Figure S1. Knockout of METTL5 inhibits the progression of oral squamous cell carcinoma *in vitro*. (A) Validation of METTL5 stable knockout in HSC3 cells using western blotting. Results of the (B) Cell Counting Kit-8, (C) colony formation, (D) wound healing and (E) Transwell assays after knockout of METTL5 in HSC3 cells. *P<0.05; **P<0.01; ***P<0.001; ****P<0.0001. METTL5, methyltransferase 5, N6-adenosine; CCND3, cyclin D3; sg, small guide RNA; NC, negative control; OD, optical density.

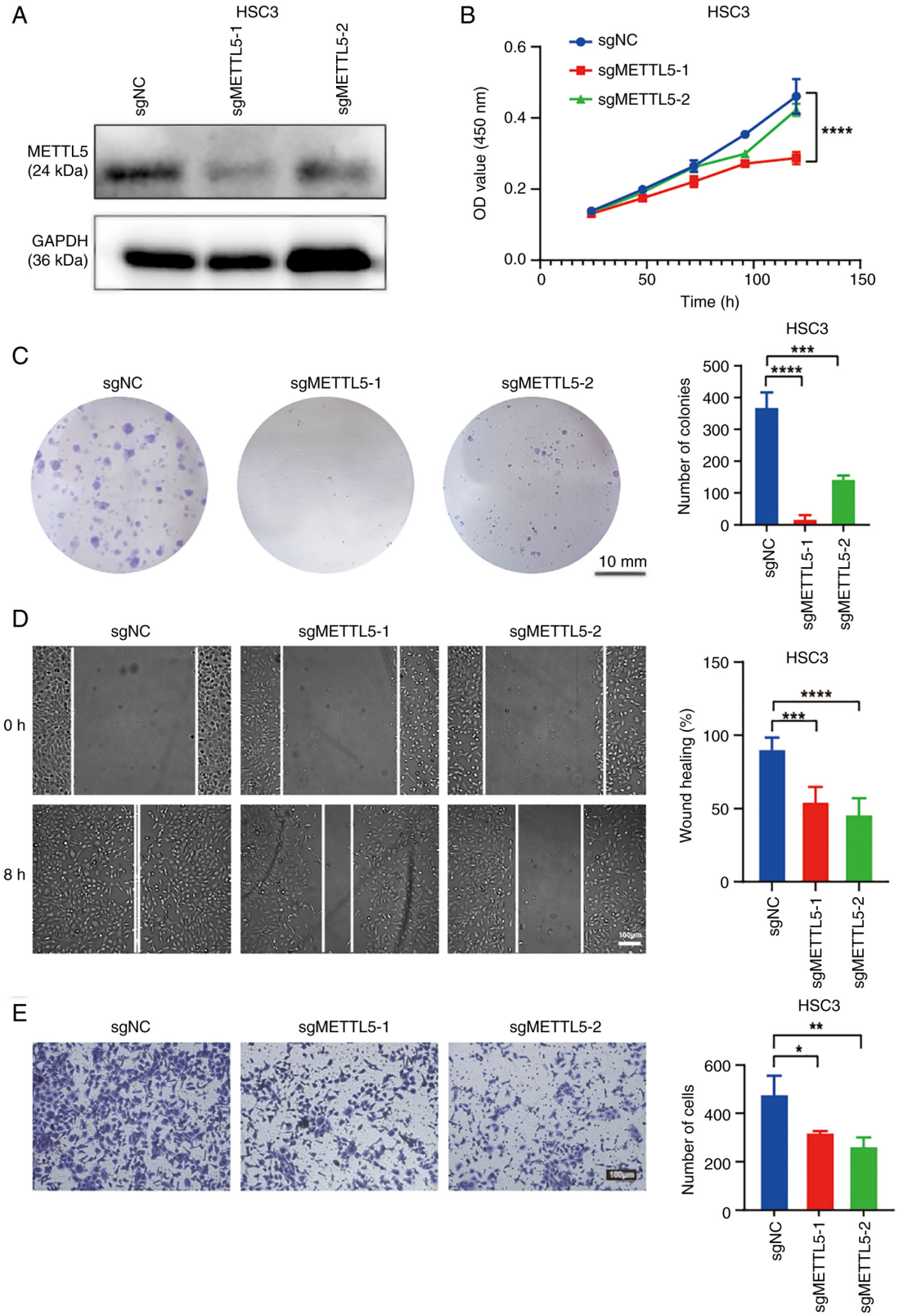

Figure S2. Overexpression of METTL5 promotes the progression of oral squamous cell carcinoma. (A) Validation of