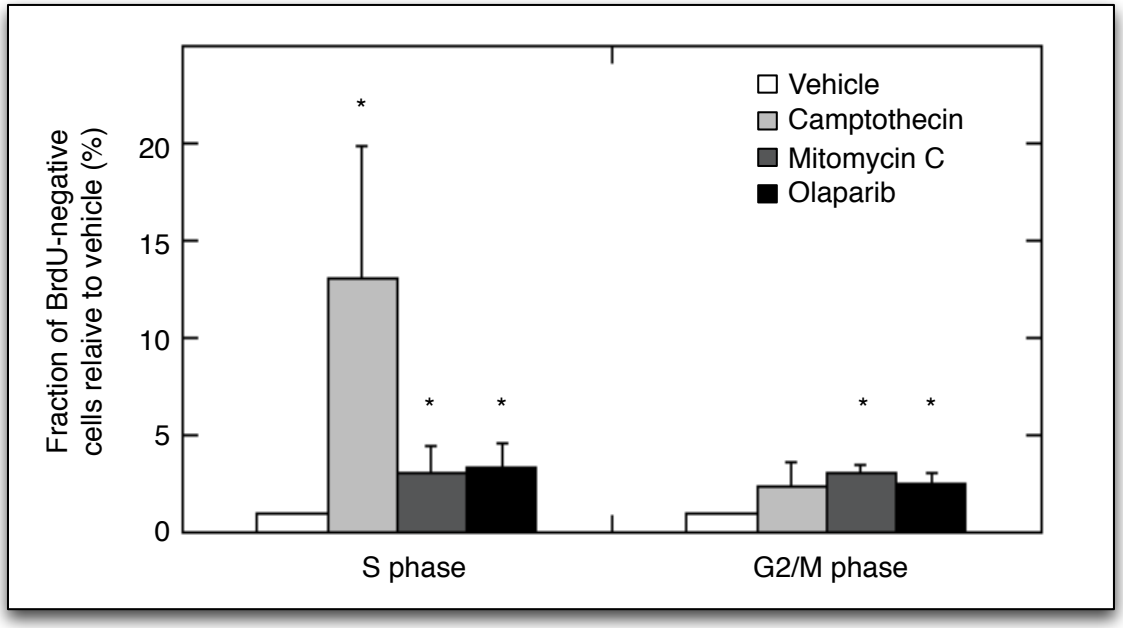

Figure 6 – figure supplement 2: MMR-deficient tumor cultures were challenged with olaparib (26μM), camptothecin (30nM) or mitomycin C (300nM) for 24 hours, pulsed with BrdU for 2 hours and analyzed for cell cycle by propidium iodide staining (DNA content analysis) using flow cytometry. The bar plot shows the fraction of unlabeled (arrested) cells in S and G2/M, normalized to the G1 fraction; bars indicate SEM; data represent the results from 7 cultures. All experiments were repeated twice. DNA damage provoked by exposure to camptothecin consistently increased stalled (BrdU-negative) cells in S phase (average 13-fold increase; P=5.23E-5). Mitomycin C caused an increase of stalled cells in S phase (3.08-fold; P=5.8E-3) and in G2/M phase (3.12-fold; P=2.2E-7). Olaparib induced, as expected, an increase in stalled cells in S and G2/M (respectively, a 3.35 and a 2.54-fold increase; P=2.1E-3 and 5.2E-4). Overall, this indicates that MMR-deficient cultures did not exhibit any loss of G2/M cell cycle checkpoints or DNA damage signaling.