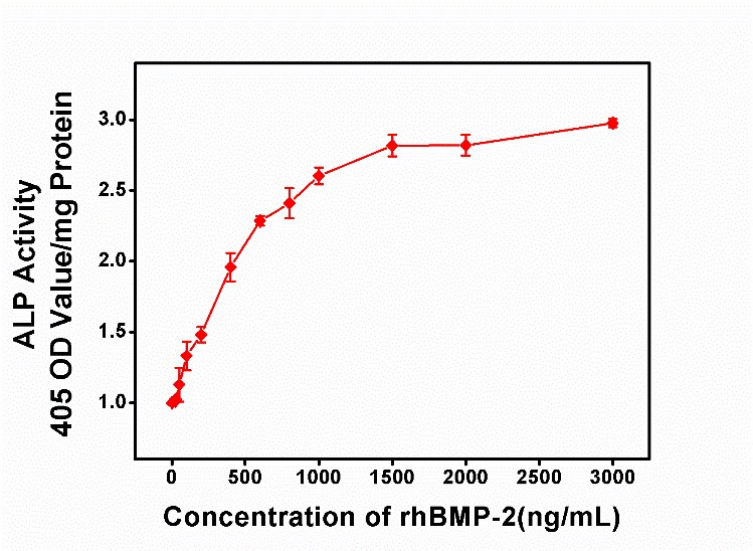

Fig. S3. Influence of rhBMP-2 concentration on relative ALP activity of BMSCs. ALP relative activity assay was proceeded after cultured in maintenance medium contain different concentration of rhBMP-2 for 7 days. Results are the mean±standard deviation (n=3).

To screen for the concentration of rhBMP-2 used in subsequent cell experiments, we determined the ALP activity of BMSCs cultured in maintenance medium containing 0, 25, 50, 100, 200, 400, 600, 800, 1000, 1500, 2000 and 3000 ng/mL of rhBMP-2, respectively. From the results of Fig.S3, it can be seen that when the concentration of rhBMP-2 is 0-200 ng/mL, the expression of ALP activity is low; when the concentration of rhBMP-2 is 400-800 ng/mL, the ALP activity increases linearly with increasing rhBMP-2 concentration; when rhBMP-2 is used at a concentration of 800-3000 ng/mL, the ALP activity slowly increases with growing rhBMP-2 concentration and gradually enters the platform phase. 400 ng/mL was finally selected as the concentration of rhBMP-2 used in the later experiments because this concentration is in the straight line segment where ALP activity is sensitive to changes in rhBMP-2 concentration.

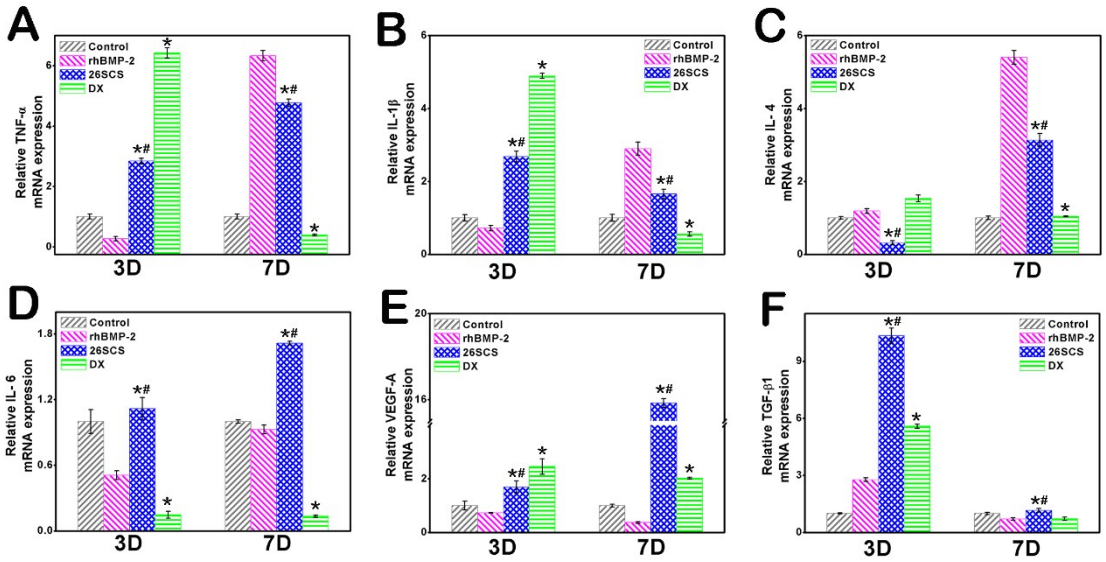

Fig. S4 Relative mRNA expression of TNF-α (A), IL-1β (B), IL-4(EC), IL-6 (D), VEGF-A (E) and TGF-β1 (G) by PM cells cultured in different treatments at day three and day seven. GAPDH was used as housekeeping gene. (*$p<0.05$ and #$p<0.05$ represent statistically a significant difference compared with rhBMP-2 group and DX group, respectively).

With the change of the PM phenotype, gene expressions in PM cells were investigated by the RT-qPCR. As illustrated in Fig.S4, the pro-inflammatory genes TNF-α (Fig. S4A) and IL-1β (Fig. S4B) as the change of phenotypic changes, which was 9.5 and 2.7 times greater in 26SCS group than in rhBMP-2 group in day three, respectively. In day seven, TNF-α and IL-1β decreased by 25% and 44% respectively by 26SCS group in comparison with rhBMP-2 group. Compare to rhBMP-2 and 26SCS group, expressions of TNF-α and IL-1β in DX group are higher in day three, and lower in day seven. The anti-inflammatory gene IL-4 (Fig. S4C) in 26SCS group was less than rhBMP-2 group both in day three and seven. It is worth noticeable that the IL-6 expression (Fig. S4D) of 26SCS group has a significant increase ($p<0.05$) compared to other groups in three and seven days. Except for these inflammatory factors, PM also secrete some osteogenesis-related genes. The gene expression for both VEGF-A and TGF-β1 (Fig. S4E, F) was significantly up-regulated ($p<0.05$) in

response to 26SCS group compared with other groups.

Table 1 Quantitative realtime RT-PCR primer sets

Gene	Direction	Sequence(5′-3′)
GAPDH (mouse)	Forward	TGACCACAGTCCATGCCATC
	Reverse	GACGGACACATTGGGGGTAG
IL-1β	Forward	CTGTCCTGTGTAATGAAAGA
	Reverse	TTGGGTATTGCTTGGGATCCACA
IL-6	Forward	ATAGTCCTTCCTACCCCAATTTCC
	Reverse	GATGAATTGGATGGTCTTGGTCC
TNF-α	Forward	CTGAACTTCGGGGTGATCGG
	Reverse	GGCTTGTCACTCGAATTTTGAGA
IL-4	Forward	TCTTGATAAACTTAATTGTCTCTCGTCAC
	Reverse	GCAGGATGACAACTAGCTGGG
VEGF-A	Forward	GTCCCATGAAGTGATCAAGTTC
	Reverse	TCTGCATGGTGATGTTGCTCTCTG
TGF-β1	Forward	CGGAAGTGAGGCAGGTAG
	Reverse	ACGTAGTAGACGATGGGCAG