

Research findings breathe new life into lung disease

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It turns out the muscle cells on the outside of blood vessels have been wrongly accused for instigating lung disease. New research shows that while these muscle cells are responsible for constricting or dilating the blood vessels, they are not responsible for sensing the amount of oxygen that gets to the lungs. That message comes from the endothelial cells – special cells that line the blood vessels – along a "signalling pathway."

When a person is low on oxygen, blood vessels throughout the body expand to improve the delivery of this vital molecule to the tissues. The one exception is that when oxygen is low in the lungs, blood vessels there constrict. When this condition persists, it causes <u>pulmonary hypertension</u> – a <u>lung disorder</u> where the arteries that carry blood from the heart to the lungs become smaller – and makes it difficult for the heart to pump blood through the lungs. This leads to enlargement of the right heart, called right heart failure.

Dr. Wolfgang Kuebler, a Li Ka Shing Knowledge Institute scientist at St. Michael's Hospital, has found that the endothelial cells play a much larger role in the constriction of blood vessels in response to the lack of oxygen and in subsequent pulmonary hypertension, than previously believed. The findings of his study, which was conducted on mice, have been published online in the <u>Journal of Clinical Investigation</u>, a high impact basic science journal.

"The vascular endothelial cells have always been regarded as a bystander, but we've discovered that lung disease in response to low oxygen



originates at this level, that the message is sent by these cells," said Dr. Kuebler. He said that if there were a way to block, or inhibit, this communication along the signalling pathway between the endothelial cells and the <u>smooth muscle cells</u>, we could potentially prevent right heart failure, a fairly common disease among patients with <u>lung disease</u>.

Provided by St. Michael's Hospital

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