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Autoimmuno-anti-tumor immunity – understanding the immune responses against “self” and “altered-self”

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The brief description of tumors being “wounds that do not heal” by Dr. Harold F. Dworkar nearly three decades ago (N Engl J Med, 1986) (1) has provided not only a vivid illustration of neoplastic diseases in general but also, in retrospect conceptually, a plausible immunological definition of cancers. Based on our current understanding in the field, it could have even a multi-dimensional meaning attached with. This relates to several important issues, which need to be addressed further, i.e., in terms of a close link between chronic inflammation and tumorigenesis widely observed; clinical and experimental evidence of immunity against tumors versus the highly immunosuppressive tumor microenvironment being associated; and their underlying immunological mechanisms, oncogenic basis, as well as the true causal relationship in question (2–5).

Recent findings from studies into the pathogenesis of autoimmunity and, more importantly, the mechanisms, which protect against it, have offered some new insights for our understanding in this direction. Chronic or persistent autoimmune-like inflammatory conditions are evidently associated with tumor development. The important question is about their true causal relationship. Chronic or persistent inflammation has been shown to contribute directly to tumor development by triggering neoplastic transformation and production of inflammatory mediators, which could promote cancer cell survival, proliferation, and invasion (2, 3). On the other hand, tumors are mutated self-tissue cells to which the host immune system is normally regulated; why dysregulation of the immune system may naturally protect against cancers, while it could also cause autoimmunity – being an evolutionally acceptable “side effect” (Chapters 1–2); followed by explaining how autoimmunity could be a “Double-Agent” involved in both tumor-killing and cancer promotion linked to inflammation (Chapters 3–5). It addresses further by dissecting the detailed cellular and molecular mechanisms potentially involved in these processes (Chapters 6–10). These together may help to provide a good basis for the development of novel therapeutic approaches, including stem cell-based immunotherapy, for future cancer treatment (Chapters 11–15). By understanding how the immune system is normally regulated, why dysregulation of which may cause the immunological–oncological related diseases,
we aim and hope that the contents of this Research Topic can also trigger further active discussions among scientists in the fields, as to how the so-called “self-reactivity” (autoimmune responses) can be alternatively switched on and redirected, immunologically or molecularly, for effective cancer treatment.

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REFERENCES

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