

## Marrow Fibrosis Does Not Account for Circulating CD34<sup>+</sup> Cells in Myelofibrosis with Myeloid Metaplasia

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In the recent article in this journal <sup>1</sup>, Leibundgut EO, *et al.*, and the accompanying editorial <sup>2</sup> by Rosti and Massa, the authors discussed a potential role for endothelial progenitor cells (EPCs) in the myeloproliferative disorders. We agree this is a very important topic since a possible involvement of a common progenitor of both myeloid and endothelial cells in myeloproliferative disorders by a common somatic mutation would be an important contribution to our understanding of the pathophysiology of these disorders. In addition to the issues raised by Dr. Yoder's letter, with whom we fully agree, we would like to comment on a statement that alteration of the bone marrow environment, as a consequence of the extensive fibrosis, might favor the progenitor cell mobilization in myelofibrosis with myeloid metaplasia (MMM). We had in past also considered the logical possibility that the fibrosed marrow is a principal cause of CD34 mobilization to peripheral blood. However, in our previously published study we have conclusively ruled out the possibility. Specifically, we evaluated 25 patients with MMM and 19 patients with secondary myelofibrosis associated with pulmonary hypertension.<sup>3,4</sup> These data demonstrate definitively that, despite the presence of marrow fibrosis in both patients population, high circulating CD34<sup>+</sup> cell count, (and also the presence of clonal platelets and granulocytes, peripheral blood dacrococytes,

and the presence of the JAK2 V617F mutation) are unique identifiers of the clonal progenitors associated with MMM. Based on these data, we submit that the hypothesis that marrow fibrosis may account for the circulating CD34<sup>+</sup> cells found in MMM should be forever abandoned.

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### References

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