Supplemental Material: Annu. Rev. Pharmacol. Toxicol. 2021. 61. https://doi.org/10.1146/annurev-pharmtox-050120-105018 Senolytic Drugs: Reducing Senescent Cell Viability to Extend Health Span Robbins et al.

**Supplemental Figure 1** Schematic of the *INK-ATTAC* (A) and *p16-3MR* (B) mice. (A) The *INK-ATTAC* mice express a caspase 8-FKBP fusion protein from a *p16<sup>Ink4a</sup>* promoter fragment. This drives apoptosis of *p16<sup>Ink4a</sup>* expressing cells in vivo after administration of the drug AP20187, which promotes dimerization of FKBP and thereby caspase 8. (B) *p16-3MR* mice express a fusion of three transgenic reporters, the renilla luciferase (LUC), monomeric red fluorescent protein (mRFP), and herpes simplex virus 1 thymidine kinase (HSV-TK) under the control of a *p16<sup>Ink4a</sup>* promoter in a BAC construct, allowing for specific killing of *p16<sup>Ink4a</sup>* expressing cells when the drug ganciclovir (GCV) is administered to mice. GCV is by converted by HSV-TK. to GCV-triphosphate, which is a toxic chain terminator of DNA replication. (Figure courtesy of Dr. Carolina Soto Palma.)



Apoptosis of senescent cells