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Endothelial p130cas confers resistance to anti-angiogenesis therapy

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In the original version of the article that was published with the January 25, 2022 issue, there were inadvertent errors introduced during figure assembly in Figures 2, 4, and 5. The original Figure 2H contained an inversion of the first two images in the upper panel, the original Figure 4D contained duplicated Hoechst and VEGFR2 images in row 1 and row 2 as well as a duplicated cell cluster in row 3, and the original Figure 5A contained duplicated animal images in day 35 (first group, positions 1 and 3) and day 50 (second group, positions 2 and 3). The correct images for each case have been located, and the correct versions of the figures now appear online with the article; these errors did not affect the scientific conclusions drawn from the figures. The authors sincerely regret the errors and any confusion they may have caused.

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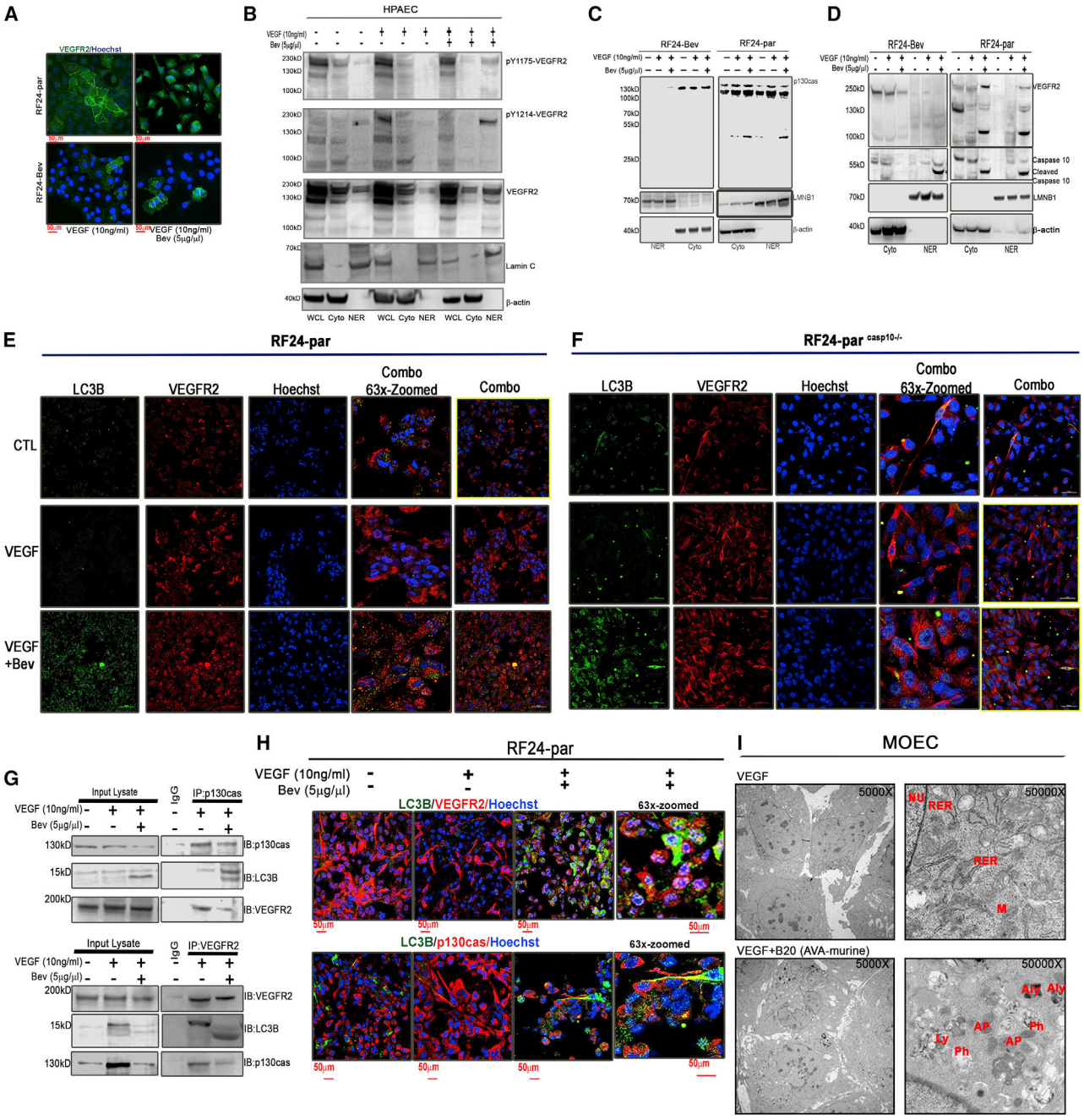

