Influence of alectinib and crizotinib on ionizing radiation - in vitro analysis of ALK/ROS1-wildtype lung tissue cells

Jost T. *et al*. 2022

Supplementary Material

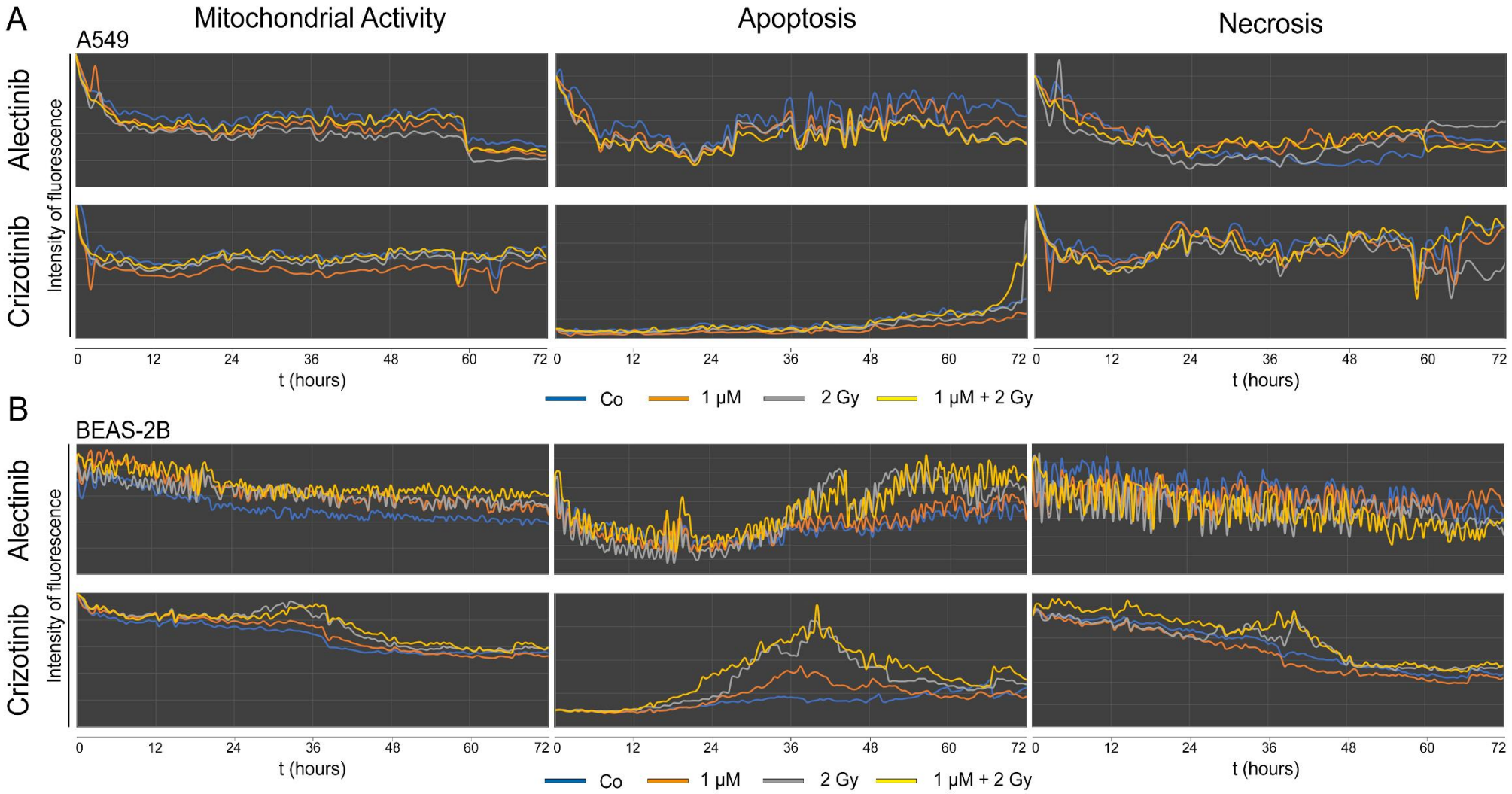

Figure S1: Live cell imaging of cell line A549 (**a**) and BEAS-2B (**b**) under treatment (Co, 1 µM KI, 2 Gy IR, combination KI + IR) from timepoint t = 0 h up to t = 48 h. Cells were grown and either treated with 1 µM kinase (orange line) inhibitor (alectinib or crizotinib), 2 Gy (grey line) or a combination of both (yellow line) in cell culture chips of ibidi (Gräfelfing, Germany), additionally to a untreated control (blue line). Tetramethylrhodamine (TMRM, Invitrogen, Carlsbad, CA, USA) was used to measure mitochondrial activity based on mitochondrial membrane potential, CellEvent Caspase 3/7 (Invitrogen, Carlsbad, CA, USA) for staining of apoptosis and DRAQ7 (Abcam, Cambridge, UK) for necrosis. Curves of intensity of fluorescence are plotted over time.

