Weekly Update

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1. Weekly Progress

1.1. Summarizing Wound Healing

1. Vascular response
   - Platelets aggregate at the wound to prevent loss of blood
   - Blood vessels near the wound site constrict or dilate to either prevent blood flow or bring in more white blood cells
2. Inflammatory response
   - Neutrophils arrive to phagocytize any bacterial inhibition
   - Monocytes phagocytize any dead tissue near the wound site
3. Granulation tissue formation
   - New fibroblasts and blood vessels (angiogenesis) enter the wound
   - New extracellular matrix (ECM) proteins are synthesized and the ECM begins to form
4. Contraction of the wound
   - Fibroblasts become more specialized and turn into myofibroblasts
   - The myofibroblasts contains actin filaments, which contract the wound
5. Wound remodeling
   - Continued formation of the ECM
   - Long-term process to regain original strength of skin

1.2. Literature Review

- Study: *A mathematical model for the capillary endothelial cell-extracellular matrix interactions in wound-healing angiogenesis* (Olsen, Sherratt, Maini, Arnold)

- STUDY: *A mathematical model of wound healing and subsequent scarring* (Cumming, McElwain, Upton)
- STUDY: *A mathematical model for collagen fibre formation during foetal and adult dermal wound healing* (Dale, Sherratt, Maini)
- STUDY: *Monitoring wound healing in a 3D wound model by hyperspectral imaging and efficient clustering* (Wahabzada, et. al.)
- STUDY: *Computational Approach To Characterize Causative Factors and Molecular Indicators of Chronic Wound Inflammation* (Nagaraja, et. al.)

2. To-Do

- ☐ How fibroblast proliferation occurs
- ☐ Number of fibroblasts/ideal density of fibroblasts
- ☐ Density vs healing time