Platelet-rich plasma in mono-segmental posterior lumbar interbody fusion: a novel molecular mechanism

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Received: 15 July 2011 / Accepted: 18 August 2011 / Published online: 30 August 2011
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To the Editor,

I read with great interest the article by Sys and colleagues [1]. This work showed that Platelet-rich plasma (PRP) can promote bone healing in spinal fusion. I would like to complete the discussion of Sys and colleagues by introducing a major route which PRP could promote healing.

Impaired fracture healing and diminished bone formation are related to the increased rates of callus resorption associated with osteoclast activity [2]. Studies have shown the essential role of the ligand for receptor activator of nuclear factor kappa B (RANKL) in recruitment of the osteoclasts. The recruitment effect of RANKL can be inhibited by osteoprotegerin (a decoy receptor of RANKL) [3, 4]. Also, inhibition of the noncanonical Wnt pathway in osteoclast precursors suppresses RANKL-induced osteoclastic differentiation [5]. Vascular endothelial growth factor (VEGF) through enhancement of β -catenin activity can inhibit the noncanonical Wnt pathway [6, 7].

Platelet-rich plasma, also named platelet-rich concentrate, is essentially small amount of plasma enriched with platelets. Recent studies have shown that PRP stimulate the osteoprotegerin production which can lead to significant reduction in RANKL. Also, PRP induce production of VEGF that triggers inhibition of osteoclastic differentiation through down regulation of the noncanonical Wnt pathway [8, 9]. Therefore, these important mechanisms should be borne in mind as the major mechanisms for PRP-induced bone healing in spinal fusion.

Conflict of interest There is no actual or potential conflict of interest.

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