By Elizabeth Macintyre, for Passaro et al. Cancer Cell

AngioCRIME in Acute Myeloid Leukemia (AML)

AMLs have stubbornly resisted attempts to improve overall outcome using chemotherapy, epitherapy and immunotherapy approaches, including stem cell transplantation. Passaro et al use intravital 2-photon microscopy to demonstrate that this may reflect too much concentration on the leukemic population and not enough on the angiocrine factors (growth factors, trophogens and chemokines) produced by bone marrow vascular endothelial cells hosting the leukemic blasts. They show that the latter induce vascular abnormalities, including leakiness and hypoxia, mediated by increased reactive oxygen and nitric oxide (NO) production, which is resistant to chemotherapy in patient-derived xenografts. NO inhibition reduced vascular permeability and improved both normal stem cell function and leukemic blast response to chemotherapy, thus raising promise for use of NO inhibitors against the angiocrine culprits in acute myeloid leukemogenesis.

Vascular Leakiness in the Bone Marrow of AML Patient-Derived Xenografts

Links to: Passaro D et al. Cancer Cell 32, 324-341, September 11, 2017, PMID 28870739; <https://www.ncbi.nlm.nih.gov/pubmed/28870739/>

And the accompanying commentary, Itkin T and Rafii S, Cancer Cell 32, 276-278; <https://www.ncbi.nlm.nih.gov/pubmed/28898691>

Vascular Leakiness in the Bone Marrow of AML Patient-Derived Xenografts

Elizabeth Macintyre

Hematology, Paris
Descartes University,
Necker-Enfants Malades

Hospital (AP-HP), and INSERM

UMR1151, Paris, France

Correspondence: Elizabeth Macintyre,

(e-mail: elizabeth.macintyre@aphp.fr).

Funding/support: nothing to disclose.

The authors has indicated that she has no potential conflicts of interest to disclose.

Copyright © 2017 the Author(s).

Published by Wolters Kluwer Health, Inc. on behalf of the European

Hematology Association. This is an open access article distributed under the terms of the Creative Commons

Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to

download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

HemaSphere (2017) 1:1(e15)

<http://dx.doi.org/10.1097/HSE.000000000000015>

HS9.000000000000015