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Complexity of angiogenesis during myocardial ischaemia

Abstract:

One of the key problems of heart disease is the failure to restore rapid revascularisation following coronary artery blockage leading to cardiomyocyte cell death resulting in reduced pumping capacity although surgical interventions have provided considerable help. Part of this problem may arise from considering all endothelial cell responses to be similar in different tissue settings irrespective of the host tissue type characteristics. Use of generic endothelial cell lines or easily available primary endothelial cells to characterise angiogenic cell responses will not be identical in all tissue types. This certainly would not apply to highly specialised tissues like the cardiac muscle that loses its ability to proliferate or regenerate during late fetal or early neonatal development. Unlike many other tissues like angiogenic responses during dermal tissue repair, VEGF supply alone fails to trigger angiogenesis during ischaemic myocardial events. This study will describe why angiogenesis in adult ischaemic myocardium is much more complex than simply upregulating a single VEGF signalling component. Furthermore, this study will highlight how the highly dynamic and dysregulated balance of selective VEGF inhibitors and enhancers contributing to this problem may be used to overcome the problem of revascularisation of ischaemic myocardium to restore function.

Biography

My initial training in muscle specialisation began in the laboratories of professor S V Perry and Prof Philip Gell using biochemical and immunochemical approaches at the University of Birmingham that later included molecular biology approaches for application to embryonic muscles. Unlike some myosins and troponin C, these studies identified troponin I and troponin T to be cardiac muscle type specific that are now routinely used to assess cardiac muscle damage in the clinic. My later study as a Fulbright scholar at the university of Pennsylvania during a sabbatical break identified a key novel enzyme important in cell signalling. The present study takes advantage of both these approaches to determine why it has been so difficult to trigger angiogenesis in ischaemic myocardium.