Editorial

Transforming growth factor beta (TGF-β): natural curing agents for repair

Volume 1 Issue 2 - 2014

Farshid Sefat
Department of Chemistry, Chemical Biology and Biomedical Engineering, Stevens Institute of Technology, USA

Correspondence: Farshid Sefat, Tissue Engineering Group, Department of Chemistry, Chemical Biology and Biomedical Engineering, Stevens Institute of Technology, Hoboken, New Jersey, USA, Tel 2017023337, Email farshid.sefat@gmail.com

Received: June 23, 2014 | **Published:** August 01, 2014

Editorial

There are various techniques to enhance tissue regeneration via the application of growth factors to the site of regeneration to induce cells to proliferate, differentiate and regenerate. Generally, direct application of growth factors has little effect[1] because the growth factor diffuses out from the site of regeneration very quickly. This is a problem that can be solved by a controlled release of growth factor at the site of action over a long period of time by use of a bioabsorbable scaffold. Growth factors are protein based molecules in the body which are produced by cells and attach to the cell surface. Growth factors bind to membrane receptors, which in turn activate an intracellular signalling pathway. This will activate or inhibit a gene causing either an up regulation or down regulation of a gene product, which then alters the cells behaviour.

The Transforming Growth Factor Beta (TGF-β) super family include different proteins such as BMPs, nodals, activins, TGF-β and inhibins.[2] TGF-β is a cytokine produced by different cell types inside the body and exists in three isomers TGF-β1, 2 and 3.[3] These isomers can act as either paracrine or endocrine hormones and are structurally similar and encoded by three distinct genes in mammalian species.[4] All three isoforms have been found[5] to have overlapping functions *in vivo* with different biological activities *in vitro*. TGF-β1 is more potent throughout adult development, whereas TGF-β3 is more potent in tissues with mesenchymal origin. The three isoforms signal through the same kinase receptor with different binding abilities.[6–8] The TGF-β family of cytokines regulate cell functions such as migration, apoptosis, proliferation and differentiation.[9] It is important to know where the target cells are located as TGF-β can have different effects in different cell environments. For example, in skin cells when fibroblasts are grown in a monolayer in the presence of epidermal growth factor, TGF-β1 causes a decrease in proliferation. In comparison, when these cells are grown in a semi-solid medium, TGF-β1 induces growth.[10] TGF-β is also associated with the scarless healing of skin[1] and it plays a role in the repair of other tissues including cartilage and bone.[11–13] This cytokine influences number of other cell activities such as differentiating, stimulating mesenchymal stem cell (MSC) growth, acting as a chemotactic factor and also enhances ECM production, secretion and bone regeneration.[14]

There is evidence that the TGF-β have significant effects on bone structure by regulating the replication and differentiation of chondrocytes, osteoblasts and osteoclasts.[15] Application of TGF-β promoted bone healing in skull defects and tibia1 fractures[16] and continuous infusion of TGF-β increased bone formation and fracture healing,[17] suggesting TGF-β as a potential therapeutic agent for fracture repair. However, previous studies have shown[1,17] that there is limited research regarding the use of TGF-β isomers in bone cells in terms of fracture repair and healing compared to other cell types such as keratinocytes,[18–20] and also compared to other growth factors such as BMP,[21] platelets-derived growth factor (PDGF), insulin-like growth factor (IGF)[22] and fibroblast growth factors (FGF).[23] Therefore, there is a huge potential to carry out further research on the effect of different TGF-β isomers as a natural curing agent on bone repair and its related mechanism.

Acknowledgements

None.

Conflict of interest

The author declares no conflict of interest.

