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Modifying mineralized collagen scaffolds to modulate the inflammatory response in craniomaxillofacial defects.

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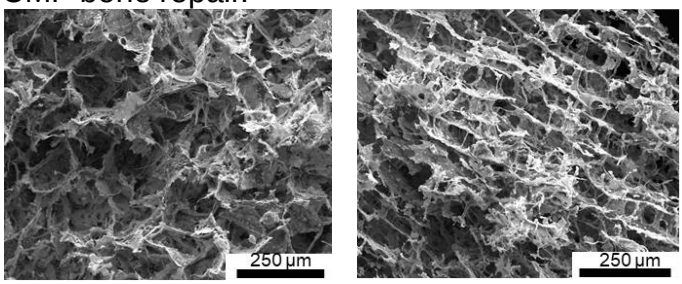
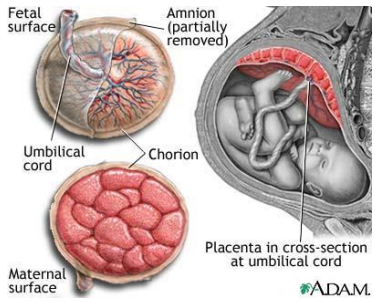
ABSTRACT:

Craniomaxillofacial (CMF) bone defects can arise from congenital, post-oncologic, and traumatic injuries. Recent studies examining battlefield injuries (BI) experienced by U.S. Soldiers in Iraq and Afghanistan found 26% of all survivable BI localized to the maxillofacial area. Current methods of repair involve autografts or allograft sources for bone. Our laboratory has developed a class of mineralized collagen scaffold able to promote mesenchymal stromal cell (MSC) osteogenic differentiation and subsequent CMF bone regeneration in the absence of exogenous growth factor supplements. While such regenerative medicine strategies offer the potential for improving the scope and speed of CMF bone regeneration, the host inflammatory environment may limit the implant integration and subsequent bone regeneration. Therefore, an opportunity exists to develop degradable biomaterials that enhance bone regeneration by modulating the host inflammatory response.

My central hypothesis is that scaffold pore size, anisotropy, and incorporation of an allogenic matrix source can modulate macrophage phenotype and resultant MSC osteogenesis. While pore size and anisotropy introduce biophysical cues, incorporation of placenta derived amniotic/chorionic matrix into the collagen scaffold may provide additional biomolecular stimuli to alter macrophage response. This work provides insight on the impact of structural and compositional biomaterial cues on the inflammatory response underlying CMF bone repair.



Pore size and anisotropy could modulate macrophage phenotype



Incorporation of an allogenic matrix could modulate macrophage phenotype

AWARDS/PUBLICATIONS:

- Chemistry-Biology Interface (CBI) program trainee