

Letter to the Editor

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The preceding letter was shown to Dr. H.G. Drexler who offers the following reply.

IN GENERAL, Orfao *et al.* confirm our conclusion that "... the FMC7 antigen was not found on immature B-cells, appeared at intermediate B-cell stages, reached the peak of expression at the late B-cell stage and was lost at the B-cell/plasma cell transition" [1]. By showing that 20% of the cases with B-CLL were FMC7-positive, Orfao *et al.* underlined the fact that this marker cannot discriminate on its own between B-CLL and B-PLL or HCL. In our study no distinction was made between "classical" B-CLL, B-PLL and intermediate groups.

Several recently published papers showed clearly that B-CLL and B-PLL are just two ends of a continuous spectrum regarding the number of prolymphocytes per case [2-4]. Melo *et al.* [2] proposed a classification system of B-CLL and PLL: "typical" CLL (< 10% prolymphocytes); a group designated as CLL/PL (11-55% prolymphocytes); and PLL (> 55% prolymphocytes). Unfortunately, these authors did not give the numbers of cases in each category which were FMC7-positive.

The intermediate CLL/PL group appeared to be heterogeneous including two types of CLL, one with increased percentages of prolymphocytes (but

otherwise typical disease), and another in "prolymphocytoid" transformation [2]. Scott *et al.* confirmed these observations and noted that while only 17% of CLL cases (38/223) with < 10% prolymphocytes were FMC7-positive, more than 90% of CLL cases (40/44) with > 10% prolymphocytes were stained by FMC7 [both groups together: 30% of CLL cases (78/267) were FMC7-positive] [3]. In another study, these authors showed that 21/22 cases of B-CLL in prolymphocytoid transformation were FMC7-positive [4]. In summary, these data clearly indicate that while PLL and HCL are generally FMC7-positive (although apparently 10% might be FMC7-negative), variable numbers of B-CLL cases are FMC7-positive due to the heterogeneity of this type of chronic B-cell disorder and because of discordant definitions of study groups.

Furthermore, differences in the staining intensities of positive cells might explain certain discrepancies. Like surface immunoglobulin (which is strongly visible by immunofluorescence on PLL cells and generally only weakly on CLL cells), some CLL cases might be stained only weakly for FMC7, and depending on other variables (quality and amount of second layer, type of microscope of flow cytometer, age of reagents, experience of observer etc.) might escape detection.

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REFERENCES

1. Drexler HG, Menon M, Gaedicke G, Minowada J. Expression of FMC7 antigen and tartrate-resistant acid phosphatase isoenzyme in cases of B-lymphoproliferative diseases. *Eur J Cancer Clin Oncol* 1987, **23**, 61-68.
2. Melo JV, Catovsky D, Galton DAG. The relationship between chronic lymphocytic leukaemia and prolymphocytic leukaemia. I. Clinical and laboratory features of 300 patients and characterization of an intermediate group. *Br J Haematol* 1986, **63**, 377-387.
3. Scott CS, Limbert HJ, Roberts BE, Stark AN. Prolymphocytoid variants of chronic lymphocytic leukaemia: an immunological and morphological survey. *Leuk Res* 1987, **11**, 135-140.
4. Stark AN, Limbert HJ, Roberts BE, Jones RA, Scott CS. Prolymphocytoid transformation of CLL: a clinical and immunological study of 22 cases. *Leuk Res* 1986, **10**, 1225-1232.