METTL5 overexpression in HSC3 cells using western blotting. (B) Results of the Cell Counting Kit-8 assay after METTL5 overexpression in HSC3 cells. (C) Results of the Transwell assay after METTL5 overexpression in HSC3 and SCC15 cells. *P<0.05; ***P<0.001. METTL5, methyltransferase 5, N6-adenosine; CCND3, cyclin D3; sg, small guide RNA; NC, negative control; oe, overexpression; OD, optical density.

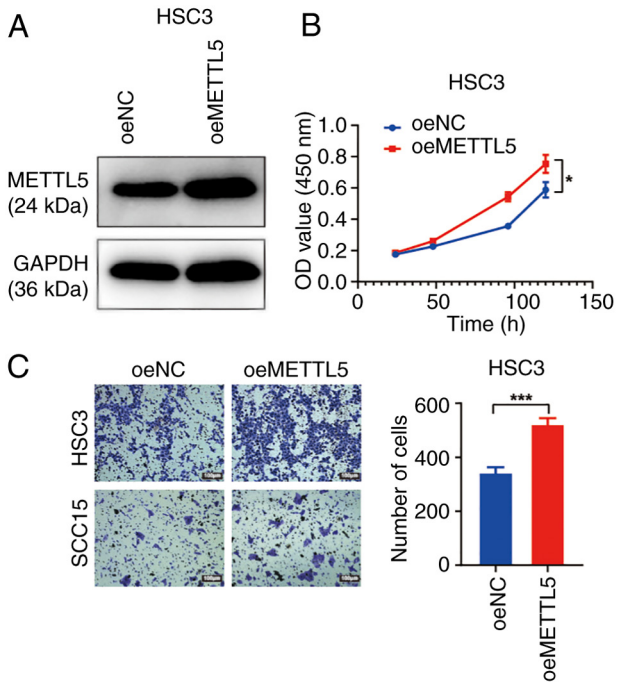

Figure S3. Results of the cell cycle assay after CCND3 overexpression in METTL5-depleted HSC3 cells. sg, small guide RNA; NC, negative control; METTL5, methyltransferase 5, N6-adenosine; CCND3, cyclin D3.