Figure 2. p130cas and VEGFR2 are internalized into autophagosomes and the nucleus, followed by caspase-10 cleavage, in ECs treated with Bev (corrected)

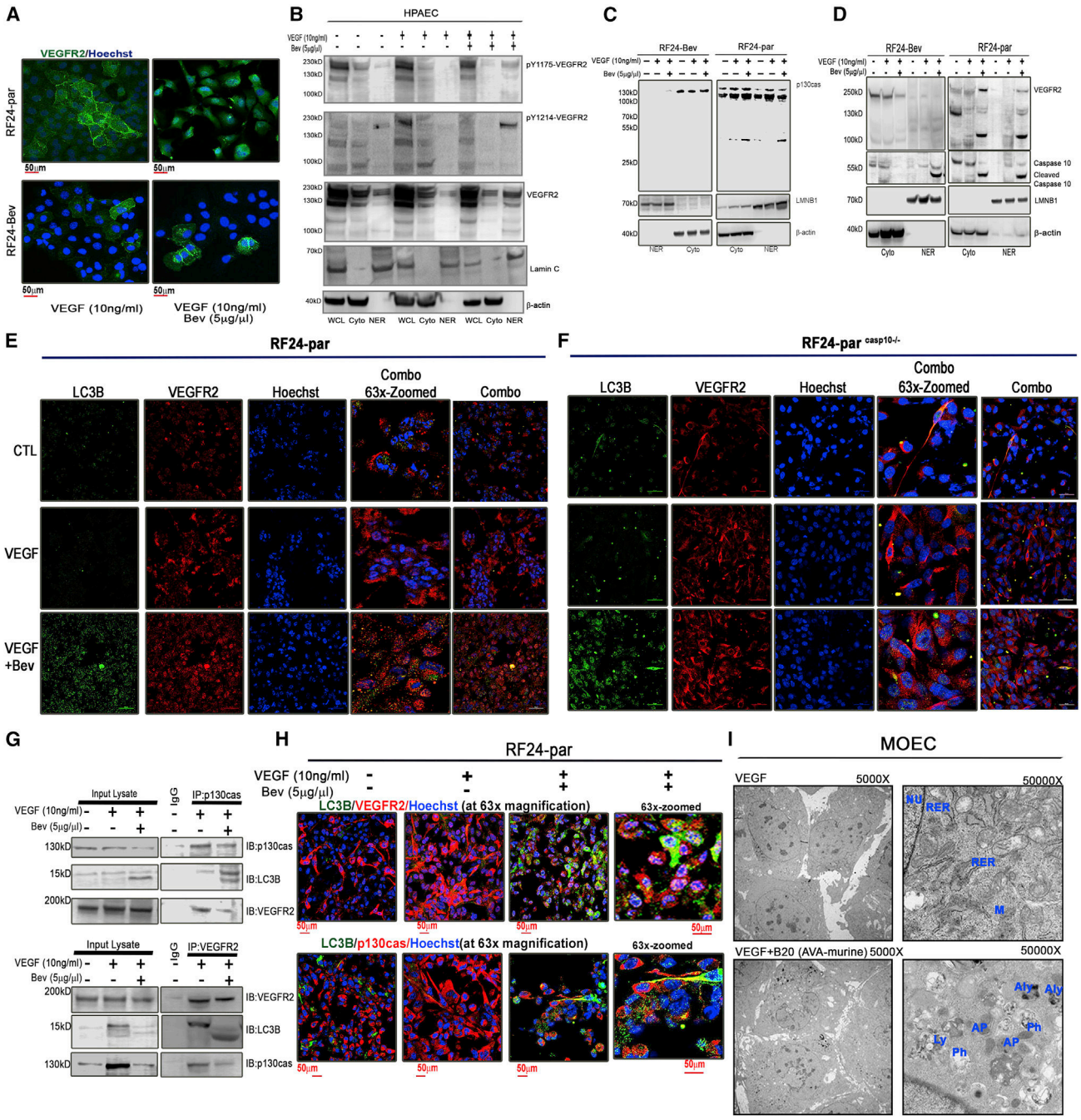

Figure 2. p130cas and VEGFR2 are internalized