


CAREER DEVELOPMENT AWARDEES

	<p>Rosa Bernardi</p>
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Title	<b>Giovanni Armenise-Harvard Career Development Awardee</b>
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Curriculum Vitae	
Education	<ul style="list-style-type: none"> <li>• 1993: Graduate in Biology, major in Molecular Biology, University of Pavia, Italy.</li> <li>• 1997: Ph.D. in Genetics and Molecular Biology, University of Pavia, Italy.</li> </ul>
Professional Experience	<ul style="list-style-type: none"> <li>• 1997-2000 Post-doctoral Fellow, Fels Institute for Cancer Research, Temple University, Philadelphia, USA.</li> <li>• 2001-2006 Postdoctoral Fellow, Memorial Sloan-Kettering Cancer Center, New York, USA.</li> <li>• 2006-2007 Senior Research Scientist, Memorial Sloan-Kettering Cancer Center, New York, USA.</li> <li>• 2007-2008 Research Associate, Beth Israel Deaconess Medical Center; Instructor, Harvard Medical School, Boston, USA</li> </ul>
Association memberships	

<p>Research Interests</p>	<p>Neo-angiogenesis is the process of forming new capillaries from pre-existing blood vessels. While it is firmly established that neo-angiogenesis plays an important role in the development, progression and metastasis of solid tumors, accumulating evidence is recently suggesting that neo-angiogenesis may also play a role in the pathophysiology of hematological malignancies. This hypothesis rests mainly on clinical studies, which show that leukemia patients express high levels of pro-angiogenic factors and have increased angiogenesis in the bone marrow. However, these findings are mostly correlative: genetic proof that angiogenesis supports leukemogenesis is still lacking and the molecular mechanisms leading to angiogenesis in leukemia have not yet been established.</p> <p>We are interested in better understanding causes and consequences of the expression of pro-angiogenic factors in leukemia. While performing these studies, we also aim at creating animal models that better mimic human leukemia and that will be utilized in pre-clinical studies to test the efficacy of anti-angiogenesis therapies for treating leukemia.</p>
<p>Selected Publications</p>	<p>Bernardi R., Scaglioni P.P., Bergmann S., Horn H.F., Vousden K.H., Pandolfi P.P. (2004) "PML regulates p53 stability by sequestering Mdm2 to the nucleolus" <i>Nat. Cell Biol.</i> 6, 665-72.</p> <p>Grisendi S., Bernardi R., Rossi M., Cheng K., Khandker L., Manova K., Pandolfi P. P. (2005) "Role of nucleophosmin in embryonic development and tumorigenesis" <i>Nature</i> 437, 147-53.</p> <p>Bernardi R., Guernah I., Jin D., Grisendi S., Alimonti A., Teuya-Feldstein J., Cordon-Cardo C., Simon M. C., Rafii S., Pandolfi P. P. (2006) "PML inhibits Hif-1<math>\alpha</math> translation and neoangiogenesis through repression of mTOR" <i>Nature</i> 442, 779-785.</p> <p>Bernardi R. and Pandolfi P. P. (2007) "Structure, dynamics and functions of promyelocytic leukaemia nuclear bodies" <i>Nature Reviews Mol. Cell Biol.</i> 8, 1006-1016.</p> <p>Ito K., Bernardi R., Morotti A., Matsuoka S., Saglio G., Ikeda Y., Rosenblatt J., Avigan D. E., Teruya-Feldstein J. and Pandolfi P. P. (2008) "PML targeting eradicates quiescent leukaemia-initiating cells" <i>Nature</i> May 11 [Epub ahead of print].</p>
<p>Laboratory Members</p>	