using autologous dendritic cells (DCs) pulsed with tumor lysate, peptides, or nucleic acids. Personalized DC vaccines loaded with oxidized whole-tumor lysate have demonstrated the ability to induce broad T-cell responses against both shared and private neoantigens in recurrent ovarian cancer, with vaccine-induced immunity correlating with prolonged progression-free and overall survival in early-phase trials. Integrating such vaccines with agents that modulate the TIME, such as bevacizumab, low-dose cyclophosphamide, or interleukin-2, can further enhance T-cell infiltration, reduce Treg frequencies, and promote polyfunctional effector responses. Neoantigen-based peptide or RNA vaccines and oncolytic viruses designed to release tumor antigens in situ are also under investigation, aiming to convert immunologically "cold" tumors into "hot" lesions amenable to checkpoint blockade (Garlisi et al., 2024).

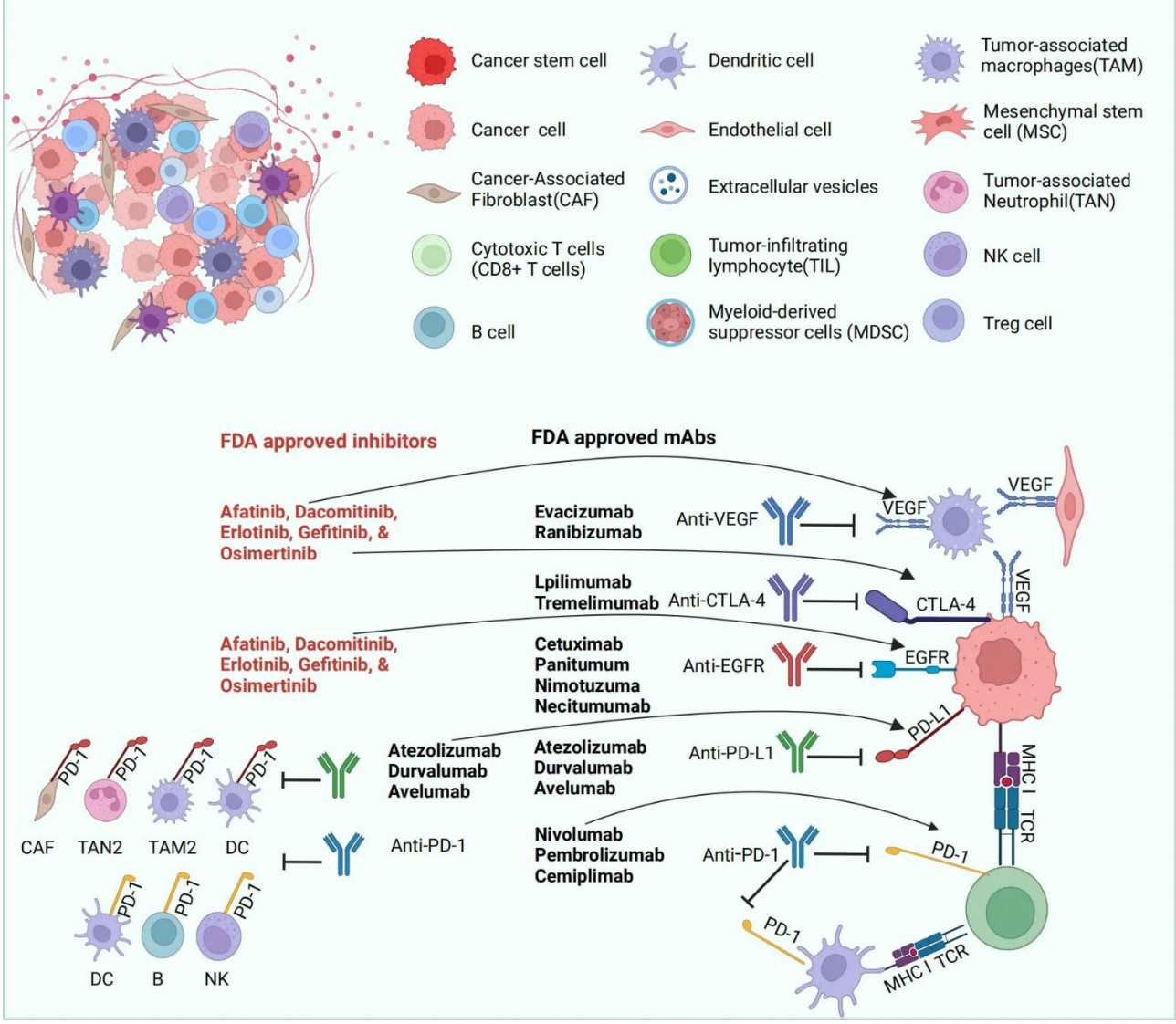

Figure 1 PD-1/PD-L1 interaction-mediated T-cell inhibition (Adopted from Chen et al., 2024a)

Image caption: Many mechanisms, such as genomic aberrations, oncogenic transcription factors and pathways, and post-translational regulation and transport, are involved in the regulation of PD-L1 expression. In addition, anti-PD-1/PD-L1 antibodies can block the activation of PD-1/PD-L1. APCs can absorb tumor antigens and regulate T-cell responses through interactions between the main MHC and TCR. APC can also regulate T-cell activity by regulating the interaction between PD-L1/PD-L2 and PD-1, as well as the interaction between B7 and CD28 (Adopted from Chen et al., 2024a)

Adoptive cell therapies (ACT) represent another major pillar of immunotherapy in advanced ovarian cancer, seeking to provide large numbers of tumor-reactive lymphocytes with enhanced functionality. Strategies include expansion of naturally occurring tumor-infiltrating lymphocytes (TILs), T cells engineered to express high-affinity T-cell receptors (TCRs), and chimeric antigen receptor (CAR)-T cells targeting ovarian cancer antigens such as