Pierce Surgical Consolidated Inc.

DESIGN NOTES

Form 0400-2	*Researcher-Potential User Notes*
Rev 00	See SOP 8800 for Use

Report Date: Dec 1-2015
Subject: Flowable BioMatrix vs PRP Web Survey
Test or Original Work Date(s):
Participants: Javin Pierce

Clin Orthop Relat Res. 2015 May;473(5):1635-43. doi: 10.1007/s11999-015-4192-2.

Platelet-rich concentrates differentially release growth factors and induce cell migration in vitro.

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Abstract

BACKGROUND:
Platelet-rich concentrates are used as a source of growth factors to improve the healing process. The diverse preparation protocols and the gaps in knowledge of their biological properties complicate the interpretation of clinical results.

QUESTIONS/PURPOSES:
In this study we aimed to (1) analyze the concentration and kinetics of growth factors released from leukocyte- and platelet-rich fibrin (L-PRF), leukocyte- and platelet-rich plasma (L-PRP), and natural blood clot during in vitro culture; (2) investigate the migration of mesenchymal stem cells (MSCs) and human umbilical vein endothelial cells (HUVECs) as a functional response to the factors released; and (3) uncover correlations between individual growth factors with the initial platelet/leukocyte counts or the induced cell migration.

METHODS:
L-PRF, L-PRP, and natural blood clot prepared from 11 donors were cultured in vitro for 28 days and media supernatants collected after 8 hours and 1, 3, 7, 14, and 28 days. Released transforming growth factor β1 (TGF-β1), vascular endothelial growth factor (VEGF), insulin growth factor (IGF-1), platelet-derived growth factor AB (PDGF-AB), and interleukin-1β (IL-1β) were measured in the supernatants with enzyme-linked immunosorbent assay. Migration of MSC and HUVEC induced by the supernatants was evaluated in Boyden chambers.

RESULTS:
More TGF-ß1 was released (mean ± SD in pg/mL of blood) from L-PRF (37,796 ± 5492) compared with L-PRP (23,738 ± 6848; $p < 0.001$) and blood clot (3739 ± 4690; $p < 0.001$), whereas more VEGF and IL-1ß were released from blood clot (1933 ± 704 and 2053 ± 908, respectively) compared with both L-PRP (642 ± 208; $p < 0.001$ and 273 ± 386; $p < 0.001$, respectively) and L-PRF (852 ± 376; $p < 0.001$ and 65 ± 56, $p < 0.001$, respectively). No differences were observed in IGF-1 and PDGF-AB released from any of the concentrates. TGF-β1 release peaked at Day 7 in L-PRF and at 8 hours and Day 7 in L-PRP and 8 hours and Day 14 in blood clot. In all concentrates, main release of VEGF occurred between 3 and 7 days and of IL-1β between Days 1

and 7. IGF-1 and PDGF-AB were released until Day 1 in L-PRP and blood clot, in contrast to sustained release over the first 3 days in L-PRF. The strongest migration of MSC occurred in response to L-PRF, and more HUVEC migration was seen in L-PRF and blood clot compared with L-PRP. TGF-β1 correlated with initial platelet counts in L-PRF (Pearson r = 0.66, p = 0.0273) and initial leukocyte counts in L-PRP (Pearson r = 0.83, p = 0.0016). A positive correlation of IL-1β on migration of MSC and HUVEC was revealed (Pearson r = 0.16, p = 0.0208; Pearson r = 0.31, p < 0.001).

CONCLUSIONS:
In comparison to L-PRP, L-PRF had higher amounts of released TGF-β1, a long-term release of growth factors, and stronger induction of cell migration. Future preclinical studies should confirm these data in a defined injury model.

CLINICAL RELEVANCE:
By characterizing the biologic properties of different platelet concentrates in vitro, we may gain a better understanding of their clinical effects and develop guidelines for specific future applications.

PMID:25690170 [PubMed - indexed for MEDLINE] PMCID:PMC4385378 [Available on 2016-05-01]

Red Blood Cells in Injectable Biologics Friend or Foe?

Endothelial nitric oxide synthase in red blood cells: Key to a new erythrocrine function? ☆

- Miriam M. Cortese-Krott⁎,
- Malte Kelm

http://www.sciencedirect.com/science/article/pii/S221323171400010X

Highlights

- We define *erythrocrine function* the ability of RBC to secret signaling entities or transmitter molecules.
- Erythocrine function include scavenging, transporting and producing NO metabolites and ATP.
- There is in vitro and in vivo evidence of a role of red cell eNOS in signaling and*erythrocrine* function.
- Red cell eNOS and erythocrine function might be involved in organ protection.

Further studies should address the role of red cell eNOS/RBC signaling in cardiovascular health.