

Platelet-Rich Plasma-Induced Analgesia in Peripheral Nerve Repair: Mechanisms, Cautions, and Clinical Promise [Response to Letter]

Damien P Kuffler¹, Onix Reyes², Ivan J Sosa³, William F Micheo⁴, Jose M Santiago-Figueroa⁵ , Christian A Foy⁵

¹Institute of Neurobiology, Medical School, University of Puerto Rico, San Juan, PR, 00901, USA; ²Doctor's Center Hospital, Manati, PR, 00674, USA; ³Department of Neurosurgery, Medical School, University of Puerto Rico, San Juan, PR, 00936-5067, USA; ⁴Department of Physical Medicine and Rehabilitation, Medical School, University of Puerto Rico, San Juan, PR, 00936-5067, USA; ⁵Section of Orthopedic Surgery, Medical School, University of Puerto Rico, San Juan, PR, 00936-5067, USA

Correspondence: Damien P Kuffler, Institute of Neurobiology, Medical School, University of Puerto Rico, 201 Blvd. del Valle, San Juan, PR, 00901, USA, Tel +1 787 721 0778, Email dkuffler@hotmail.com

Dear editor

The authors appreciate the letter to the editor regarding our paper and fully agree with all its statements and the critical questions raised. Like the authors of the letter, the authors of the paper were equally surprised by the consistent, rapid, and long-term neuropathic pain suppression/elimination. The speed of action supports the letter authors' suggestion that the analgesia was exerted by platelet-released factors acting at the site of PRP application by suppressing nociceptive axon excitability. In agreement with the letter, our paper states that leukocytes in the PRP are known to release pro-inflammatory cytokines and TNF- α , suggesting that their presence may be counterindicated. However, under stressful conditions, leukocytes also secrete opioids, which activate peripheral opioid receptors.¹ They also release other analgesic mediators, including anti-inflammatory cytokines, somatostatin, and endocannabinoids, which induce analgesia by inhibiting the excitability of sensory nerves and/or releasing excitatory neuropeptides.¹ The letter authors also correctly point out that leukocytes can induce fibrotic scar formation, which can restrict axon regeneration. However, because every subject whose nerve gaps were repaired using this PRP recovered sensory and motor function,² indicating that, if scar tissue was induced, it did not prevent axon regeneration. This is supported by our observation of one subject, whose pain was eliminated while recovering excellent sensory and motor function, with whom we had the opportunity to examine their nerve gap repair site one year post-repair. Scarring was only seen around the proximal repair end, while the rest of the regenerated nerve appeared normal and scar-free. The letter authors question whether the collagen tube might exert adverse effects. None were encountered, and in the case mentioned above, no vestiges of the collagen tube were seen. A significant comment in the letter is that there is no consistency in PRP composition in different studies, resulting in some PRP inducing little to no analgesia, while others induce far more. Moving forward requires others testing the efficacy of the PRP we used, but also, as the letter points out, determining the best way to prepare and apply PRP to evoke maximum and long-term analgesia, and determining the mechanisms by which PRP reduces/eliminates neuropathic pain.

Disclosure

The authors report no conflicts of interest in this communication.

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