References

1. Khan SN, Bostrom MP, Lane JM. Bone Growth factors. *Orthop Clin North Am*. 2000;31(3):375–388.
2. Derynck R, Miyazono K. TGF–β and the TGF–β Family. In: Derynck R, Miyazono K, editors. *The TGF–β Family*. USA: Cold Spring Harbor Laboratory Press; 2008. p. 29–43.
3. Worthington JJ, Klementowicz JE, Travis MA. TGFβ: a sleeping giant awoken by integrins. *Trends in Biochem Sci*. 2011;36(1):47–54.
4. Mehta JL, Attramadal H. The TGFβ superfamily in cardiovascular biology. *Cardiovasc Res*. 2007;74(2):181–183.
5. Sodeka J, Lia WC, Lib H, et al. The role of TGF–β and BMP–7 in regenerating bone and soft tissues. *Materials Science and Engineering C*. 1994;2(1–2):19–26.
6. Massague J. TGF–beta signalling: receptors, transducers, and Mad proteins. *Cell*. 1996;85(7):947–950.
7. Barrientos S, Stojadinovic O, Golinko MS, et al. Growth factors and cytokines in wound healing. *Wound Repair Regen*. 2008;16(5):585–601.
8. Sefat F, Beggs CB, Denyer MCT, et al. The Effects of different HCl concentrations on wound healing of bone cell monolayer. *Proceedings of the World Congress on Engineering (WCE)*. 2010;1:542–549.
9. Krauss G. *Biochemistry of signal transduction and regulation*. Wiley–VCH; 2003. 3 p.
10. Border WA, Noble NA. Transforming growth factor beta in tissue fibrosis. *N Engl J Med*. 1994;19(10):1286–1292.
11. Sefat F, Youseffi M, Berends RF, et al. Wound Closure Properties of TGF–β3, BSA/HCl and HCl in Cultured Human Bone Cell Monolayer. *Proceedings of the World Congress on Engineering (WCE)*. 2009;2:1866–1871.

12. Beggs CB, Denyer MCT, Lemmerz A, et al. The effect of transforming growth factor beta (TGF–β3) and Sanicle on wound healing. *Proceedings of the World Congress on Engineering (WCE)*. 2010;1:572–577.

13. Sefat F, Denyer MCT, Youseffi M. Effects of different Transforming Growth Factor Beta (TGF–β) Isomers on Wound Closure of Bone Cell Monolayers. *Cytokine*. 2014;69(1):75–86.

14. Lind M, Schumacker B, Soballe K, et al. Transforming growth factor–beta enhances fracture healing in rabbit tibiae. *Acta Orthop Scand*. 1993;64(5):553–556.

15. Sefat F, Denyer MC, Youseffi M. Imaging via Widefield Surface Plasmon Resonance microscope for studying bone cell interactions with micropatterned ECM proteins. *J Microsc*. 2011;241(3):282–290.

16. Critchlow MA, Bland YS, Ashhurst DE. The effect of exogenous transforming growth factor beta–2 on healing fractures in the rabbit. *Bone*. 1995;16(5):521–527.

17. Tosounidis T, Kontakis G, Nikolaou V, et al. Fracture healing and bone repair: an update. *Trauma*. 2009;11(3):145–156.

18. Sun T, Adra S, Smallwood R, et al. Exploring hypotheses of the actions of tgf–β1 in epidermal wound healing using a 3d computational multiscale model of the human epidermis. *PLoS One*. 2009;4(12):e8515.

19. Shirakata Y. Regulation of epidermal keratinocytes by growth factors. *J Dermatolo Sci*. 2010;59(2):73–80.

20. Mauviel A. Transforming growth factor–beta signaling in skin: stromal to epithelial cross–talk. *J Invest Dermatol*. 2009;129(1):7–9.

21. Croteau S, Rauch F, Silvestri A, et al. Bone morphogenetic proteins in orthopaedics: from basic science to clinical practice. *Orthopaedics*. 1999;22(7):696–700.

22. Thaller SR, Dart A, Tesluk H. The effect of insulin like growth factor–1 on critical– size calvarial defects in Sprague–Dawley rats. *Ann Plast Surg*. 1993;31(5):429–433.

23. Hurley MM, Abreu C, Harrison JR, et al. Basic fibroblast growth factor inhibits type 1 collagen gene expression in osteoblastic MC3T3E–1 cells. *J Biol Chem*. 1993;268(8):5588–5593.

Citation: Sefat F. Transforming growth factor beta (TGF-β): natural curing agents for repair. *MOJ Cell Sci Rep*. 2014;1(2):35–36.
DOI: 10.15406/mojcsr.2014.01.00009