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patients with aml carry innate and adaptive immune aberrations at diagnosis that lead to immune suppression, exhaustion, and senescence (909). multiple studies have shown that upregulation of ir, such as the programmed-death 1 (pd-1)/pd-l1 axis, plays a role in immune evasion by leukemic cells (1117). pd-1 is expressed on the surface of activated t cells, b cells, and natural killer cells. when bound by its ligands, pd-1 stimulation leads to suppression of t-cell activation and inhibition of t-cell responses. preclinical models have shown that pd-1-expressing cd8+ t cells and regulatory t cells (treg) accumulate during aml progression, leading to t-cell exhaustion, which can be restored by treg depletion followed by pd-1/pd-l1 blockade (12). further, pd-1 knockout mice have less leukemia burden and improved os (12). upregulation of pd-l1 on leukemic blasts is more frequently observed at relapse than at diagnosis and is associated with poor prognosis (13, 18). similarly, the frequency of t cells coexpressing multiple irs increases with disease progression (11, 17). reversibility of the phenotypic and transcriptional signatures of cd8+ t cells in patients with aml who achieve cr suggests that t-cell exhaustion, an important feature of r/r aml, may be susceptible to therapeutic intervention such as pd-1/pd-l1 axis blockade (11). the success of pd-1/pd-l1 blockade in cancer treatment is inseparable from the foundation laid by preclinical experiments. in preclinical research, the selection of tumor cells and animal models is critical to obtain clinically translational data. therefore, we will briefly describe the tumor cells and animal models for pd-1/pd-l1 interaction studies in melanoma and lung cancer. table 4

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