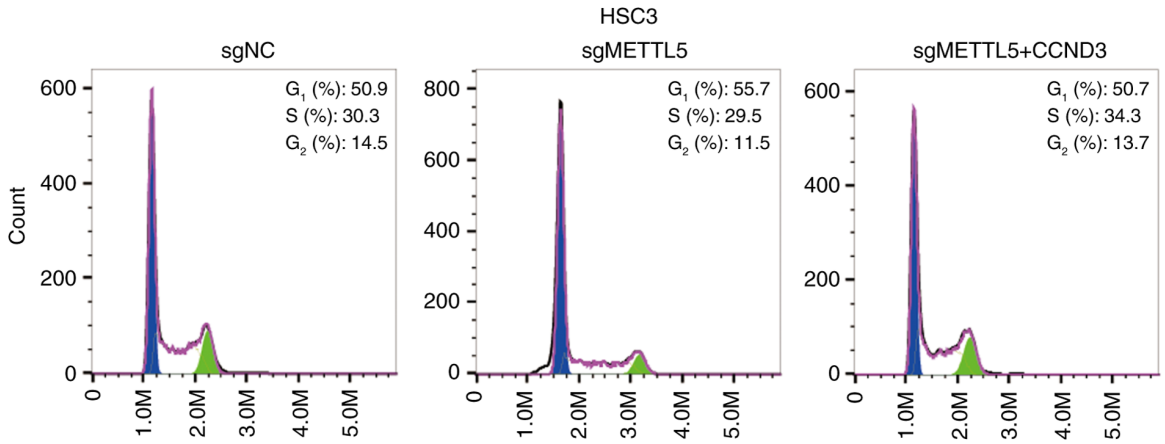
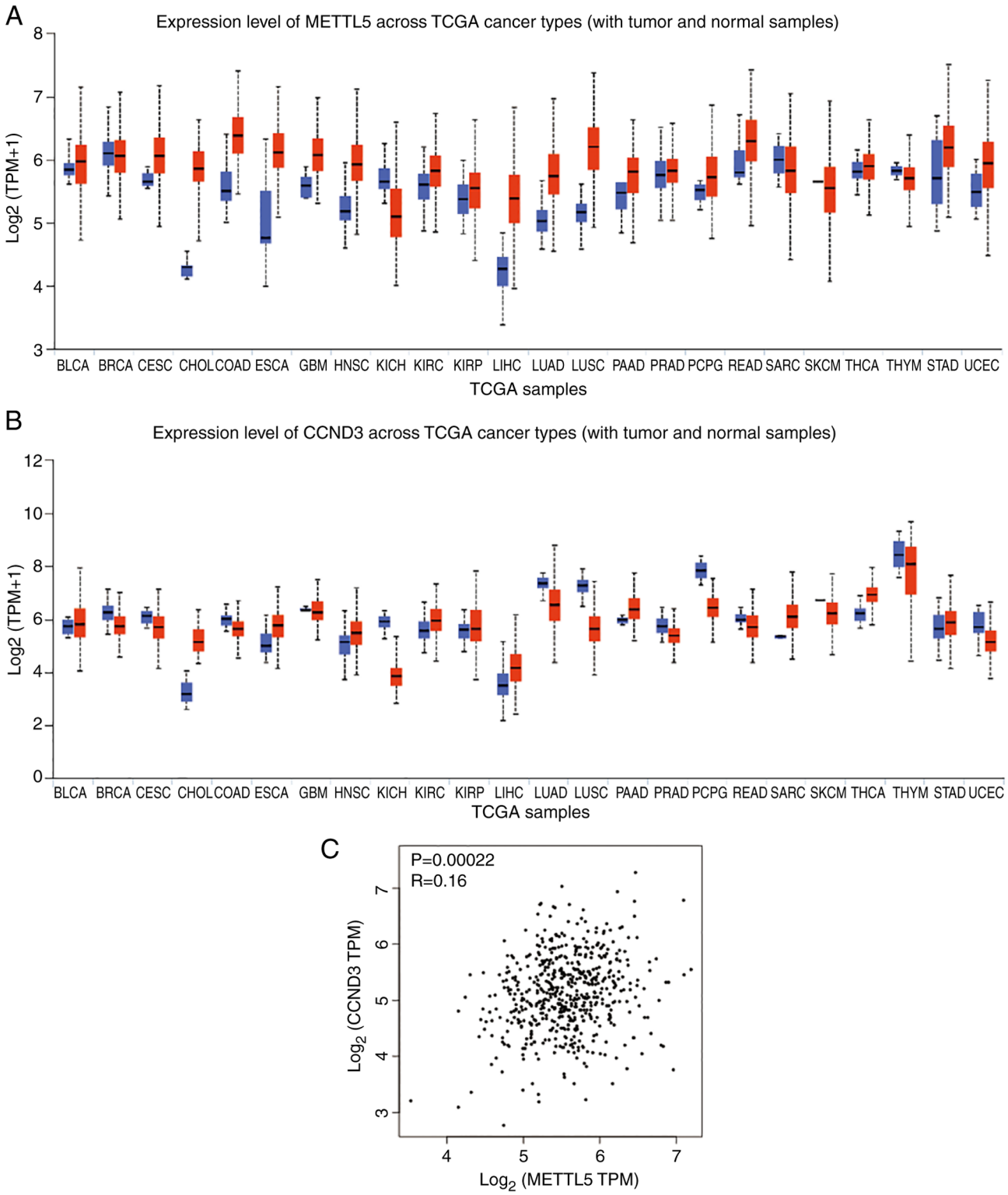
Figure S4. Pan-cancer expression patterns and the correlation between METTL5 and CCND3. (A) Box plots displaying the expression levels of METTL5 across several TCGA cancer types, with tumor samples (red boxes) and matched normal samples (blue boxes) compared. (B) Box plots depicting the expression levels of CCND3 across the same TCGA cancer types, with tumor samples (red boxes) and normal samples (blue boxes) presented. (C) Scatter plot illustrating the correlation between METTL5 and CCND3 expression levels across all TCGA samples. METTL5, methyltransferase 5, N6-adenosine; CCND3, cyclin D3; sg, small guide RNA; NC, negative control; TPM, transcripts per million; TCGA, The Cancer Genome Atlas.