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Technical Data Sheet:
PANC-1 ECAD-EmGFP MET Reporter Cell Line

ATCC® Number	CRL-1469MET™
Organism	*Homo sapiens,* human
Tissue/Disease Source	Pancreas, pancreatic carcinoma
Product Description	The PANC-1 E-cadherin (ECAD)-EmGFP reporter cell line was created by knocking-in an EmGFP reporter gene into the endogenous E-cadherin gene using CRISPR/Cas9 technology. We confirmed that EmGFP activities accurately report E-cadherin gene expression, and showed that ECAD-EmGFP expression can be induced by microRNA-200 treatment, indicating cells transition to epithelial status.
Application	This cell line is not only a useful in vitro model for dissecting the molecular switches underlying EMT and MET, but could also be used for screening compounds targeting EMT or MET in pancreatic cancer.

E-cadherin gene expression in PANC-1 ECAD-EmGFP cells

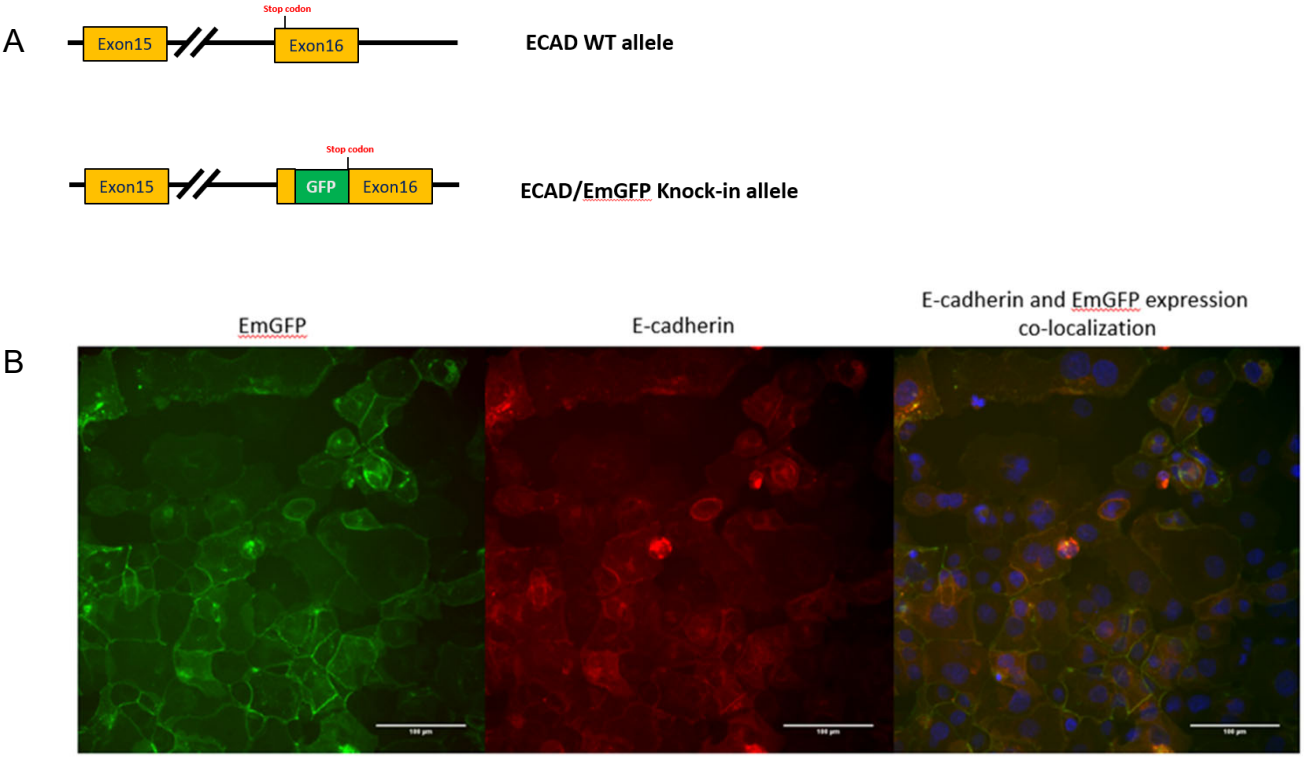

Figure 1. EmGFP activities accurately report E-cadherin gene expression in PANC-1 ECAD-EmGFP cells (ATCC® CRL-1469MET™). (A) Partial schematic diagram of the E-cadherin wild type allele and ECAD-EmGFP knock-in allele, in which EmGFP is incorporated adjacent to endogenous E-cadherin in Exon 16. (B) Endogenous EmGFP (left, green) co-localized with E-cadherin detected by immunofluorescence assay (middle, red) as shown in the merged image (right).

Cell Morphology

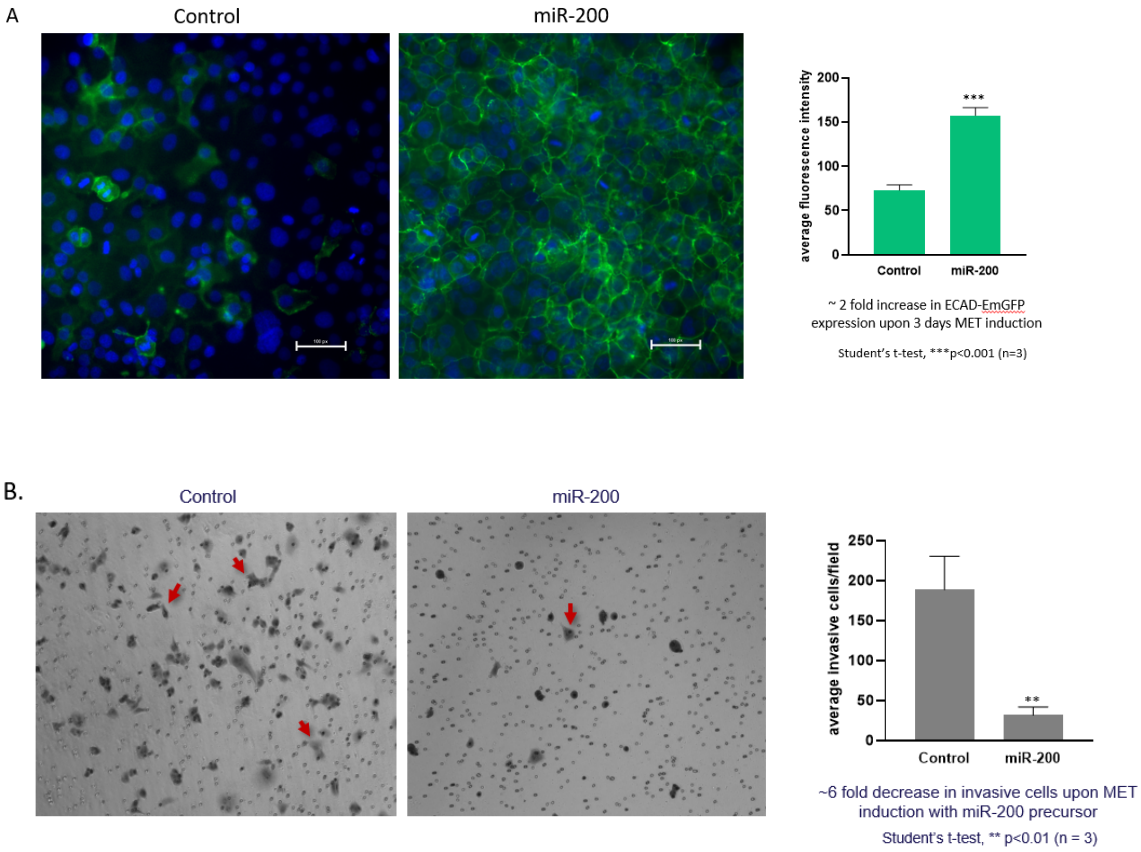

Figure 2. MicroRNA-200 treatment results in the induction of ECAD-EmGFP expression, decrease in SNAIL expression, and decrease in the invasive capabilities of PANC-1 ECAD-EmGFP cells. (A) PANC-1 ECAD-EmGFP cells were treated with miRNA-200 or an equivalent volume of 1x PBS (as a control) for 3 days. miRNA-200 treatment induced a significant increase in ECAD-EmGFP expression. The nuclei of the cells were counterstained with DAPI. (B) After 3 days miR-200 treatment, PANC-1 ECAD-EmGFP cells were monitored over a 48 hours period for invasion through a trans-well invasion assay. The induced cells showed a significant decrease in invasive capacity.