HOKKAIDO UNIVERSITY

Title	Chemotherapy-Induced IL34 Enhances Immunosuppression by Tumor-Associated Macrophages and Mediates Survival of Chemoresistant Lung Cancer Cells
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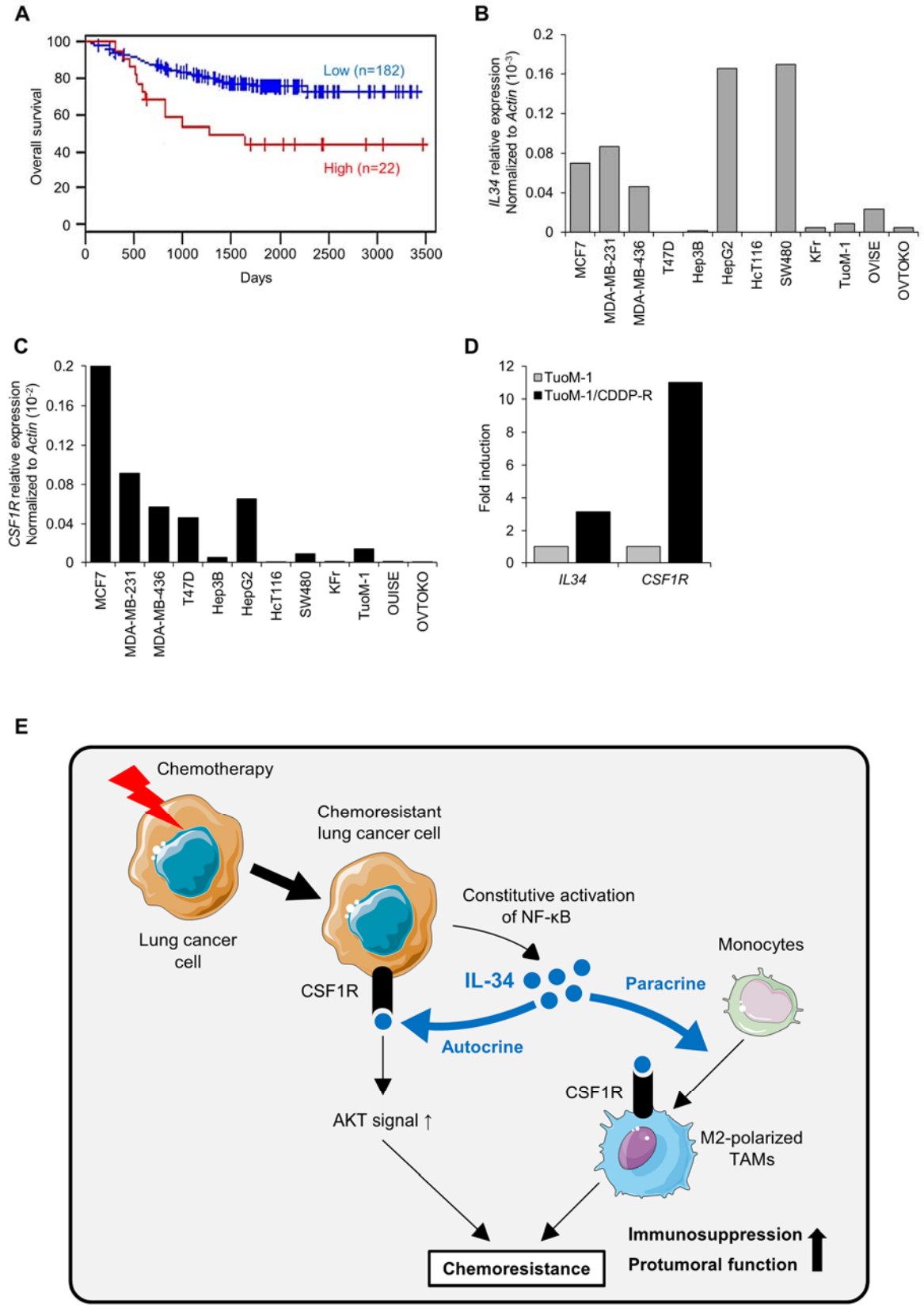
Supplementary figure 6



IL-34 expression in cancers. A, PrognoScan database-based Kaplan–Meier analysis of the overall survival of 204 lung cancer patients stratified by high (red) and low (blue) IL-34 levels (high: n=22, low: n=182; log-rank tests $P<0.05$). B and C, quantitative RT-PCR analysis of *IL34* (B) or *CSF1R* (C) mRNA expression in breast (MCF7, MDA-MB-231, MDA-MB-436, T47D), liver (Hep3B, HepG2), colon (HCT116, SW480) or ovary (KFr, TuoM-1, OVISE, OVTOKO) cancer cell lines. D, quantitative RT-PCR analysis of *IL34* and *CSF1R* mRNA expression in normal or cisplatin-resistant TuoM-1 ovarian mucinous cystadenocarcinoma cells. E, A scheme of mechanism describes the dual role of IL-34 in chemoresistance. Chemoresistant cancer cells produce IL-34, which mediates the recruitment of protumoral M2-polarized TAMs with enhanced immunosuppressive and protumoral properties (paracrine effect). Additionally, IL-34 enhances the activation of sustained AKT-mediated survival signal downstream of CSF1R, and thus helps to maintain chemoresistance in cancer cells (autocrine effect).