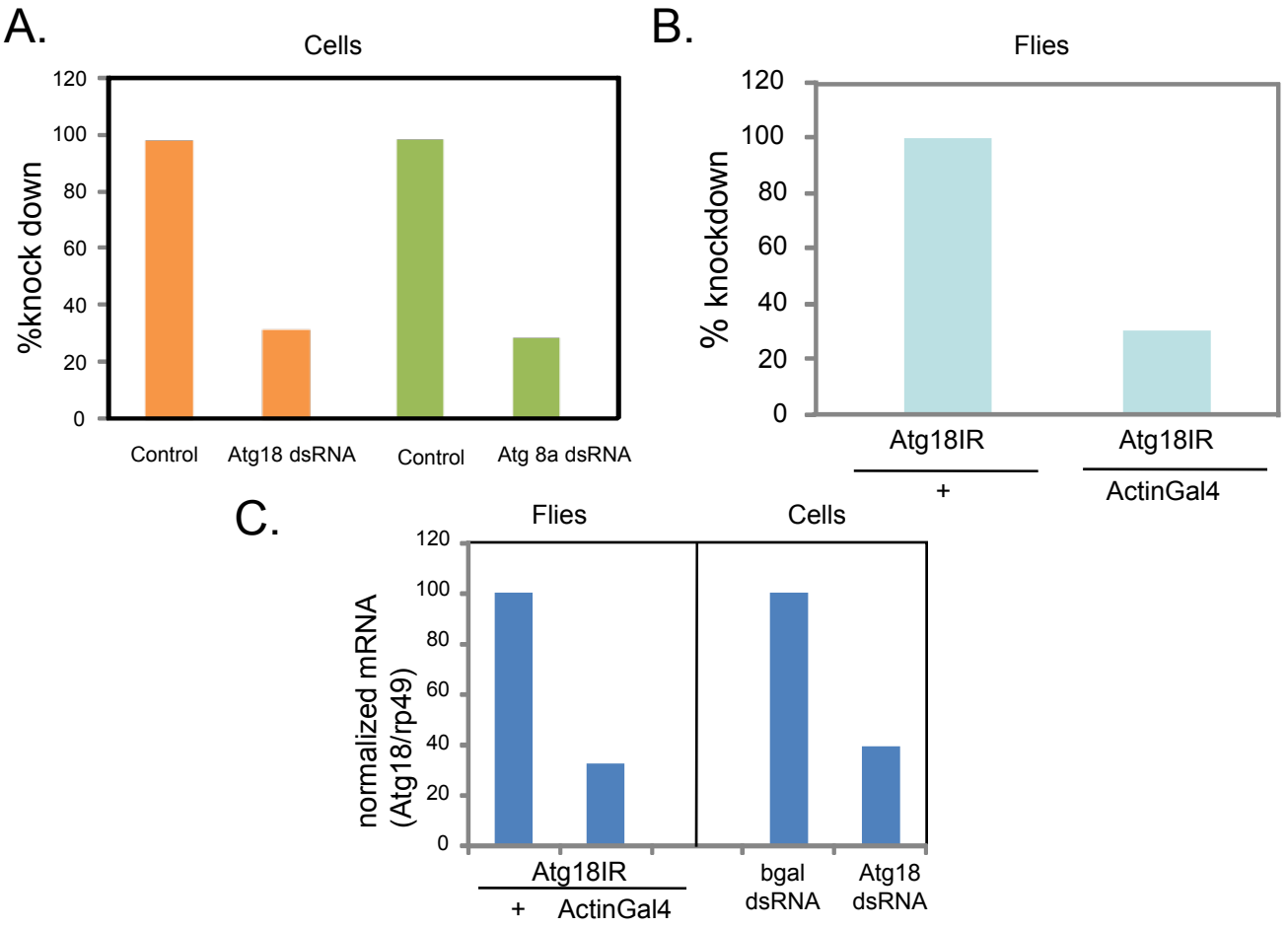
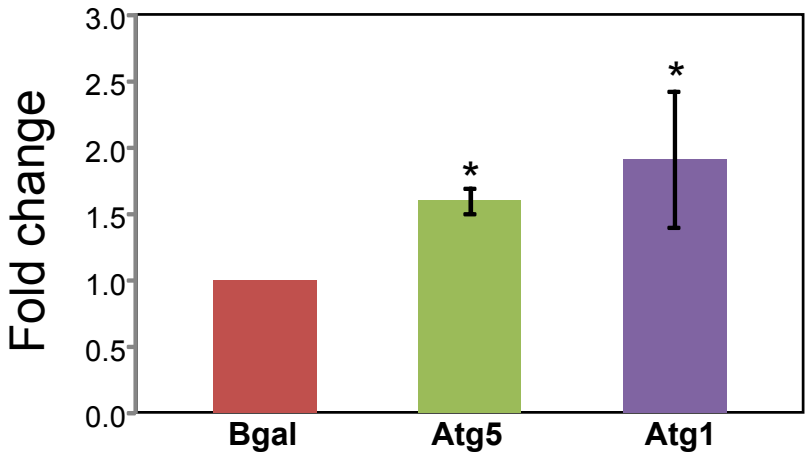
Shelly_Supplementary Figure 1



