Coming Events

2014

Neuroscience 2014
November 15–19, 2014
Washington, DC
www.sfn.org

EM Structure Determination
November 17–20, 2014
La Jolla, CA
http://nramm.scripps.edu/2014-workshop

Electron Tomography Conference
November 17–20, 2014
Riviera Maya, Mexico
www.zingconferences.com/conferences/electrontomography

MRS Fall Meeting
November 30–December 5, 2014
Boston, MA
www.mrs.org/fall2014

ASCB Annual Meeting
December 6–10, 2014
Philadelphia, PA
http://am.ascb.org/meetings

2015

Quantitative BioImaging Conference
January 7–9, 2015
Institute Pasteur, Paris, France
www.quantitativebioimaging.com

MSC-SMC Annual Meeting
May 26–29, 2015
Hamilton, Ontario, Canada
www.bimr.ca/events/msc-smc-annual-meeting-2015

Microscopy & Microanalysis 2015
August 2–6, 2015
Portland, OR
www.microscopy.org

2016

Microscopy & Microanalysis 2016
July 24–28, 2016
Columbus, OH
www.microscopy.org

2017

Microscopy & Microanalysis 2017
July 23–27, 2017
St. Louis, MO
www.microscopy.org

2018

Microscopy & Microanalysis 2018
August 5–9, 2018
Baltimore, MD
www.microscopy.org

2019

Microscopy & Microanalysis 2019
August 4–8, 2019
Portland, OR
www.microscopy.org

More Meetings and Courses
Check the complete calendar near the back of this magazine.

Carmichael's Concise Review

Coronary Arteries Arise from Two Distinct Sources

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The development of coronary arteries that supply the heart muscle (myocardium) is a fundamental biologic question with profound ramifications for human health and disease. A better understanding of the embryology of these vessels could potentially lead to the development of therapies to repair damaged myocardium and reduce deaths from heart disease.

Although not rigorously tested, coronary vessels have been presumed to arise as a continued growth of embryonic vessels after an animal is born. Recently Xueying Tian, Tianyuan Hu, Hui Zhang, Lingjuan He, Xiuzhen Huang, Qiaozhen Liu, Wei Yu, Liang He, Zhen Yang, Yan Yan, Xiao Yang, Tao Zhonu, William Pu, and Bin Zhou [1] have demonstrated in a postnatal mouse model that there is another source of coronary arteries that allows the rapidly enlarging heart to receive adequate vascular support (Figure 1).

Early in embryonic development, coronary vessels form as a vascular plexus that covers the outer surface of the heart. At this early stage the underlying ventricular walls are composed of a relatively thin compact layer and a much more extensive trabecular layer, the latter made up of multiple fingerlike projections into the ventricular cavities. With ongoing development, the compact myocardium develops coronary vessels from within, whereas the trabeculations directly exchanges nutrients with blood within the chamber without requiring blood vessels. But prior to birth the trabecular myocardium coalesces with the outer compact myocardium and now requires its own blood supply. The mechanism for the ongoing postnatal vascularization of these newly developed compact ventricular walls has not been known.

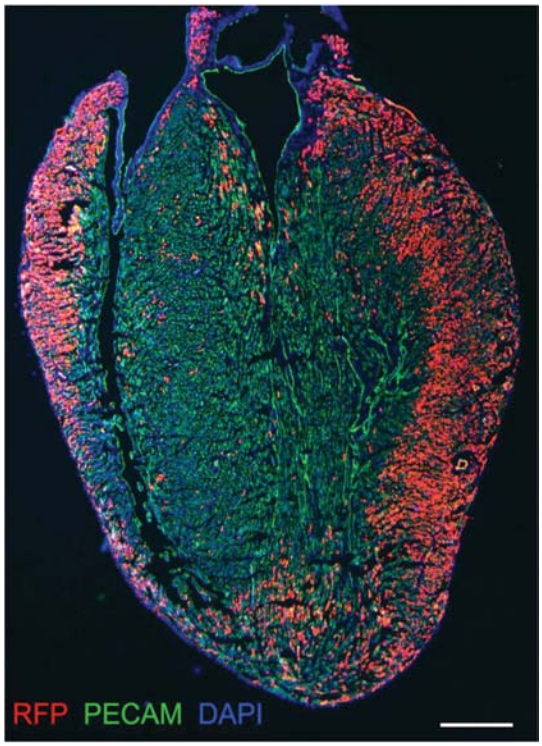

Figure 1: Section of tamoxifen-treated (at embryonic day 10.5) mouse heart at postnatal day 7 stained with red fluorescent protein (RFP), platelet endothelial cell adhesion molecule (PECAM), and 4',6-diamidino-2-phenylindole (DAPI) a well-known marker for nucleic acids. The RFP stains cells that arise from embryonic vessels, the cells stained with PECAM are bona fide coronary vessels that arise postnatally, and the DAPI stains nuclei. Scale bar is 0.5 mm.

Several different microscopic techniques were used to differetiate this second source of coronary arteries from the ones that originated prenatally. These include stereo microscopy, immunofluorescence microscopy, and confocal microscopy. Tian et al. first permanently labeled embryonic vascular endothelial cells (VECs). They found that at certain postnatal time periods these labeled cells were confined to the outer portions of the myocardial wall. Unlabeled endothelial cells accumulated in the inner portions of the wall. They were identified by positive markers for VECs, but clearly they were not derived from the embryonic vessels.