into autophagosomes and the nucleus, followed by caspase-10 cleavage, in ECs treated with Bev (original)

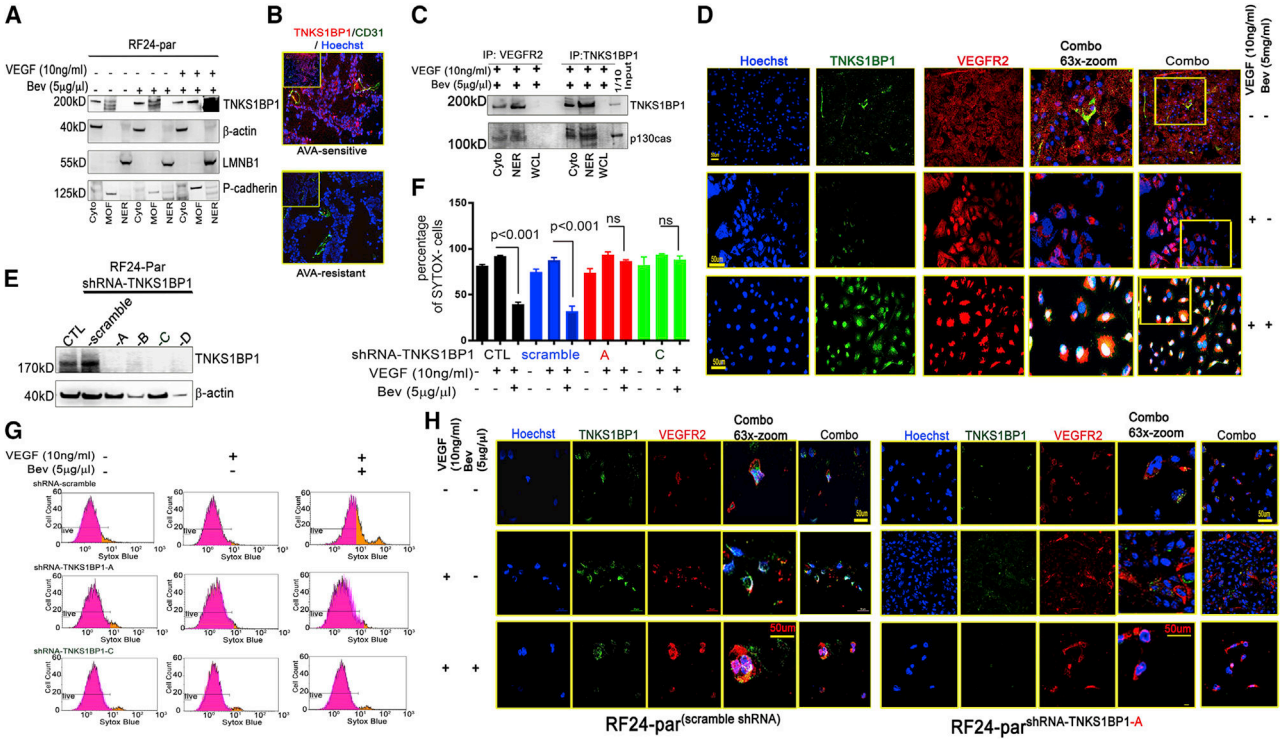

Figure 4. TNKS1BP1 and nuclear VEGFR2 mediate AVA therapy-induced EC death (corrected)

