ATCC® Epithelial-mesenchymal Transition Reporter Cell Line

A549 Vim RFP (ATCC® CCL-185EMT™) is a reporter line 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.

VALIDATION DATA

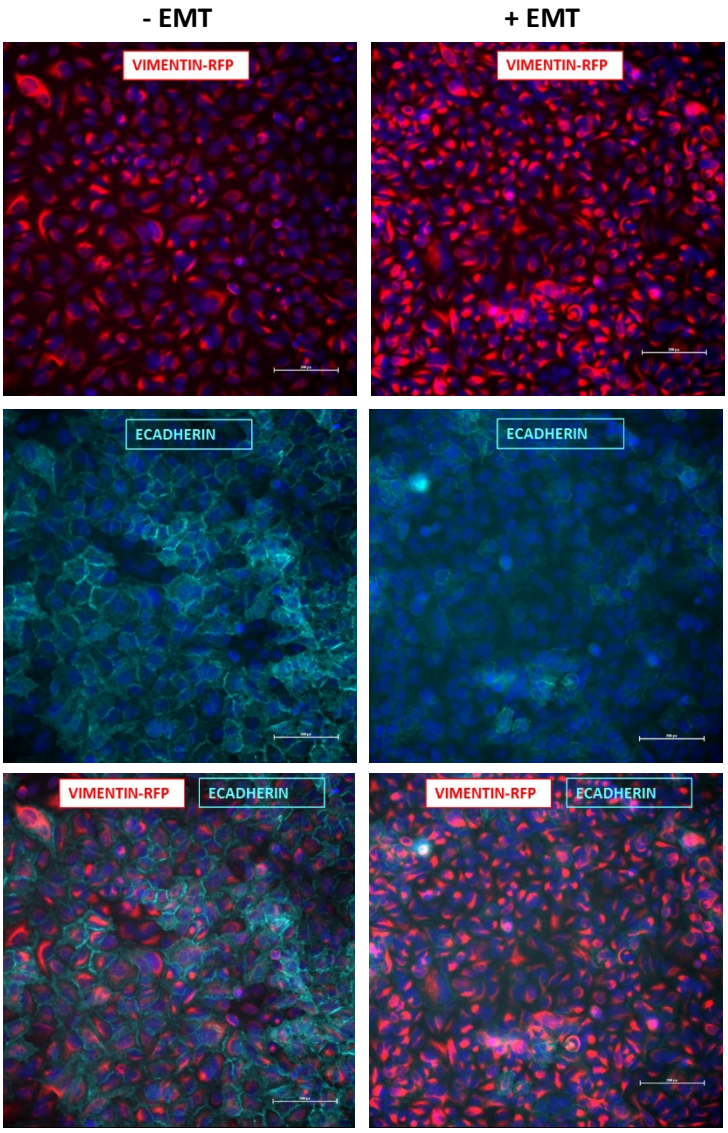


Figure 1. A549-VIM-RFP shows increased mesenchymal marker protein expression in addition to decreased epithelial marker protein expression after EMT. A549-VIM-RFP cells were incubated in complete growth media supplemented with either 2.5 ng/mL TGF-β1 (right column) or an equivalent volume of 1X Dulbecco's phosphate buffered saline (as a no EMT control; left column) for 5 days. Treatment of A549 Vim RFP with TGF-β1 induced EMT and resulted in increased vimentin-RFP expression (red; top left and right). Additionally, a decrease in E-cadherin expression (cyan; middle left and right) was observed. The nuclei of cells were counterstained with NucBlue Fixed Cell ReadyProbes Reagent (blue). The bottom panels are an overlay of the top and middle left and the top and middle right panels.

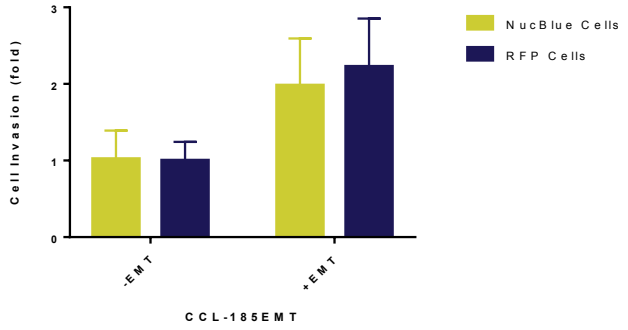
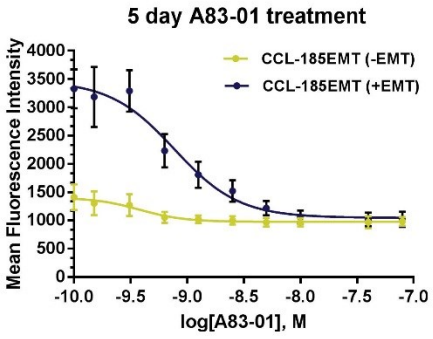
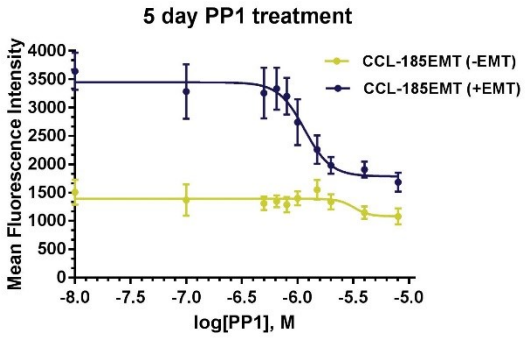


Figure 2. TGF-β1 EMT-induced CCL-185EMT cells have increased invasion capacities. After a 5-day incubation with (+EMT) or without (-EMT) TGF-β1, A549-Vim-RFP cells were monitored over a 24 hr period for invasion through an 8 µm pore filter of the basement membrane of the BD 24 well fluoroblock cell invasion 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: TGFβ and SRC using A8301 and PP1, respectively. In both cases, TGFβ1-induced EMT was inhibited by the compound.