# THE ROLE OF ANGIOGENESIS IN TISSUE TRANSPLANT

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**Background**. Ischemia reperfusion injury (IRI) is one of the most important mechanisms involved in delayed or reduced graft function after transplantation. It is a complex pathophysiological process, followed by a pro-inflammatory response that enhances the immunogenicity of the graft and the risk of acute rejection. Tracheal, cartilage and skin allotransplantation have historically been unfeasible because of the absence of a vascular pedicle for anastomosis. Stimulating angiogenesis may be a solution.

**Objective of the study**. To elucidate the mechanism of angiogenesis in order to combate ischemia reperfusion injury in transplants.

**Materials and methods**. A review of the literature from 2016-2026 was performed, using 11 articles, including data from ScienceDirect, PubMed Central, Biomed Central, MedScape, and others.

**Results**. Angiogenesis is the process of new blood vessel formation from pre-existing network through *endothelial cell* (EC) migration and proliferation playing a crucial role in post-transplant recovery and organ function, with decreased activity potentially indicating graft failure. In rewieved literature have been mentioned effectiveness of supplementing transplants with *Fibroblast Growth Factor 2* (FGF-2) or *Vascular Endothelial Growth Factor (*VEGF), VEGF hydrogel*,* fibrin precipitate, *sphingosine-1-phosphate* (S1P), erythropoietin, *induced pluripotent stem cells* (iPSCs). Unfortunately, these methods promote the formation of small-caliber vessels that are prone to stenosis and thrombosis. While angiogenesis is induced by hypoxia and results in new capillaries, arteriogenesis is induced by physical forces, most importantly fluid shear stress and mast cells in orchestrating leukocyte function in this process. Fluid shear stress, induce the adherence of platelets to ECs, a process mediated by the interaction of the platelet receptor *Glycoprotein Ib α* (GPIbα) with the endothelial *von Willebrand factor* (vWF). Subsequently resulting in *platelet–neutrophil aggregate* (PNA) formation and neutrophil extravasation.

**Conclusions.** In conclusion it may be effective to use physical methods in promoting arteriogenesis. For example, vacuum aspiration of the plague before skin-graft transplantation. On other hand it may be efficient to supplement transplants with preconditioned mast cells originate from hematopoietic stem cells.

**Key-words:** Neoangiogenesis, Arteriogenesis, IRI, Fluid shear stress.