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Melanoma Immunotherapy: Promising against Cancer Cells Growth and for Survival Increase?

Editorial

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Treatment options for advanced cutaneous melanoma remain limited. Yet, melanoma represents only 2-4% of all malignant tumors but remains the most deadly type of skin cancer worldwide (> 50.000 deaths annually equivalent to 75% of all cutaneous cancer-related deaths) due to systemic metastatic spread (about 12% of the cases) [1,3]. The incidence of malignant melanoma has increased fivefold from 1980 to 2009 in Brazil [5].

Currently, there are no effective treatment options for late-stage metastatic melanoma patients (stage III or IV), in spite of few FDA-approved biodrugs (e.g. ipilimumab, vemurafenib, bevacizumab in combination with temolozolomide) that have showed some benefits over dacarbazine (DTIC), a DNA-alkylating agent, in terms of tumor regression, overall survival rate which remains low (about 15%), duration of response, and the median overall survival rarely exceeds 12 months [3].

Undoubtedly, recent immunotherapies constitute innovative cancer strategies [3], beneficiating of studies over the past two decades which have provided insight into several complex molecular mechanisms (e.g. immune escape or suppression for tumor survival). Thereby, a synthetic tyrosinase (*TYR*)-based cancer vaccine targeting melanoma was recently shown a valuable DNA-based therapeutic option due to its efficiency to induce a robust and broad immune response in prophylactic and therapeutic animal models, and direct cancer-killing CD8-T cells infiltration into tumor sites where it prevented tumors, controlled tumor growth, changed the tumor micro-environment by "turning off" cells that suppress T-cell activity (e.g. myeloid-derived suppressor cells (MDSCs)) through the down-regulation of monocyte chemoattractant protein 1 (MCP-1), interleukin-10 (IL-10), chemokine CXCL5 and arginase II, factors important for MDSC expansion, and also increased survival in melanoma-challenged mice versus a control group [9]. A recent study also reported objective responses that have been obtained by CTLA-4 inhibition in metastatic melanoma after *B-RAF* V600-mutant melanoma inhibitor failure [6]. Nevertheless, in spite of recent advances targeted therapy effects in melanoma, often resulting in relatively high response rate and limited duration of response, could be counter balanced if combined with immunotherapy, which usually showed lower response rate but higher duration of response [8,3]. Also, sub-population(s) of melanoma initiating/propagating cells (the so-called "melanoma-stem cells") and their possible specific genetic/genomic alterations could constitute other(s) target(s) [2-4]. The use of innovative technologies and methods (e.g. Carbone-Fluorine Spectroscopy) recently allowed to demonstrate, from animals and humans, the importance of such cell sub-populations in the melanoma spreading [Menaa et al., manuscript in preparation]. New insights shall definitively come up using rationally-designed combinatorial strategies (e.g. drugs plus cell and gene therapies) for enhanced and safer advanced cutaneous melanoma therapy.

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