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The brief description of tumours being "wounds that do not heal" by Dr Harold F. Dworak nearly three decades ago (N Engl J Med 1986) 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 tumourigenesis widely observed; clinical and experimental evidence of immunity against tumours versus the highly immunosuppressive tumour microenvironment being associated; and their underlying immunological mechanisms, oncogenic basis, as well as the true causal relationship in question. 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 tumour development by triggering neoplastic transformation and production of inflammatory mediators which could promote cancer cell survival, proliferation and invasion. On the other hand, tumours are mutated self-tissue cells to which the host immune

system is largely tolerized otherwise. Although the mutations may give rise to the expression of tumour-specific antigens (TSA) or tumour-associated antigens (TAA), most of these TSAs/TAAs are found to be poor immunogens. The ongoing inflammatory conditions may therefore reflect a desperate attempt of the host immune system to mount anti-tumour responses, though ineffectively, being a consequence of the continuous yet largely futile triggering by those poorly immunogenic TSAs/TAAs. Furthermore, during autoimmune or overtly persistent immunological responses, many regulatory mechanisms are triggered in the host in attempts to limit the ongoing harmful inflammatory reactions. Such a negative feedback regulation is known to be crucial in preventing normal individuals from immune-mediated diseases. As a result of the negative feedback loop, however, an excessive production of anti-inflammatory or immunosuppressive molecules followed by the exhaustion of the immune effector cells may instead lower the ability of the host immune system to mount specific anti-tumor responses, allowing the escape of tumour or mutated cells from immunosurveillance. This may also help to explain why the most effective way to enhance host immunity against cancer is by targeting the negative arm of immune regulation. In this Frontiers Research Topic, we aim to gather current views from experts in these inherent overlapping fields of oncology, autoimmunity and tumour immunology, and to make them available to our potential readership who may be particularly interested in this cutting-edge area. By understanding how the immune system is normally regulated, why dysregulation of which may cause the immunological-oncological related diseases, we also encourage further discussions as to how the so-called "self-reactivity" (autoimmune responses) can be alternatively switched on and redirected, immunologically or molecularly, for effective cancer treatment.