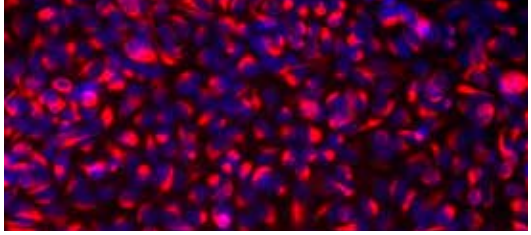
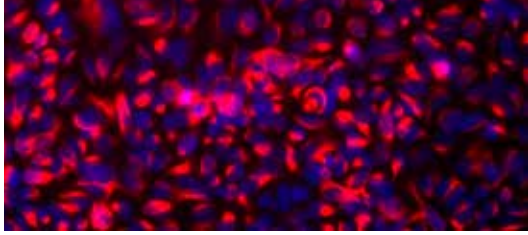
THE ESSENTIALS OF
LIFE SCIENCE RESEARCH
GLOBALLY DELIVERED™





ATCC®

EPITHELIAL-MESENCHYMAL TRANSITION REPORTER CELL LINE

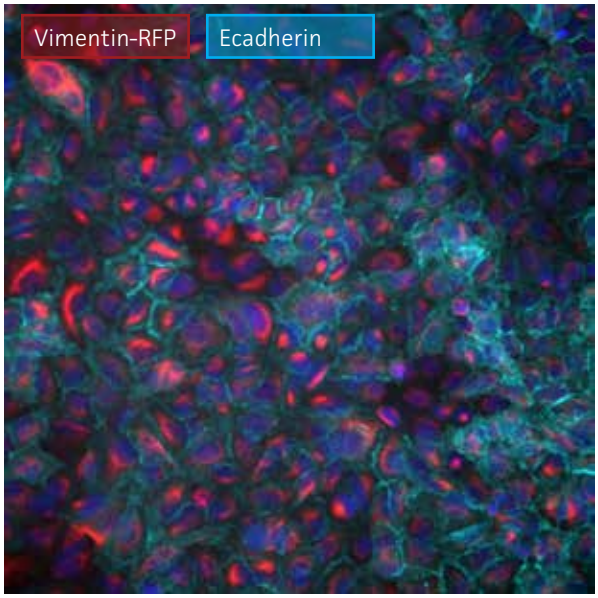
Epithelial-mesenchymal transition (EMT) and its reverse, mesenchymal-epithelial transition (MET) are developmental processes which have been shown to play critical roles in promoting metastasis and invasion in carcinoma. Recent studies have shown that EMT of cancer cells not only causes tumor metastasis but also contributes to drug resistance. To help researchers investigating this phenomenon, ATCC has employed CRISPR/Cas9 gene editing to develop A549 Vim RFP (ATCC® CCL-185EMT™).

This reporter line is designed to enable the real-time monitoring of the changing status of cells from epithelial to mesenchymal via the expression of red fluorescent protein (RFP)-tagged vimentin. This cell line is not only an aid in dissecting the EMT/MET pathway in the research field, but also is a robust platform for new cancer drug development.

- CRISPR/Cas9 gene-edited vimentin-RFP fusion protein
- Strong RFP signal due to upregulated vimentin upon EMT induction
- Physiological E-cadherin expression in the absence of EMT
- Similar growth kinetics as the parental A549
- TGF-β1-responsive
- Increased invasive capacity following EMT
- EMT sensitive to A83-01 and PP1 inhibition

ATCC No.	Designation	Volume	Cells/vial
CCL-185EMT™	A549 Vim RFP	1 mL	1 x 10^6

VALIDATION DATA



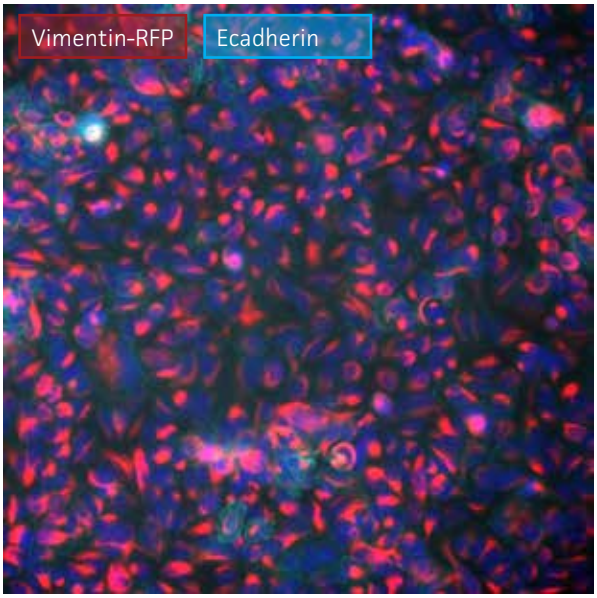


Figure 1. A549-VIM-RFP shows increased mesenchymal and decreased epithelial marker protein expression after EMT. Treatment of A549 Vim RFP with the EMT induction agent TGF-β1 results in increased vimentin-RFP expression (red) and decreased E-cadherin expression (cyan). The cells in both panels were counterstained with NucBlue fixed cell ReadyProbes reagent (blue).

