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Overcoming blood group and antibody barriers by kidney paired donation

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Approximately one third of kidney transplant candidates has medically acceptable living donors but is unable to receive transplants due to donor - recipient incompatibilities. Kidney Paired Donation (KPD) is a strategy that matches incompatible pairs in order to find compatible matches, thus increasing living donor transplantation. Significant strides have been made since the idea was first conceptualised. However, the technique remains under utilized. This lecture is intended to highlight the current advances, types of paired donation programs, registries, world-wide experience, outcomes, barriers and limitations.

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miRNA-200b inhibits epithelial-mesenchymal transition in TGF-β1 induced human bronchial epithelial cells

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Introduction: The role of TGF-β1 in mediating extracellular matrix remodeling during the pathogenesis of lung fibrosis has been well defined. Dysregulated expression of miRNA is increasingly implicated in various pathological processes and may play an important role in progressive loss of lung function.

Material & Methods: The differential expression of miRNAs was assayed using NanoStringTM in untreated and 1, 4 and 24 hours TGF-β1 treated BEAS-2B (immortalized human bronchial epithelial cells) cells. BEAS-2B and PBECs were transfected with miR-200b mimics to study expression of EMT markers at mRNA and protein level. MiRNA targets were identified and validated using multiple computational tools and qRT-PCR respectively. In situ hybridization allowed detection of miR-200b in tissues samples at cellular level.

Results: NanoStringTM allowed identification of differentially expressed miRNAs post TGF-β1 treatment. MiR-200b mimic transfection followed by TGF-β1 treatment demonstrated a significant increase in E-cadherin ($p \leq 0.05$, $p \leq 0.001$) and a significant decrease in fibronectin ($p \leq 0.001$, $p \leq 0.01$) in BEAS-2B cells and PBECs. Protein studies suggested a similar trend in both the cells. MiR-200b significantly reduced the expression of its targets ZNF532 ($p \leq 0.01$) and ZEB2 ($p \leq 0.001$) in BEAS-2B cells and ZNF532 ($p \leq 0.01$) in PBECs post TGF-β1 treatment. Furthermore, In situ hybridization allowed localization of miR-200b in airway epithelium of normal human lung sections.

Conclusion: The findings suggest that ectopic expression of miR-200b restore TGF-β1 induced EMT in BEAS-2B cells and PBECs. The outcomes from this study may offer new insights into mir-200b regulation in fibrosis and have potential for therapeutic application in progressive airway diseases.

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