5q− Acute Myelogenous Leukemia: Reply

To the Editor:

Professor Van den Berghe and his colleagues have made important observations on the clinical states associated with the 5q− abnormality in patients with hematologic disorders. The lack of reports confirming the presence of this abnormality should not lead to the impression that it is only a "Belgian disease."

I have studied five patients who had the 5q− chromosome. Although each patient had a different clinical history, they all terminated in acute myelocytic leukemia (AML), usually with a relatively rapidly fatal course. All five of the patients were female. One had AML on presentation (karyotype 43, XX, 2q−, inv3, −4, 5q−, −7, 17p+, −19); another (Case 1 of Ref. 1) had subacute leukemia for 4 mo prior to the acute phase (karyotype 48, XX, +1, −5q, +11), and a third had pancytopenia with a hyperplastic marrow (Vilter type IV) for 8 mo prior to AML (karyotype 46, XX, 4q−, 5q−, −16, −17, +2 markers). The fourth patient had myelofibrosis with myeloid metaplasia complicated by red cell aplasia requiring frequent transfusions for 3½ yr prior to the onset of the leukemic phase. A significant percentage of circulating blasts was present for 1 yr prior to the need for chemotherapy. On the initial marrow examination, she showed a mosaic pattern, 46, XX, 5q−/47, XX, 5q−, +21, which persisted until her death. The fifth patient had had multiple myeloma for three years, and had developed acute myelogenous leukemia when her chromosomes were first examined [45, XX, t(13q;14q), 5q−]; the translocation was a constitutional abnormality. Conditions in which the 5q− chromosome may be observed may include a broader spectrum than the usual myeloproliferative disorders, as evidenced by its presence in a patient who had multiple myeloma. As with the patients described by Van den Berghe et al., most of these cases showed other aberrations superimposed on the 5q− abnormality.

The fourth patient was followed with chromosome analyses for 3½ years prior to the development of acute leukemia. Sokal et al.[2] have reported observation periods of up to 5 yr. It is apparent, however, that many more patients will have to be followed carefully before we can be certain that the 5q− abnormality is invariably a preleukemic sign.

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Folate Radioassay and Crude Milk Binder

To the Editor:

In a recent paper from our laboratory on serum and red cell folate radioassay,[1] we reported that an agent present in some milk samples seemed to release serum folate from its endogenous binding protein. This agent was removed during purification of milk by acid precipitation. For these reasons, the assay we described used crude milk as a source of binder, removing the necessity for a heating step[2,3] to release bound folate.

Further studies in our laboratory support the concept that levels of saturated serum folate binder are elevated in certain subjects, most commonly in uremia and pregnancy.[4] Folate radioassay[1] in some of these subjects gives very low results compared with *L. casei* microbiologic assay, even when the binder used is crude milk containing the agent which seems to release serum folate from its binder. Attempts are currently being made to purify this agent in the hope that it will, in increased concentration, release folate from binders in all sera. However, we have not excluded the possibility that qualitative rather than quantitative alterations in binder account for the low results mentioned

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