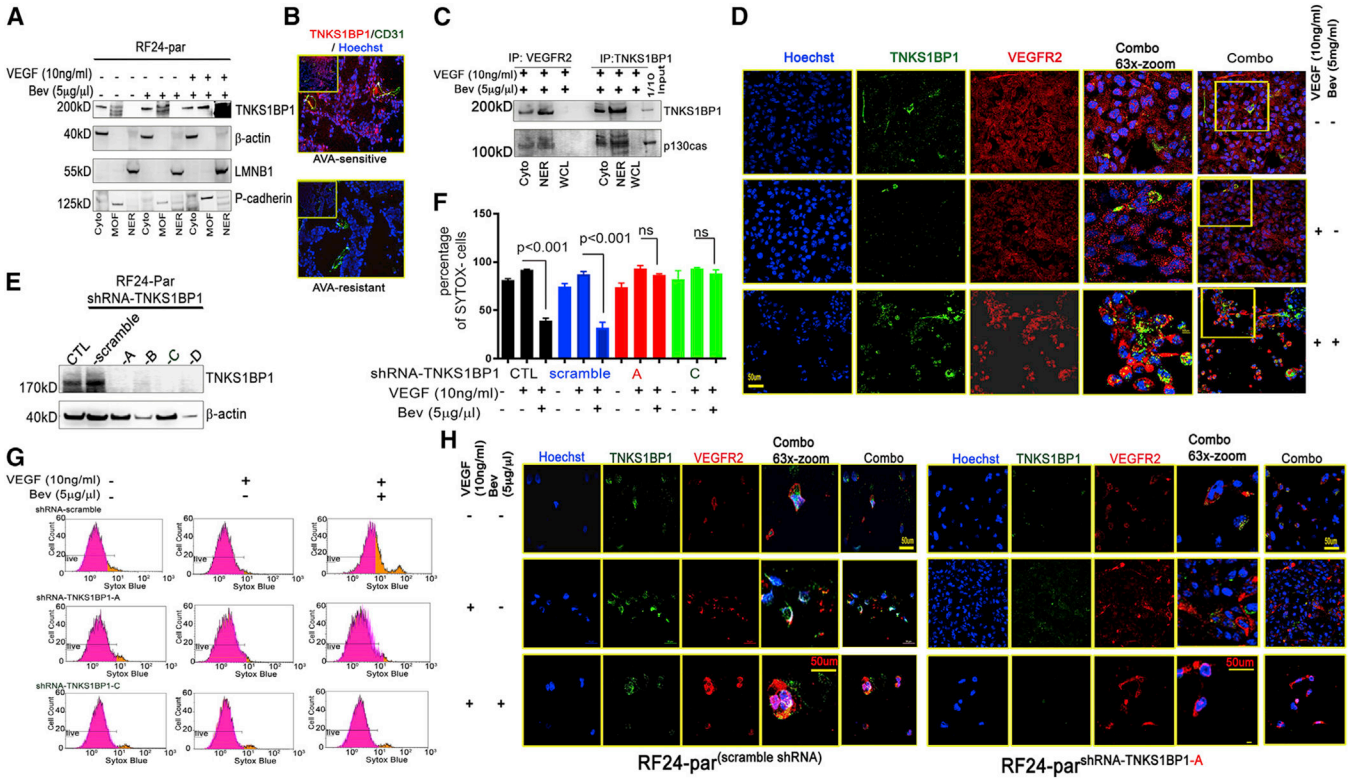

Figure 4. TNKS1BP1 and nuclear VEGFR2 mediate AVA therapy-induced EC death (original)