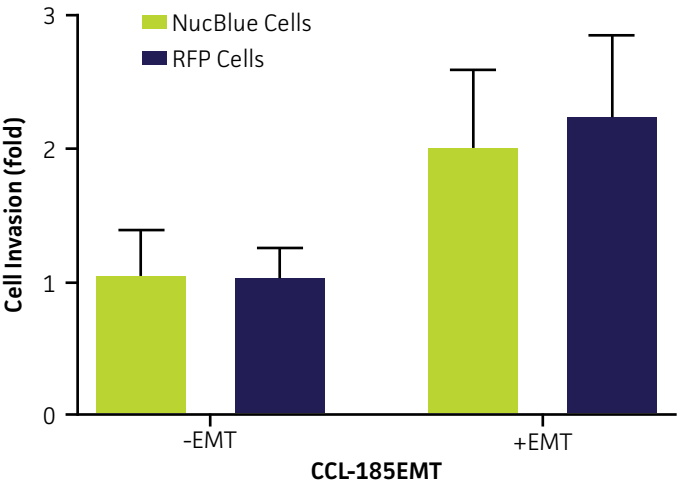
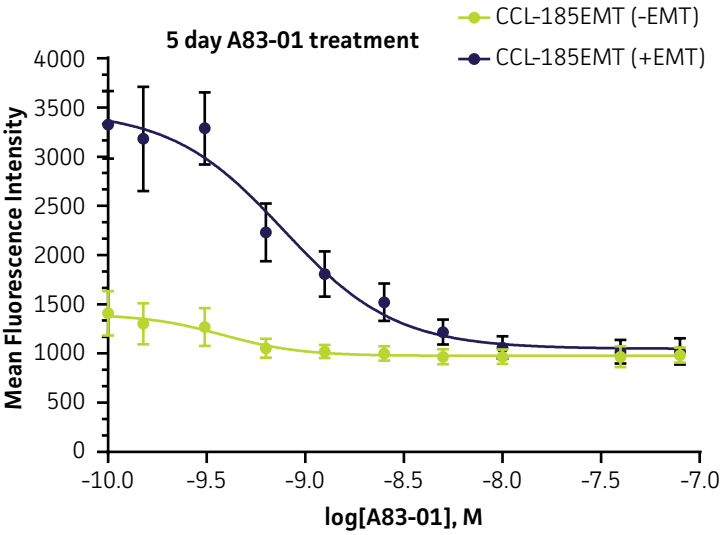
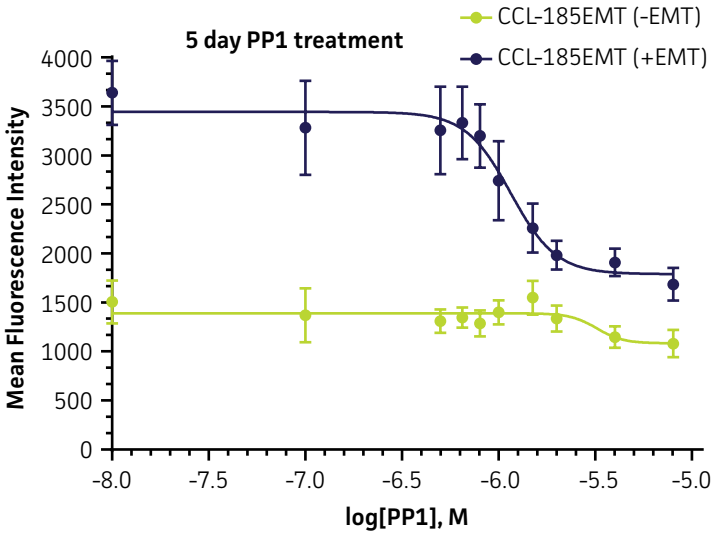


Figure 2. After a 5-day incubation with (+EMT) or without (-EMT) TGF-β1, A549-Vim-RFP cells were monitored over a 24 hr period for invasion through an 8 µm pore filter of the basement membrane of the BD 24 well fluoroblock cell invasion system. EMT induced A549 Vim RFP cells show increased invasive capacity. The similar number of RFP positive and NucBlue nuclear counterstained cells depict the utility of RFP expression to monitor invaded cells.



	CCL-185EMT (-EMT)	CCL-185EMT (+EMT)
IC50	3.822e-010	7.733e-010



	CCL-185EMT (-EMT)	CCL-185EMT (+EMT)
IC50	3.22e-006	1.154e-006

Figure 3. Small molecule EMT inhibitors block transition in A459 Vim RFP cells. Two pathways associated with EMT were targeted: TGF-β and SRC using A8301 and PP1, respectively. In both cases, TGF-β1-induced EMT was inhibited by the compound.

Visit www.atcc.org/EMT for more information.

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