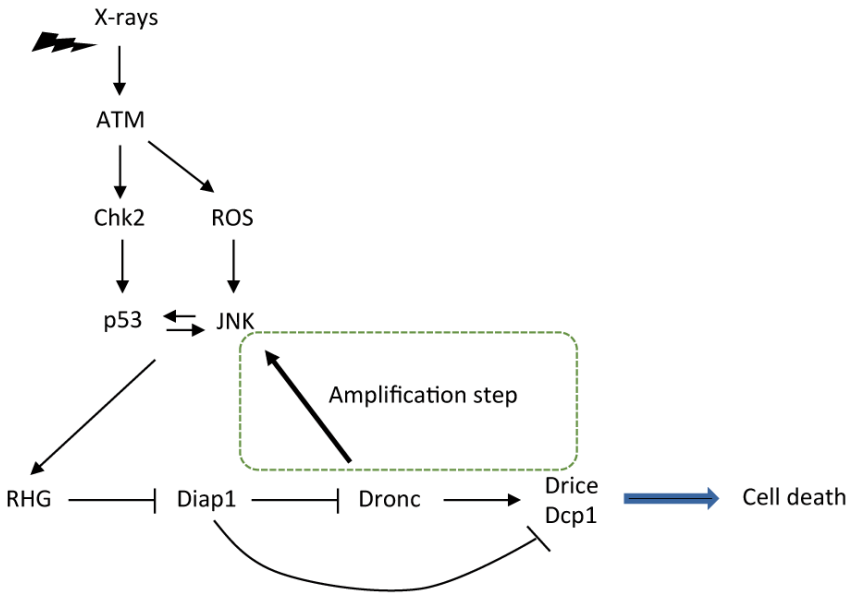

Figure S1. Simplified version of the apoptosis program of *Drosophila*, as triggered by X-rays.

The DNA double-strand breaks caused by the irradiation activates the Ataxia Telengiestasia Mutated (ATM) kinase, which in turn activates the Checkpoint2 (Chk2) kinase and also induces the production of Reactive Oxygen Species (ROS). These events trigger the function of *p53* and of the Jun N-Terminal Kinase (JNK) pathway, known to stimulate each other. The expression of *p53*/JNK transcriptionally activates the pro-apoptotic genes *reaper (rpr), head involution defective (hid)* and *grim*, which cause ubiquitination of the Drosophila inhibitor of apoptosis protein1 (Diap1) and allow activation of the apical caspase Dronc and subsequently of the effector caspases Drice and Dcp1. In addition of activating the effector caspases, Dronc stimulates JNK levels, thus establishing an amplification loop, necessary for the full apoptotic response. The amplification step is squared for emphasis.

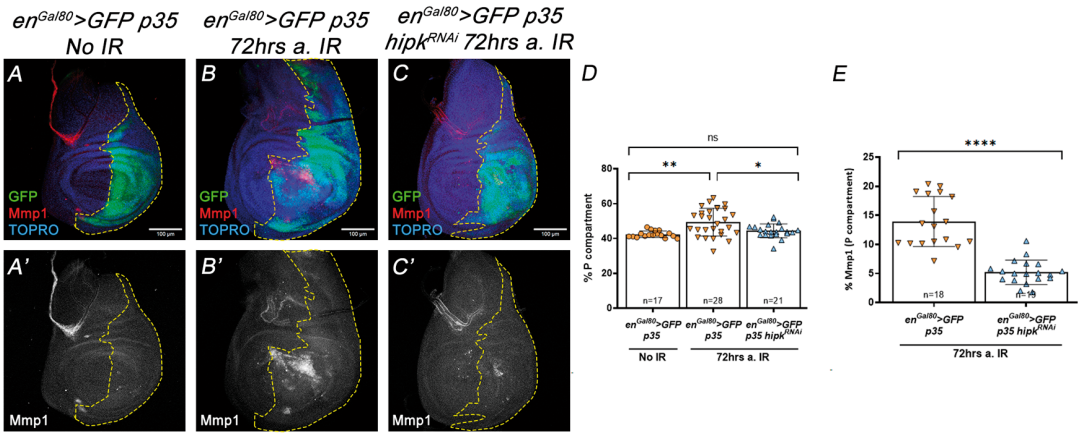

Figure S2. *hipk* is required to maintain JNK function and size increase after irradiation (IR)

Genotypes on top of the panels. (A, A') Non-irradiated discs show no JNK activity, monitored here by the presence of Metalloprotease 1, Mmp1 (in red), a target of JNK. (B, B') In irradiated discs of the *en^{Gal80}>GFP p35* genotype, the presence of the baculovirus protein P35 in the P compartment allows the survival of cells in which JNK has been induced by IR, thus increasing Mmp1 signal. Size of the compartment is also increased. (C, C') In *p35*-expressing irradiated discs in which *hipk* function is reduced by the expression of a *hipk^{RNAi}* construct, JNK activity and compartment size are much diminished. Quantifications in D, E. Date are shown as the means ± SD, the significant level was identified by *p<0,05; **p<0,01; ***p<0,001 and ****p<0,001.; ns: no significant.

