in a limited number of cases (14 normal subjects, and three patients with acute nonlymphoblastic leukemia) that leukemic progenitors were less sensitive than normal CFU-GM to alpha and gamma recombinant interferon (Boehringer Ingelheim).

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REFERENCES

To the Editor:

We are appreciative of the thoughtful comments by Delforge et al. With regard to their first point, it is possible that gamma, as well as alpha, interferon was present in our lymphocyte conditioned medium. Nevertheless, it is unlikely that significant differential effects resulted as linear dose-response curves were observed with each interferon. Furthermore, Broxmeyer et al reported similar data on day 14 granulocyte/monocyte colony-forming cells (CFU-GM) using cell line giant cell tumor (GCT) conditioned medium as a source of colony-stimulating activity, thus lacking any potential interferon contamination.1

Second, the colonies in our cultures were clearly CFU-GM in morphology and we have confirmed this through Wright-Giemsa staining. Leukemic colonies from donor I did not display typical CFU-GM morphology but, instead, were less well differentiated. Significant lymphocyte colony contamination seemed a most unlikely possibility, given that 100% of the patient’s leukocytes were blasts.

Finally, we look forward to the evolution and publication of their preliminary studies on leukemic progenitor cells. Potential differences between their observations and ours may result as a consequence of their use of two sources of colony stimulating activity in their cultures, as well as the innate variability of myeloid leukemia cells.

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REFERENCE

ANTI-COMMON ACUTE LYMPHOBlastic LEUKEMIA ANTIBODY (CALLA) (J5) REACTIVITY BY SMALL CELL LUNG CANCER (SCLC) CELLS

To the Editor:

In a recent article in Blood, Bunn et al1 reported the expression of lymphocytic antigens (Leu-7, Leu-11, OKT10, OKM1, Leu-M1, Leu-M2, OKT9) as detected by immunofluorescence analysis on cell lines deriving from oat cell carcinoma of the lung. In another article in Science, Ruff and Pert2 describe the presence of OKM1 antigen on two SCLC cell lines and four SCLC autopsy specimens; they propose that the observed expression of OKM1 by SCLC may indicate the origin of SCLC from bone marrow macrophages; in contrast to the proposed theory, Gazdar et al3 point out that shared antigenicity does not necessarily indicate common lineage or embryologic origin.

We are reporting here for the first time the binding of the J5 (anti-CALLA)4 monoclonal antibody to SCLC cells deriving from a bone marrow specimen of a patient with lung, bone, and bone marrow involvement by SCLC at an advanced disease stage. Flow cytometric analysis of the SCLC cells stained by an indirect immunofluorescent technique showed that 16% of such cells were J5 positive. In addition, 41% of the small cells were Leu-M1 positive and 22% were Leu-9 positive. The La (HLA-DR related) antigen as well as kappa and lambda chains and the MO2 antigen were not detected on the cell surface. Leukemic cells were not observed in the bone marrow smear or on biopsy specimen.

The J5 monoclonal antibody has also been reported to react with adult and fetal renal proximal tubule cells, adult breast myoepithelium5 as well as common bile duct cells. Although CALLA antigenic expressions occur in cultured marrow and skin fibroblasts6 there is not enough evidence to prove that SCLC derives from such ancestor cells. Mature granulocytes also express CALLA.7 The biologic significance of such expression by normal or cultured cells remains to be proven.

We agree with Gadzar et al that caution should be applied in

REFERENCES
Anti-common acute lymphoblastic leukemia antibody (CALLA) (J5) reactivity by small cell lung cancer (SCLC) cells [letter]

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