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Please note that an error appeared in the original online early release version of this article. A portion of text from the second paragraph in the Results and Discussion section was erroneously deleted. The current html, print, and pdf versions appear correctly. For reference, the corrected paragraph appears below in its entirety.

We next assessed if these long-lived t(14;18)-bearing B cells in HI already transited through the GCs. Circulating naive and memory B cells can be distinguished on the basis of the CD27 cell surface marker and sIg isotypes (11–13). Fresh PBMCs from 10 healthy donors were collected, and $IgD^+/CD27^-$ naive, $IgD^+/CD27^+$ memory, and $IgD^-/CD27^+$ switched memory B cell subsets isolated by cell sorting (Fig. 2 A). The occurrence of t(14;18) was then assessed in total PBMCs and in each fraction by the short-range $BCL2/J_H$ PCR assay (Fig. 2 B and Table II). As a first approach, we pooled data from the two $CD27^+$ memory subsets and examined the overall contribution of naive ($CD27^-$) and memory ($CD27^+$) B cells to the total $t(14;18)^+$ frequency calculated as a fraction of $CD19^+$ B cells (Fig. 2 C). t(14;18) frequencies of $CD19^+$ B cells ranged from $<1/10^5$ to the unexpectedly high rate of $\sim$1/3.500 B cells in some individuals (Fig. 2 C, squares). Strikingly, although the level of naive $t(14;18)^+$ cells constantly remained at baseline (Fig. 2 C, circles), $CD27^+$ B cells accounted in a large part for the amplitude of t(14;18) frequencies (Fig. 2 C, triangles). This clearly indicates that circulating $t(14;18)^+$ clones in HI are indeed predominantly B cells which transited through the GCs. To determine if the presence of high levels of t(14;18) in some individuals was caused by a higher incidence of distinct translocations or to the clonal expansion of a given $t(14;18)^+$ B cell, we cloned and sequenced 55 out of 61 $BCL2/J_H$ fragments from the $CD27^+$ subset. One major $BCL2/J_H$ junction was observed in most individuals (Fig. 2 D, black bars), indicating that only one clone mainly accounted for t(14;18) frequencies. Remarkably, this data demonstrate that the wide modulation of t(14;18) frequency in HI is not caused by the accumulation of clonally unrelated t(14;18) naive B cells in some individuals, but rather to the clonal expansion in the GCs of $t(14;18)^+$ B cells.