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Editorial Open Acces

Editorial

T ere is no doubt that cancer is a smart entity, which is evident by several treatment failures in preclinical and clinical trials. In part, host genetic mutations are known to be responsible for the limited success [1]. However, in most of the cases, following therapy, tumors itself acquires resistant properties. In this case, tumor is initially sensitive to the applied treatment and evolves itself to counteract the anti-tumor e fects of drug. On the other hand, de novo resistance is the part of primary refractoriness to a therapy that should have been e fective based on the underlying biology or genetics. According to Newton's third law "for every action, there is an equal and opposite reaction". In cancer therapeutics, tumor exerts strong pro-tumor response against applied treatment. Most of the therapies with short and transient bene f t (measured in weeks or months), have witnessed relapse of malignant tumor growth [2,3]. Resistant tumors are characterized by hyper-vascularity and hyper-invasiveness, for example; breast cancer and glioblastoma [4,5]. Tumor cells become quiescent by reprograming into mesenchymal phenotypes. In epithelial tumors, this phenomenon is well evident and known as epithelial to mesenchymal transition (EMT) [6,7]. In addition, resistant tumor secretes several immunomodulatory signals in the form of secreted factors such as chemokines and growth factors [8]. By doing so, tumor modulates immune cells and governs to impose pro-tumor properties. Heterogeneous population of tumor associated macrophages (TAMs) and regulatory cells are the most abundant pro-tumor and immune-suppressive immune cells known,