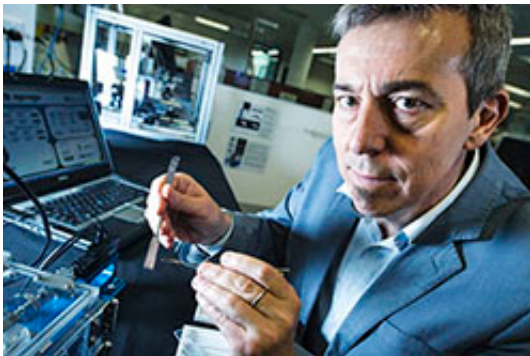


Therapy to prevent failure of 3-D printed body parts discovered

December 20 2016, by Niki Widdowson



Professor Dietmar W Hutmacher. Credit: Queensland University of Technology

A key physiological process holding back the successful integration of 3-D printed replacement body parts for many patients has been discovered by a team of biomedical scientists.

An article published in *Nature Biomedical Engineering* details how the researchers found that an anti-osteoporotic drug and a vascular growth factor inhibitor could prevent the process that leads to implant failure.

Professor Dietmar W Hutmacher, from QUT's Australian Research Centre in Additive Biomanufacturing based in the Institute of Health and Biomedical Innovation, said medical devices and implants often failed because the patient's body rejected them as foreign bodies.

"These implants elicit a foreign body response (FBR) and cause fibrotic encapsulation or scar [tissue](#) to develop around the implant which impairs its intended function in the body," Professor Hutmacher said.

"We had to understand better the mechanisms behind the body's response before we could find ways to change and/or guide the process so the 3-D printed biodegradable scaffold can slowly dissolve away as it is replaced by new living tissue at later stages in the regeneration process.

"We know that when tissue is exposed to biomaterial it triggers a step-wise process of inflammation, followed by wound healing and, if not resolved from a pro-inflammatory to a pro-reparative or regenerative mode of action, tissue fibrosis and scarring."

Professor Hutmacher said the team studied the mechanistic roles of two white blood cell types - monocytes and macrophages – key to the body's [regeneration process](#).

"There are two types of macrophages – M1 macrophages cause inflammation and M2 macrophages are pro-regenerative.

"Both are implicated in the formation of fibrous scarring around the implant yet we did not fully understand the mechanism by which they either enhanced or counteracted the formation of fibrotic scarring in the context of an additive biomanufactured scaffold.

"Our research into their role found that M1 macrophages joined and became immobilized along the scaffold surface before forming multinucleated giant cells which initiated a network of neovessels within the scaffold pores that acted in a way similar to the neovessels that support the growth of fibrous tissue around a tumour.

"These macrophages and their giant cells produced a growth factor which led to the formation of the dense collagen capsule around the implant two to four weeks later."

Professor Hutmacher said these insights into the scaffold integration [process](#) within soft tissue suggested therapeutic intervention.

"We found that administration of clodronate, an anti-osteoporosis drug, and VEGF Trap, an anti-vascular endothelial [growth factor](#) drug, significantly reduced giant-cell accumulation, and the formation of neovessels and fibrosis.

"This represents a promising strategy to modulate both early inflammation and later stage tissue remodelling (weeks to month) to minimize chronic FBR and implant failure."

More information: Eleonora Dondossola et al. Examination of the foreign body response to biomaterials by nonlinear intravital microscopy, *Nature Biomedical Engineering* (2016). [DOI: 10.1038/s41551-016-0007](#)

Provided by Queensland University of Technology

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