## Sample Abstract

The following sample abstract is taken from: *Phoenix, Kathryn (2008).*


The Role of Activation of AMP-dependent Kinase (AMPK) in Endothelial Cell Proliferation

AMP-dependent kinase (AMPK) is a primary energy sensor that controls energy use and production during metabolic cellular stress, such as hypoxia and nutrient deprivation. AMPK activation results in inhibition of anabolic processed and promotion of catabolic processes. AMPK has also been shown to be a target of metformin, a first line therapy for type 2 diabetes. Treatment with metformin has been shown to potently decrease cell proliferation. Preliminary studies revealed that metformin promoted angiogenesis and vascular stability in an *in vivo* breast tumor model. Importantly, clinical studies have revealed that type 2 diabetic patients treated with metformin experienced improved vascular function when compared to those on other treatments. The major aim of this study was to evaluate the *in vitro* effects of metformin on endothelial cell proliferation as a possible mechanism for increased cell survival and angiogenesis. Human umbilical vein endothelial cells were treated with metformin and evaluated for cell proliferation, viability and kinase activation. Metformin treatment resulted in decreased cell numbers without affecting viability. AMPK activity was increased with metformin treatment. Interestingly, mitogen activated protein kinase (MAPK), a kinase involved in proliferation control, was increased with metformin treatment despite the significant reduction in cell numbers. Additionally, AMPK activation has been shown to promote the expression of a

major angiogenic cytokine vascular endothelial growth factor (VEGF) VEGF expression was increased in response to metformin treatment. Since endothelial cells express VEGF receptors, the promotion of mitogenic signaling possibly resulted from autocrine signaling with increased VEGF expression. Stimulation of this pathway promotes angiogenesis. This study demonstrates that while metformin decreases proliferation of endothelial cells, it is not through the repression of the MAPK pathway. The combination of these events could lead to the improved angiogenesis seen in vivo with metformin treatment and result in improved vascular stability and function patients with diabetes.