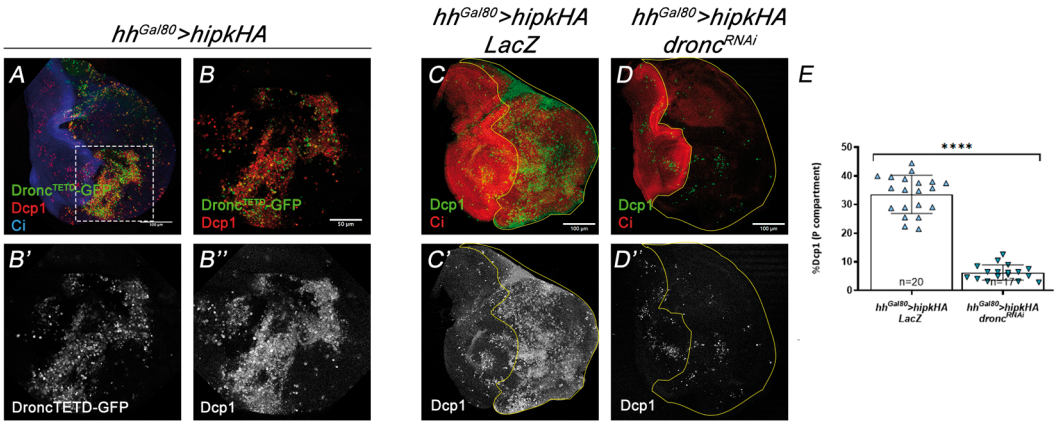

Figure S3. Overexpression of *hipk* results in *dronc*-dependent apoptosis

Genotypes on top of the panel. (A-B'') Overexpression of *hipk-HA* induces the expression of the Dronc-activity reporter *dronc^{TETD}-GFP* and Dcp1 antibody signal (A). B-B'' are amplifications of the inset in A. (C-D'') The Dcp1 signal induced by overexpression of *hipk-HA* (C, C') is significantly reduced by the simultaneous inactivation of *dronc* (D, D'). Ci, in red, marks the A compartment (in blue in A, in red in C, D). Quantifications in E. Date are shown as the means ± SD, the significant level was identified by *p<0,05; **p<0,01; ***p<0,001 and ****p<0,001.; ns: no significant.

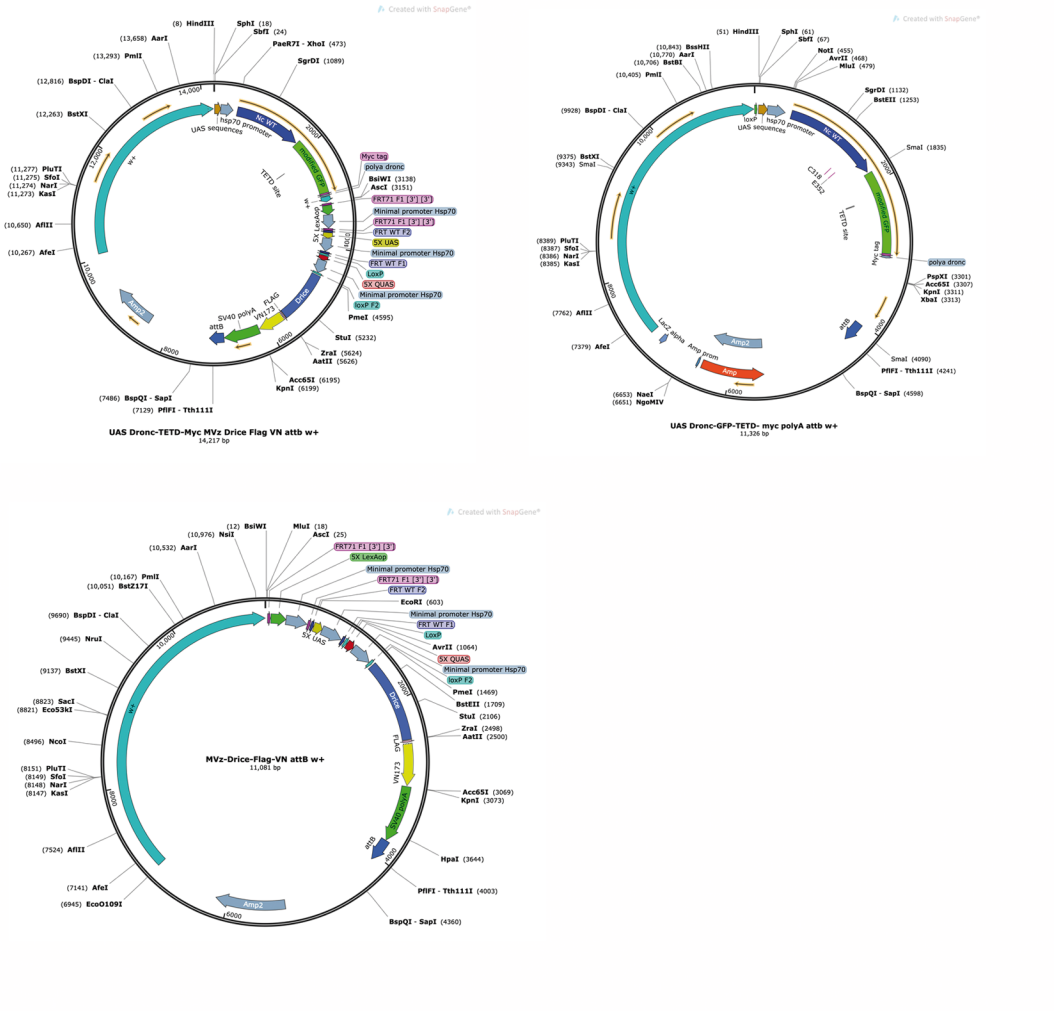

Figure S4. Plasmid maps of the MVz-Drice-Flag-VN, UAS-Dronc-GFP-Myc and UAS-Dronc-GFP-Myc/MVz-Drice-Flag-VN plasmids.