Dendritic Cell Activation Kinetics and Cancer Immunotherapy
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Lack of Association between Human Switch Recombination Breakpoints and the Secondary Structure of Targeted DNA Regions

As class switch recombination (CSR) is a region, rather than a site-specific event, a relationship between the recombination breakpoints and the structural character of the switch (S) regions involved has been sought. It has earlier been suggested that CSR preferentially occurs at transitions from a stem to a loop structure in ssDNA (microsites) in S regions from a variety of species (1, 2). However, only a limited number of breakpoints have been analyzed (1, 2). In the October 1, 2003 issue of The Journal of Immunology, Cameron et al. (3) showed that three of the four breakpoints described (Sμ, Sε, and Sγ) from nasal tissue also mapped to microsites and suggested that their observations represented the first evidence for a structural recognition pattern in primary human B cells. However, we have previously shown, using a large number of Sγ breakpoints from in vivo switched human B cells, that the percentage of breakpoints at microsites is not higher than expected by chance (4). We have now reanalyzed our previously published Sγ breakpoints (n = 68) and added another 130 Sμ and 62 Sε breakpoints, using the standard applied by Tashiro et al. (2). As shown in Table I, the percentage of breakpoints at microsites is not higher than expected by chance (χ² test), even though many S junctions are indeed located at (position 0), or in the proximity of (position 1), these sites (for full data see www.biosci.ki.se/users/qipa/microsites). Therefore, new ways of exploring the role of secondary and tertiary structure of the S regions are required to explain the location of the switch recombinational breakpoints.

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References

Dendritic Cell Activation Kinetics and Cancer Immunotherapy

In a recent paper published in The Journal of Immunology, Watanabe and colleagues (1) report on the impact of dendritic cell (DC) activation kinetics on the in vivo priming of Ag-specific T lymphocytes. Thus, the authors focus on a central issue in DC-based immunotherapy, which has already been addressed both in vitro (2, 3) and in vivo (4), reaching very similar conclusions. Worthy of note, Watanabe and colleagues (1) show that as early as 3 h after anti-CD40 Ab stimulation, DCs already developed into powerful cellular vaccines. We found that DCs produced IL-12 and marginally up-regulated cell surface molecules as early as 15 min after exposure to promaturation stimuli, but as for human DCs (2) the peak of IL-12 production was reached at 8 h, whereas MHC and costimulatory molecules expression significantly increased only later on (4). Unfortunately neither we (4) nor Watanabe and colleagues (1) show that as early as 3 h after anti-CD40 Ab stimulation, DCs exposed to maturation stimuli produced IL-12 and significantly up-regulated cell surface molecules as early as 15 min after exposure to promaturation stimuli, but as for human DCs (2) the peak of IL-12 production was reached at 8 h, whereas MHC and costimulatory molecules expression significantly increased only later on (4). Unfortunately neither we (4) nor Watanabe and colleagues (1) compared the therapeutic efficacy of 3-h and 8-h DCs. Nonetheless, those data strengthen the conclusion that recently activated DCs (i.e., DCs exposed to maturation stimuli for ≤8 h) or even immature DCs injected into adjuvant-conditioned skin (5, 6) are to be preferred to fully matured DCs (i.e., DCs exposed to maturation stimuli for ≥24 h). Considering that most of the published DC-based vaccination protocols in humans made use of either immature DCs or DCs exposed to maturation stimuli ≥24 h (7), the findings described above suggest that clinicians should revise the schedule for DC activation before in vivo injection.

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References


The Authors Respond

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he letter by Drs. Jeruissen and Bossuyt challenges the veracity of a statement we made in our recent paper by Khan et al. (1) that IgG responses to purified TI-2 (polysaccharide) Ags are T cell independent, unlike the IgG anti-polysaccharide responses that we observe upon challenge with intact extracellular bacteria, which are critically dependent on CD4+ T cells. They cite their own recently published work (2) and that from B. J. Zegers’ lab (3), although they omit citing the much earlier extensive work from P. J. Baker’s group (for review

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play a role in the Ab response to [purified TI-2 Ags]”. Instead, the differences between their published study, and those of others need to be resolved, and thus we should consider this issue as currently controversial.

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References


