Growing new arteries, bypassing blocked ones

Yale UniversityYale University

Scientific collaborators from Yale School of Medicine and University College London (UCL) have uncovered the molecular pathway by which new arteries may form after heart attacks, strokes and other acute illnesses — bypassing arteries that are blocked. Their study appears in the April 29 issue of Developmental Cell.

Arteries form in utero and during development, but can also form in adults when organs become deprived of oxygen — for example, after a heart attack. The organs release a molecular signal called VEGF. Working with mice, the Yale-UCL team discovered that in order for VEGF-driven artery formation to occur, VEGF must bind with two molecules known as VEGFR2 and NRP1, and all three must work as a team.

The researchers examined mice that were lacking a particular part of the NRP1 molecule that transports VEGF and VEGFR2 to a signaling center inside blood vessel walls. They observed that the internal organs of these mice contained poorly constructed arterial branches. Further, the mice where unable to efficiently repair blood vessel blockage through the formation of new arteries.

“We have identified an important new mechanism that regulates VEGFR2 transport in vascular cells,” said corresponding author Michael Simons, professor of medicine and cell biology, and director of the cardiovascular research center at Yale School of Medicine. “This opens new therapeutic opportunities for developing drugs that would either stimulate or inhibit blood vessel formation — important goals in cardiovascular and anti-cancer therapies, respectively.” Simons also has an appointment as honorary professor of medicine at UCL.

The Yale-UCL collaboration began more than three years ago, as an intensive global effort to improve the human condition through biomedical research and translational medicine. The Yale-UCL alliance has provided many opportunities to date for high-level scientific research, and clinical and educational collaboration.

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