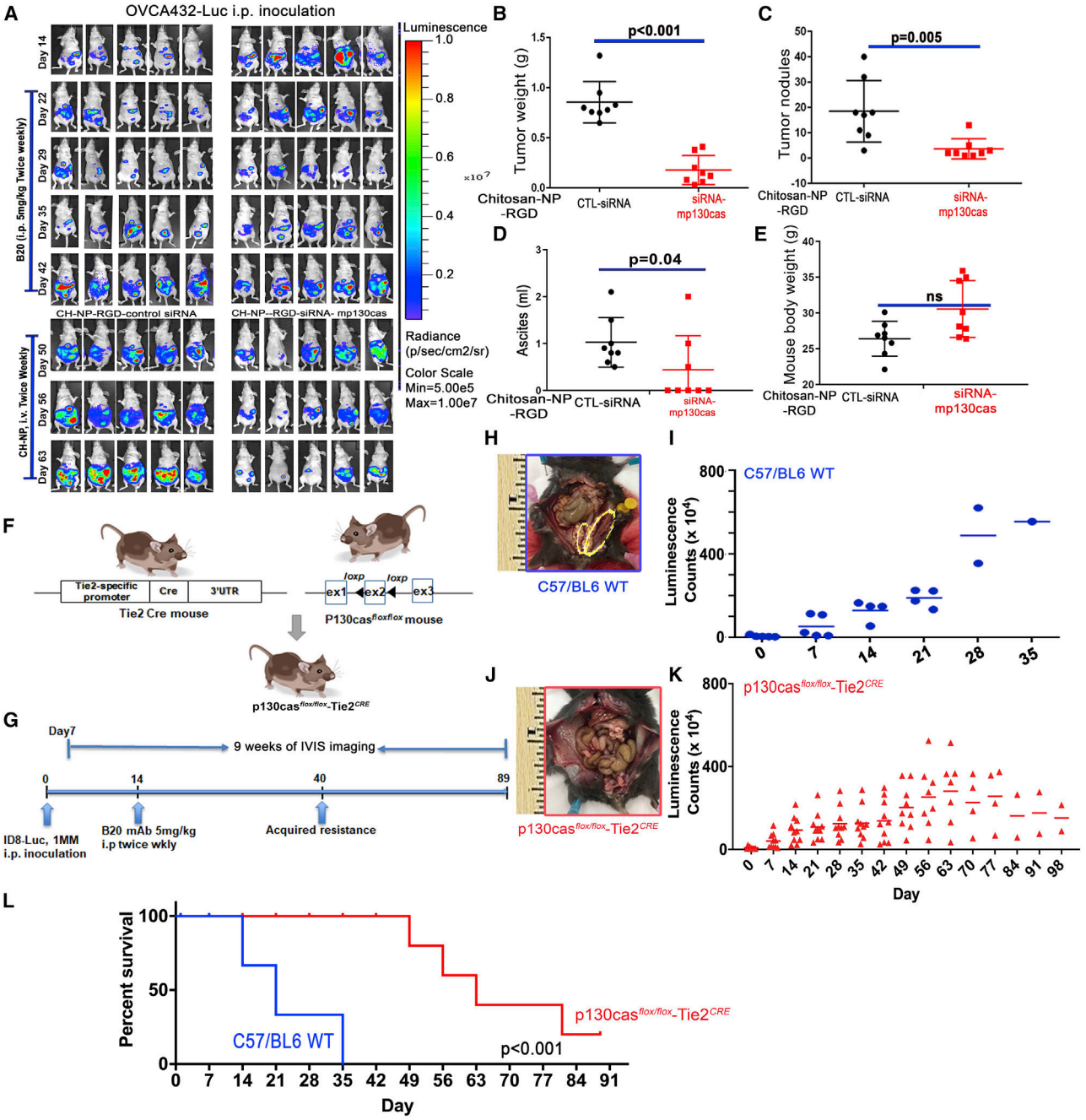

Figure 5. Ablation of vascular p130cas delays progression of tumors with adaptive resistance to AVA therapy (corrected)