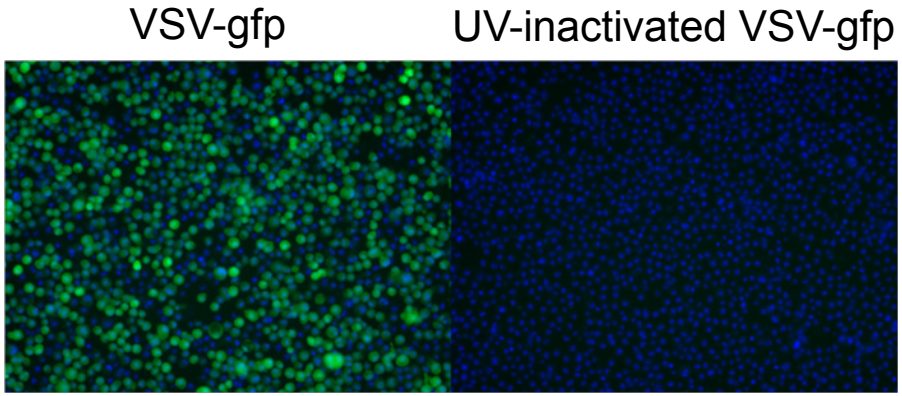
Supplementary Figure 1: dsRNA against autophagy genes is efficacious both in cells and flies as measured either by semi-quantitative RT-PCR or RT-qPCR. **A**. >75% depletion of the cognate message as measured by semi-quantitative RT-PCR against Atg18 (orange) or Atg8a (green) normalized to a cellular control (clathrin heavy chain). Control dsRNA treated samples are set to 100%. **B**. Semi-quantitative RT-PCR against Atg18 normalized to control (clathrin heavy chain). Adult flies expressing ubiquitous and high level dsRNA against Atg18 (Actin-Gal4; UAS-Atg18IR) were compared to sibling controls (+; UAS-Atg18IR) which were set to 100%. **C**. RT-qPCR against Atg18 normalized to control (rp49) in adult flies as in **B**. or in cells pre-treated with the indicated dsRNAs.

Shelly_Supplementary Figure 2



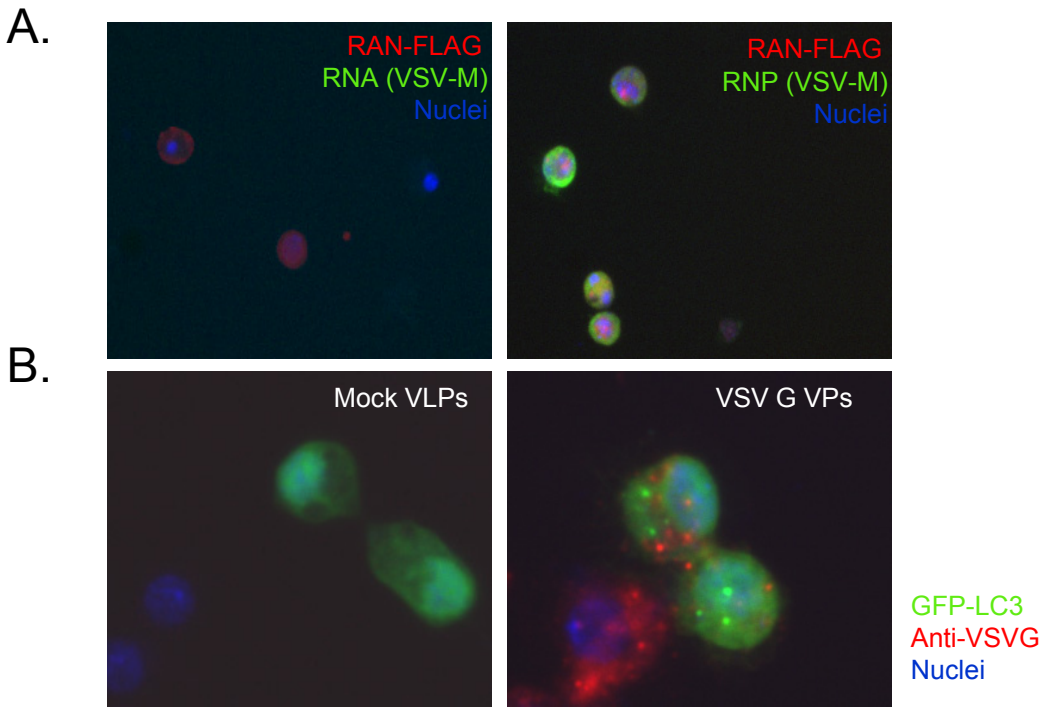
Supplementary Figure 2: Loss of autophagy genes leads to increased viral production. Viral titers of cells pre-treated with the indicated dsRNAs at 48 hours post infection with an MOI=0.1. Data presented as the fold change from control treatment of four averaged experiments; error bars are standard deviation; *p<0.01 student's ttest.

