patients with past thrombotic diathesis presented the same abnormality.

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To the Editor:

In their study of the DNA levels (ploidy) of murine megakaryocyte colonies, Chatelain and Burstein evaluated the effects of acute thrombocytopenia upon the ploidy of megakaryocyte colonies derived from mice that had received platelet antiserum. They reported that colonies derived from bone marrow, obtained 24 hours after the administration of platelet antiserum, demonstrated the same ploidy distribution as did control colonies, in agreement with our observations when marrow or spleen was cultured 48 hours after the administration of platelet antiserum.

However, the authors' sweeping conclusion that "An acute thrombocytopenic environment does not influence the ultimate ploidy of the progeny of CFU-M" is not justified. While they are correct in stating that a similar result was described by us two days following thrombocytopenia, they also should have indicated that when colonies were derived from bone marrow or spleen four or five days after the induction of acute thrombocytopenia, we observed a marked shift in ploidy of megakaryocyte colonies. Although the basis for the intriguing observation that CFU-Meg are not reprogrammed to generate colonies of increased ploidy until four to five days after production of acute thrombocytopenia with platelet antiserum remains to be explained, our previously published data establish that a thrombocytopenic environment can influence the ultimate ploidy distribution of the progeny of CFU-Meg. In addition, our studies emphasize the importance of serial observations if one is to detect important but delayed alterations in megakaryopoiesis, manifested in cultures of bone marrow or splenic hematopoietic cells.

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REFERENCES


To the Editor:

We agree with Dr Levin that useful insights may be garnered from serial observations following administration of platelet antiserum (APS). However, the purpose of our experiment was to examine the ploidy of the progeny of megakaryocytic colony-forming cells (CFU-Meg) at a time when these progenitors were exposed to an "acute thrombocytopenic environment." Twenty-four hours following a single injection of APS, the platelet count is at its nadir; therefore, we chose this time to perform our experiment. Four to five days following APS injection, the environment is not thrombocytopenic; rather, the platelet count has recovered to normal or above normal levels. Thus, we disagree that previously published data "establish that a thrombocytopenic environment can influence the ultimate ploidy distribution of CFU-Meg." On the contrary, it remains to be established whether the changes described by Levin et al., occurring four to five days following a single dose of APS (at a time when the platelet count had returned to normal), were related to the preceding thrombocytopenia, rather than some other consequence of APS administration.

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Ploidy distribution of CFU-Meg progeny [letter]

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