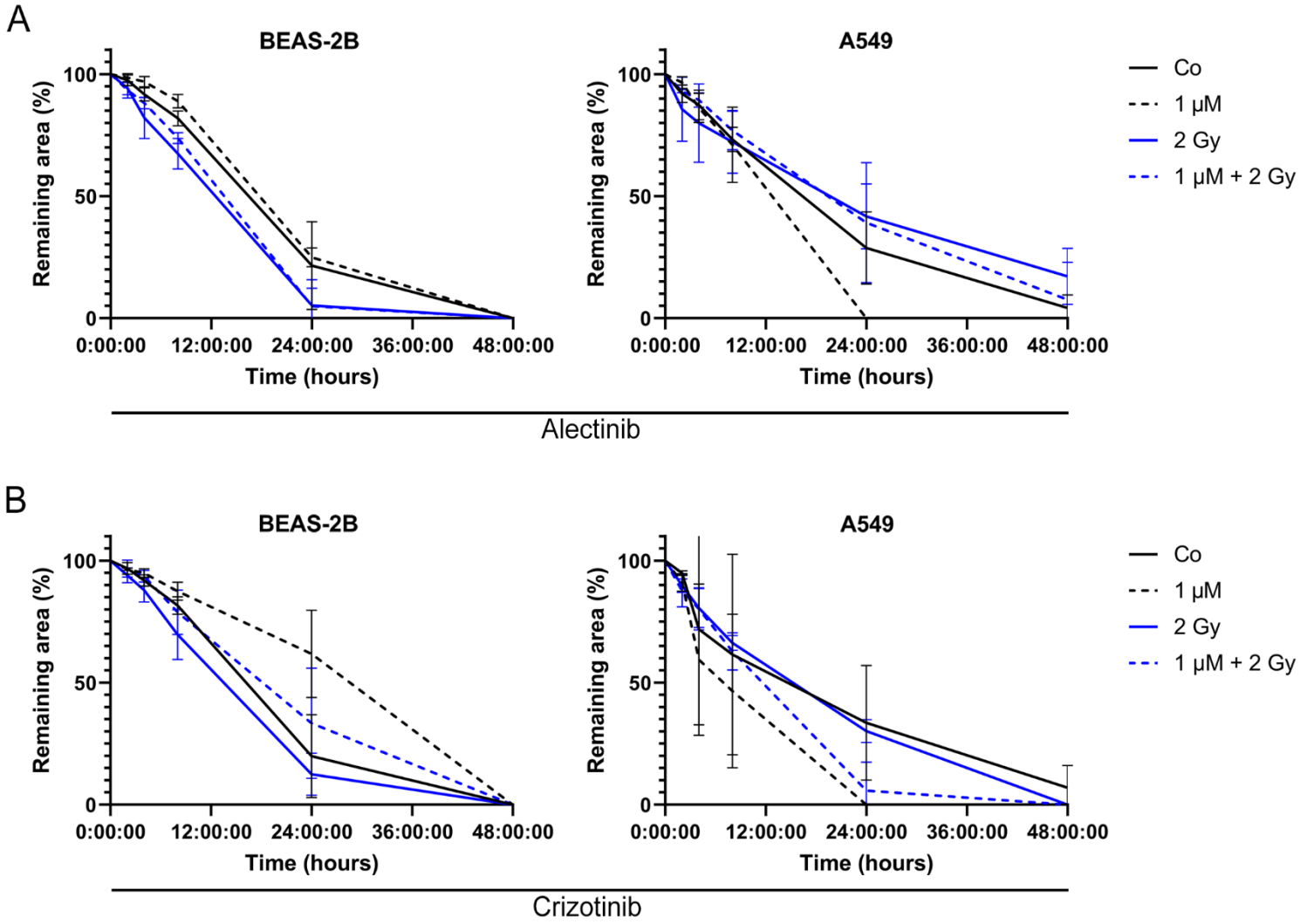

Figure S2: Influence of ALK/ROS1 inhibitors alectinib and crizotinib on migration behavior of lung cell lines BEAS-2B and A549. Both ALK/ROS1 wildtype lung cells were treated with 1 µM alectinib or (**a**) crizotinib (**b**) w/o 2 Gy IR for 48 h and decrease of scratch area was measured over time. Each value represents mean ± SD (n = 4).

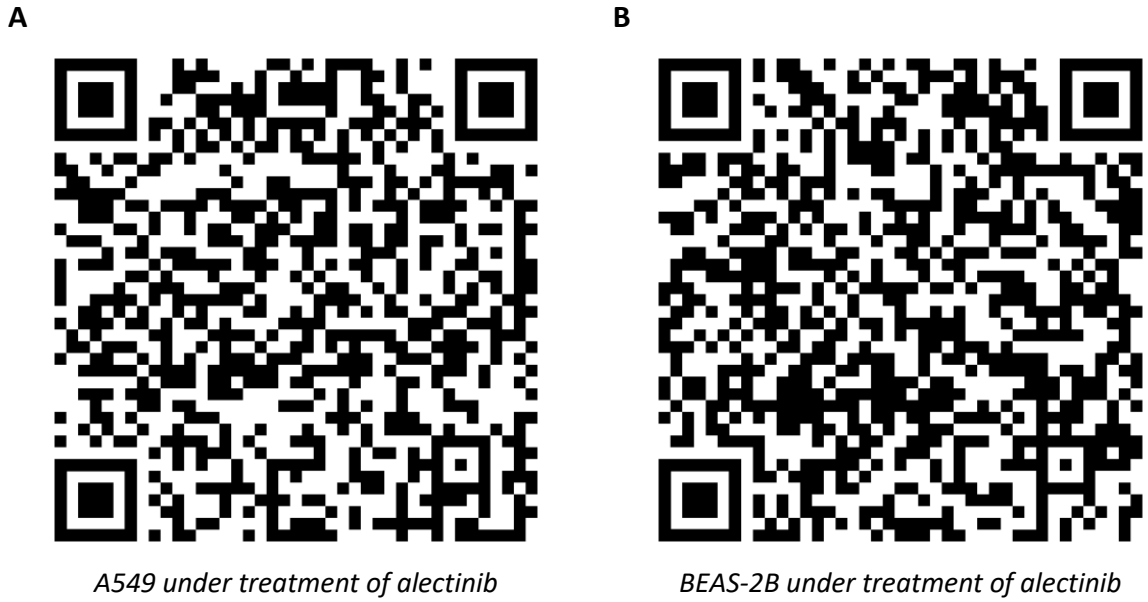

Figure S3: Representative example of live cell imaging of cell line A549 (**a**) and BEAS-2B (**b**) under treatment over a time-period of 72 h. Cells were grown and treated with 1 μM alectinib in cell culture chips of ibidi (Gräfelfing, Germany). Tetramethylrhodamine (TMRM, Invitrogen, Carlsbad, CA, USA) was used to measure mitochondrial activity based on mitochondrial membrane potential (green), CellEvent Caspase 3/7 (Invitrogen, Carlsbad, CA, USA) for staining of apoptosis (blue) and DRAQ7 (Abcam, Cambridge, UK) for necrosis (red).