Comment to: The clinical presentation and prognosis of diffuse large B-cell lymphoma with t(14;18) and 8q24/c-MYC rearrangement. Haematologica 2007; 92:1335-1342

In a series of sixteen cases of diffuse large B-cell lymphomas (DLBCL) with t(14;18)(q32;q21) and 8q24/MYC rearrangements, le Gouill *et al.*[1] report one case with a t(8;9)(q24;p13) translocation. They assert, what is not true, that this 8q24/*MYC* translocation variant was never reported in DLBCL before. In a recent article,[2] we described a series of B-cell lymphomas carrying 8q24 rearrangements with non-immunoglobulin partners. Among them were three cases harbouring a t(8;9)(q24;p13) translocation, each of them found in DLBCLs with a t(14;18). In the discussion, le Gouill et al write that *PAX5* was identified as the partner gene but do not provide any indication regarding their technical approach, nor any experimental result. We cannot agree with this way to deliver such an affirmation, as searching for PAX5 rearrangement in our cases, we found a more complex situation in all cases. In our work, two breakpoints from the t(8;9) were cloned and a third one mapped using FISH. All three breakpoints were located several hundred kilobases upstream from *PAX5*. In one cloned case the breakpoint was located upstream *ZBTB5* exon2 and in the other case upstream *ZCCHC7*. Molecular cytogenetics of the third case showed that the breakpoint was inside *ZCCHC7*. RT PCR experiments showed that these two genes were expressed (not shown). To assess a possible distant effect of the translocation on *PAX5* regulation, we measured its relative expression by real-time quantitative RT PCR using the Taqman technology. Primers and probe were: 5'-TCCCAGCTTCCAGTCACAGC-3', 5'-ATCCGTGCTCACCGAGGAC-3', and 5'-CCACTGGCTCCGTGACGCAGG-3', respectively. Eleven DLBCL with a t(14;18) but without 9p13 rearrangement were used as controls. Relative expression levels (mean[95% confidence interval]) for controls and t(8;9) translocations were 0.661 [0.397-0.925] and 0.709 [0.349-1.068], respectively.

The absence of a significant difference of *PAX5* expression in samples with/without the t(8;9), together with distant breakpoints from the gene let us to conclude that *PAX5* cannot be considered, in our series, as the partner gene of the translocation. Our three cases displayed breakpoints dispersed on a 200-300kb region and we do not exclude the possibility that, in other t(8,9), a breakpoint located more closely to PAX5 could deregulate its expression. However, this must be demonstrated and rather than affirmed.

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