Possible Role of VEGF-B/VEGF-A Relationship in Ischemic Coronary Artery Disease

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Ischemic heart disease is a major global health issue, responsible for 17.9 million deaths annually. In Qatar, 69% of deaths result from three major diseases: cardiovascular disease (24%), cancer (18%), and diabetes (7%). Vascular endothelial growth factors (VEGFs) play a crucial role in blood vessel growth and repair. Among them, VEGF-B is involved in metabolism regulation, vascular development, and protection against oxidative stress. This study explores how VEGF-A and VEGF-B work together to influence heart function and regeneration in ischemic heart disease. To replicate heart attack conditions, we exposed cultured heart cells and zebrafish models to hypoxia (low oxygen). Various treatments were applied, including VEGF-A, VEGF-B, their combination, and hypoxia alone. The expression of genes linked to inflammation and heart health was analyzed, while zebrafish heart function and development were carefully monitored. Results showed that hypoxia increased inflammation in endothelial cells and suppressed essential cardiac genes in cardiomyocytes. It also reduced zebrafish survival, heart rate, and heart chamber size. However, VEGF-A and VEGF-B had protective effects. Their combined overexpression under hypoxic conditions reduced inflammation, enhanced cardiac gene expression, and improved zebrafish heart function and beat rate. These findings confirm that VEGF-B has a protective role against hypoxia. Combining VEGF-A and VEGF-B enhances heart function and regeneration, making it a potential strategy for treating myocardial infarction and heart failure.

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