Erythrocytes (RBCs) play an important role in the regulation of the microcirculation via the release of the potent vasodilator, adenosine triphosphate (ATP) when these cells are exposed to reduced oxygen tension. In skeletal muscle, the release of ATP from RBCs in areas of the muscle in which oxygen need increased ensures that blood flow (oxygen delivery) is accurately matched with tissue oxygen need.

In type two diabetes this mechanism is defective, that is, low oxygen-induced ATP release is severely impaired. In this talk I will present evidence in support of the use of insulin and C-peptide to correct this defect in erythrocyte physiology. In addition a role for phosphodiesterase 5 inhibitors in the therapy of the peripheral vascular disease of type 2 diabetes will be suggested. Both approaches suggest new approaches to the treatment of peripheral vascular disease as well as delayed wound healing in humans with type 2 diabetes.
Recent relevant publications:


