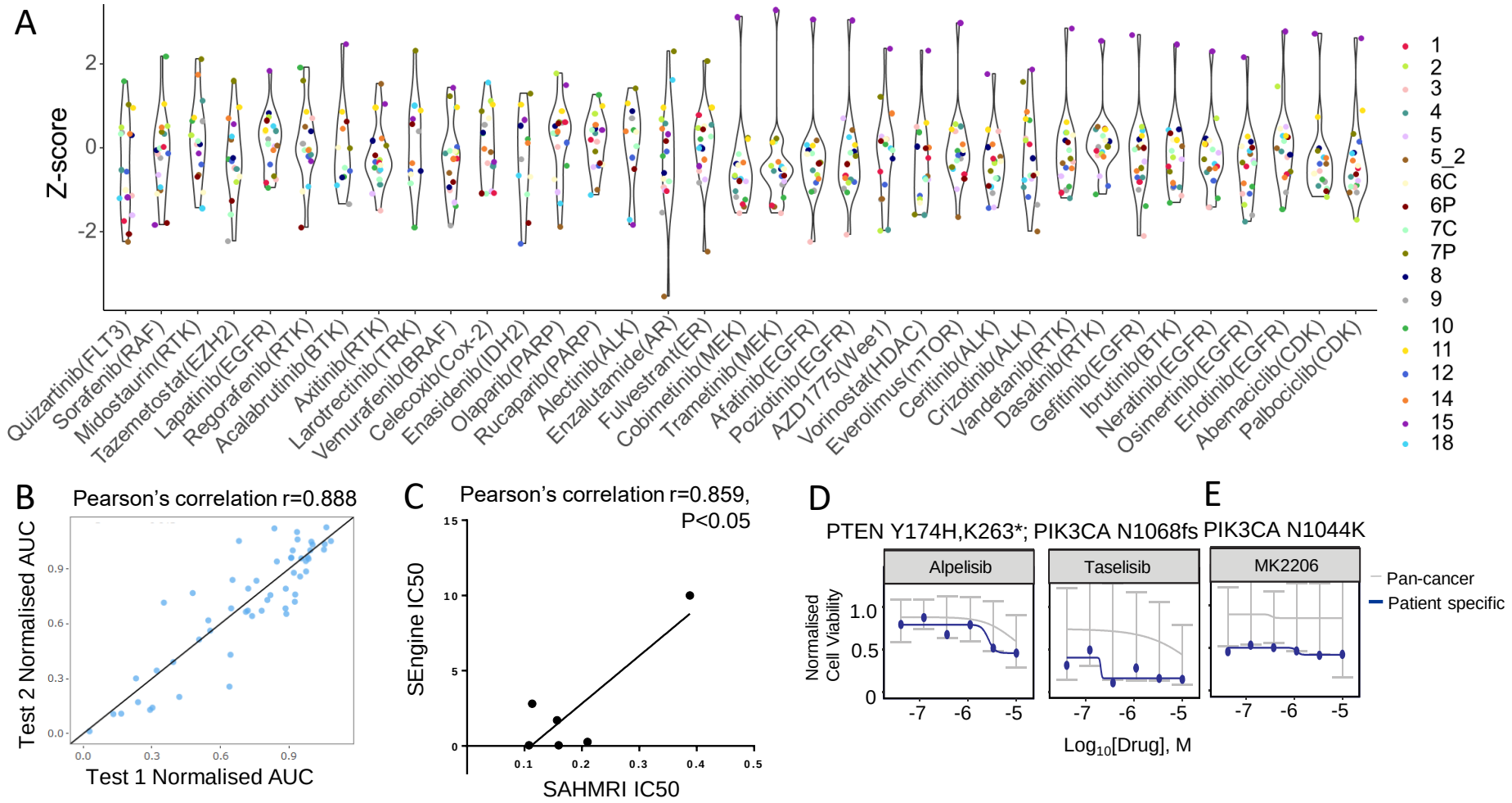

Supplementary Fig 3 (A) Highlighting patient-specific relative responses to each drug. Z-score transformed AUC drug response data from Figure 2 *ex vivo* medium-throughput drug testing of organoids. Legend indicates color for each patient sample. Matched samples: C, tumoroid derived from primary colorectal cancer; P, peritonoid derived from CRPM. **(B) Accuracy of medium-throughput drug testing.** Correlation of normalised AUC data obtained from two independent drug screens using peritonoids from patient 5 two weeks part at SEngine Precision Medicine. **(C)** Effect of Wee1 inhibitor, Adavosertib, on six peritonoid lines significantly correlated between different test sites and platforms (SEngine Precision Medicine, USA & SAHMRI, Australia). SEngine: Peritonoids from 6 patients were split to single cells and plated immediately into a 6-dose dilution series of Wee1 inhibitor, Adavosertib, for 6 days in 384 well format. SAHMRI: The same peritonoid lines were grown for two days in 96 well format and then treated with an 8 dose dilution series of Adavosertib (in quadruplicate) for 5 days. Cell viability was assessed at both sites using CellTitre Glo (Promega) and IC50 (µM) calculated from dose response curves. Pearson's correlation coefficient was calculated to compare data generated at the same site on different days or at the different sites. **(D-E) Genomically predicted peritonoid drug sensitivities continued from Fig 3.** Dose response curves for: **(D)** Patient 5 PTEN Y174H,K263*; PIK3CA N1068fs peritonoids treated with p110a inhibitors, Alpelisib and Taselisib; **(E)** Patient 1 PIK3CA N1044K peritonoids treated with AKT inhibitor, MK2206. Blue line on dose response curves is patient specific response, grey line indicates average response for cancer organoid and cell lines previously screened at SEngine. Error bars indicate standard deviation.