

oxidation, the tyrosinase-related proteins TRP-1 and TRP-2, as well as MelanA/MART. Trager et al. disclose the issue of immune tolerance, in their writing that, “[h]owever, as [melanoma antigens] . . . are self-antigens, it is known that the immune system establishes immunological tolerance to them either in the thymus or in the periphery.” To summarize, immune tolerance is a problem to be overcome in immunotherapy against various cancers.

III. CONCLUDING REMARKS

The mechanism of action of the study drug is included in the Sponsor’s submissions to the FDA and is included in the package insert of the marketed drug. As a word of caution, there does not exist any “generic immune mechanism” that is applicable to all drugs that

influence the immune system. For any given drug, and for any given disease that is treated with that drug, the mechanism of action of the drug will be different. For any given anticancer drug, that drug will influence the immune system with a different mechanism, depending on the type of cancer. In initiating the task of drafting the mechanism of action of a given drug, the medical writer should separately consider the influence of the drug on the following cells:

1. the cancer cells,
2. CD8⁺ T cells,
3. CD4⁺ T cells,
4. myeloid DCs,
5. plasmacytoid DCs,
6. macrophages,
7. NK cells, and
8. Tregs.