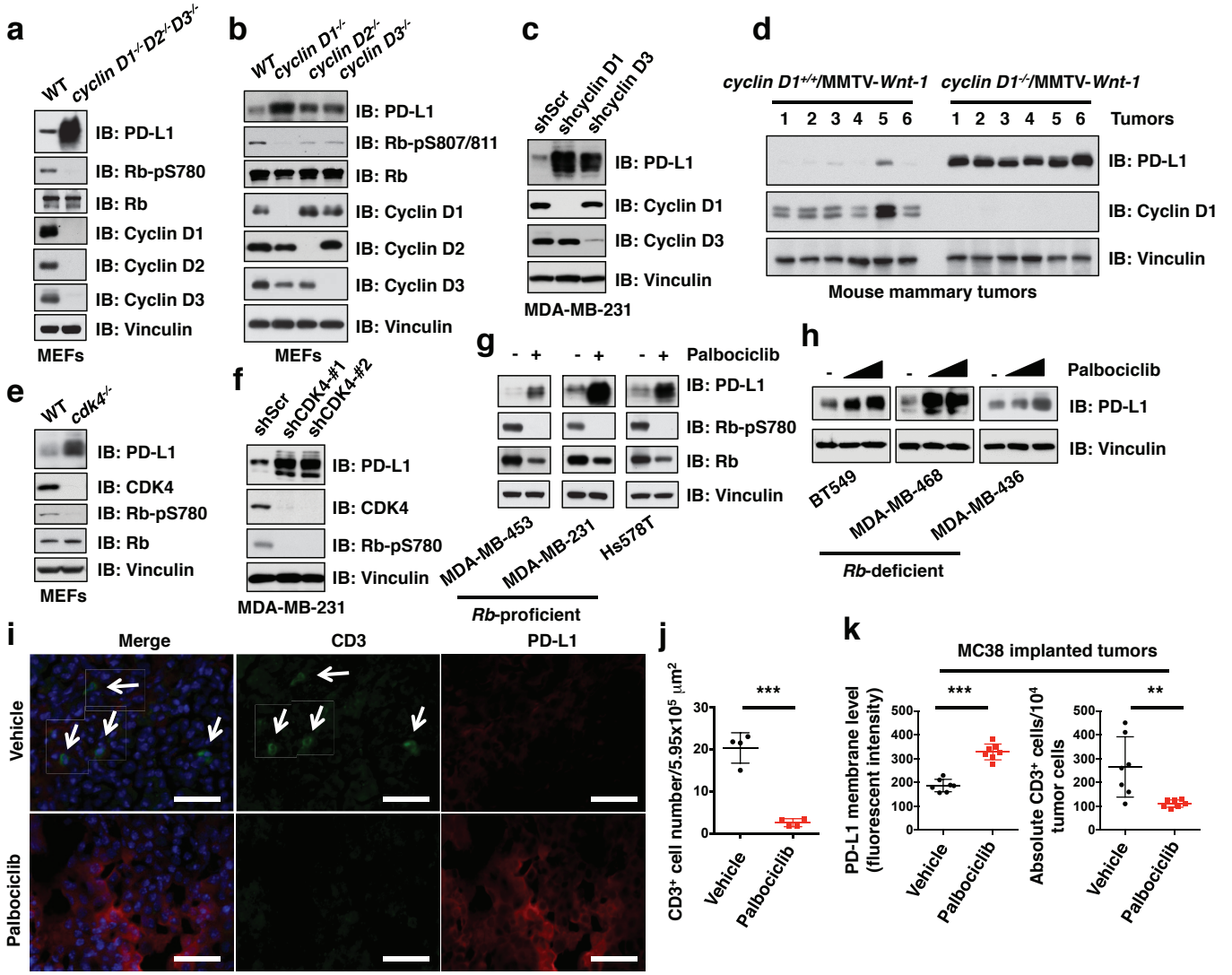

Figure 2 | Cyclin D-CDK4 negatively regulates PD-L1 protein stability. **a-d,** IB analysis of WCL derived from wild type versus combinational (*cyclin D1*$^{-/-}$*D2*$^{-/-}$*D3*$^{-/-}$) (**a**) or single isoform *cyclin D* knockout MEFs (**b**), MDA-MB-231 cells depleted *cyclin D1* or *cyclin D3* using shRNAs (**c**), or MMTV-*Wnt1* induced mouse mammary tumors with/without genetic depletion of *cyclin D1* (**d**). **e-h,** IB analysis of WCL derived from wild type versus *cdk4*$^{-/-}$ MEFs (**e**), MDA-MB-231 cells depleted *CDK4* using shRNAs (**f**), or multiple breast cancer cell lines treated with palbociclib (0.5, 1 μM) for 48 hours (**g**, **h**). **i, j,** Immunofluorescence staining of PD-L1 and CD3 in mouse mammary tumors induced by MMTV-*ErbB2* treated with vehicle or palbociclib as described in Method (**i**) and the quantification of CD3$^+$ T cell population (**j**). The scale bar: 50 μm. **k,** FACS analysis for PD-L1 or CD3$^+$ T-cell populations from MC38 implanted tumors treated with vehicle or palbociclib for 7 days. Vehicle, n = 4 for (**i, j**) or 7 mice for (**k**); palbociclib, n = 4 for (**i, j**) or 7 mice for (**k**). Error bars, ± s.d., two-tailed *t*-test, $^{**}P < 0.01$, $^{***}P < 0.001$ (two-tailed *t*-test).