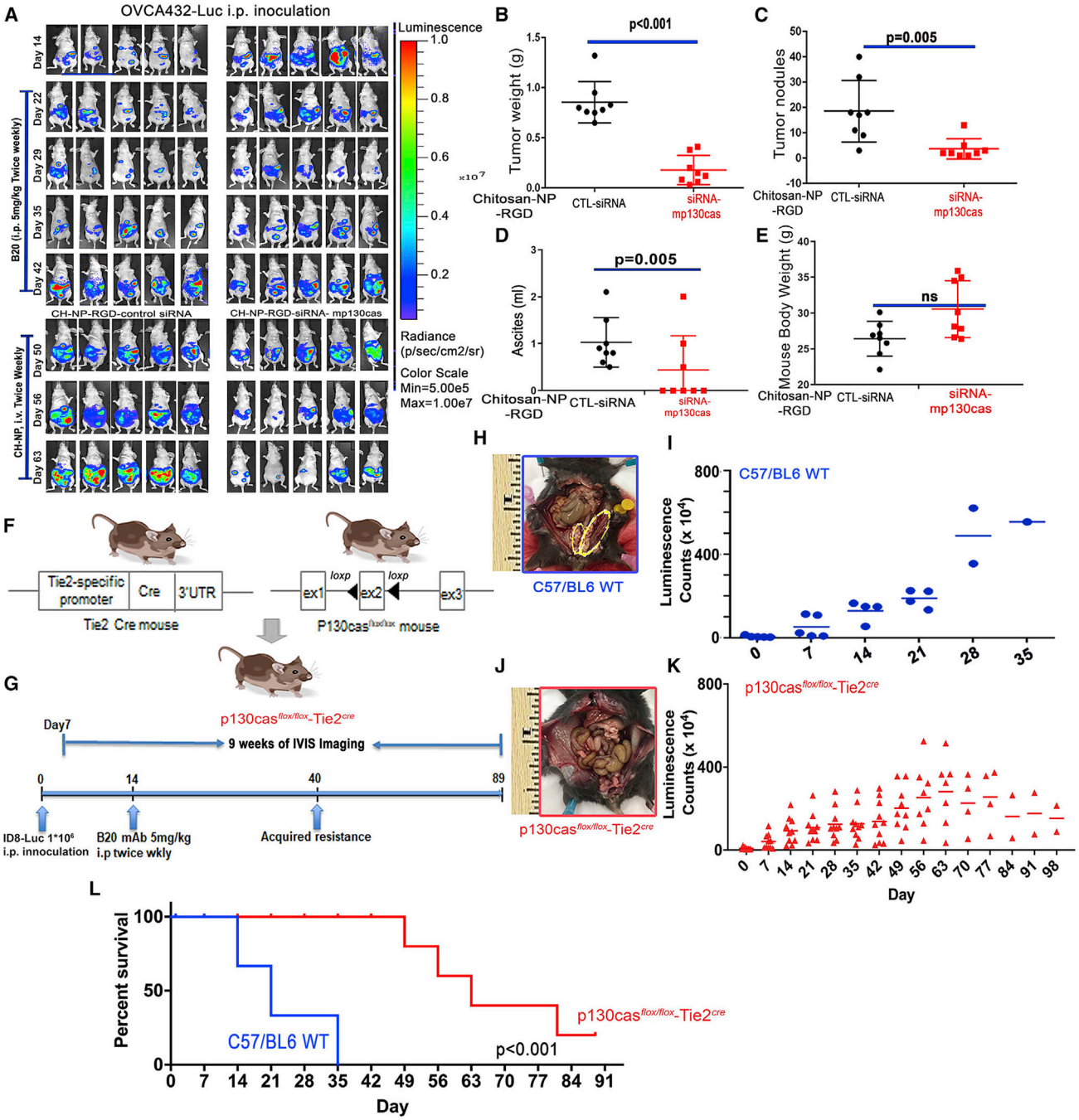

Figure 5. Ablation of vascular p130cas delays progression of tumors with adaptive resistance to AVA therapy (original)