To define the developmental origin for the vessels formed *de novo* in the inner portions of the

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heart wall, Tian et al. developed a strain of mice that allowed embryonic endothelial cells to be labeled distinctly from the cells that developed postnatally. A series of ingenious experiments showed that in the inner myocardial wall, VECs arise by lineage conversion of neonatal endocardial cells. The interventricular septum, like the inner myocardial wall, is also vascularized by lineage conversion of endocardial cells to coronary VECs. The authors suggest that there had been postnatal "compaction" of the trabeculations to form the innermost layer of myocardium. This aspect remains controversial. Tian et al. nonetheless offer compelling evidence that this inner layer is supplied by vessels that arise *de novo*, and not from the embryonic VECs

This newly discovered dual origin of coronary arteries presents a new mechanism for a rapidly developing functional vascular supply of the myocardium. This novel discovery will need to be verified and the responsible molecular mechanisms elucidated. It will also need to be established whether the trabecular layer of the myocardium does, indeed, compact to form the inner part of the ventricular walls. Still, the findings regarding the formation of the coronary vasculature are not only an exciting addition to our knowledge of heart development, but have important implications for cardiac diseases and cardiac regenerative medicine!

References

[1] X Tian et al., *Science* 345 (2014) 90–94.

[2] The authors gratefully acknowledge Dr. Bin Zhou for reviewing this article.

MT

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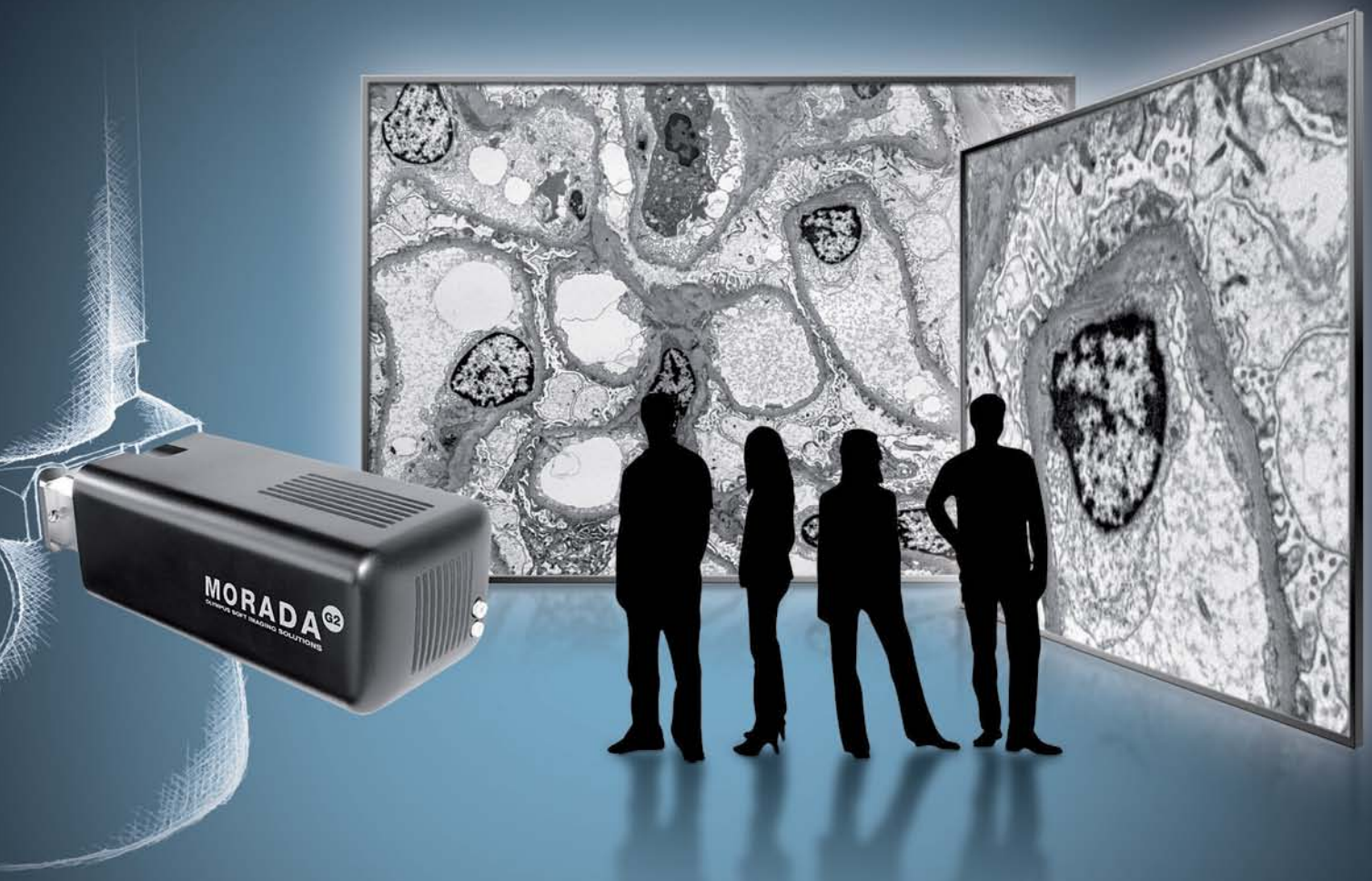

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