Shelly_Supplementary Figure 3



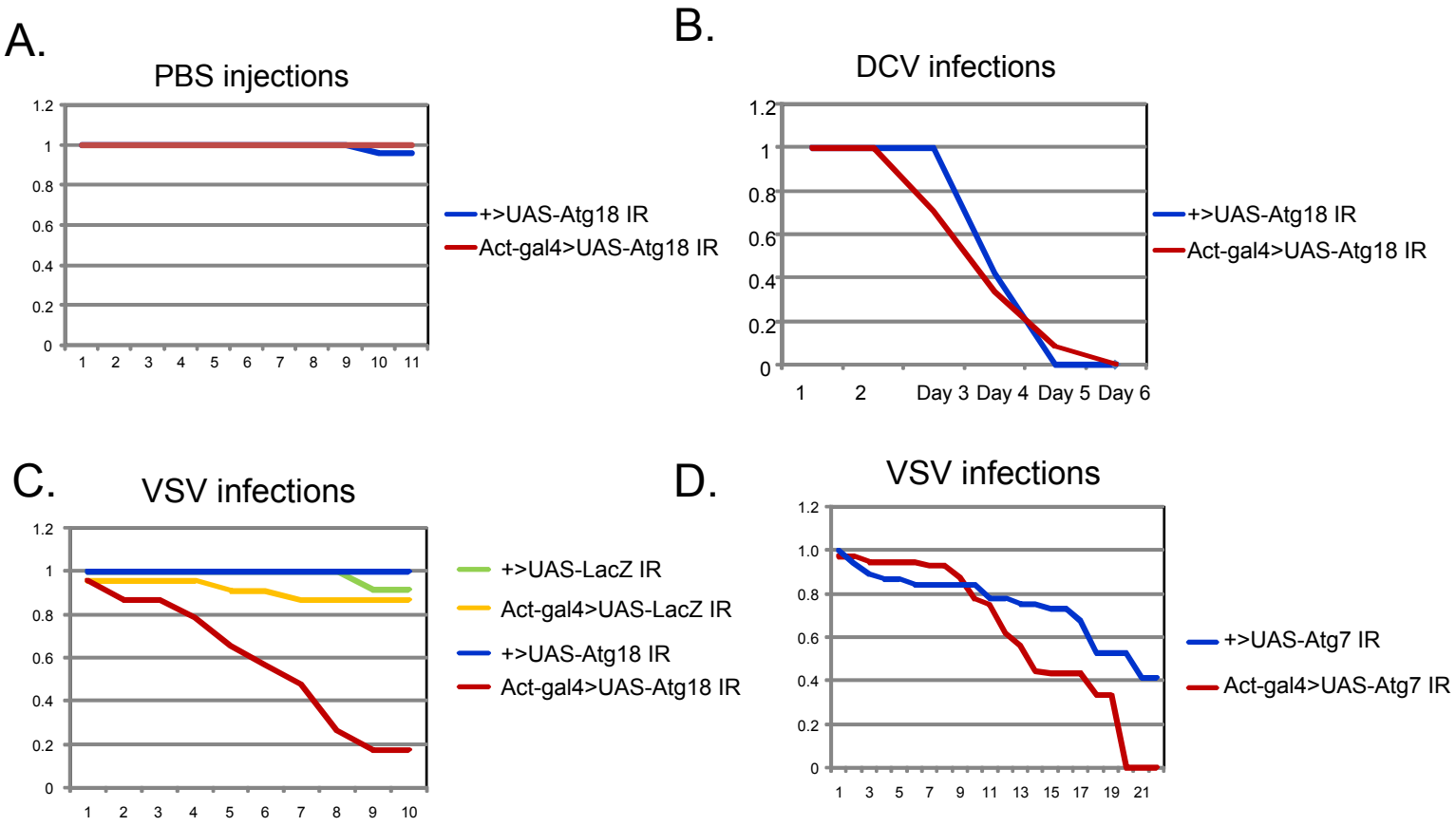
Supplementary Figure 3: Schneider cells were infected with VSV-GFP (MOI=9) or UV-inactivated VSV-GFP (equivalent volume) for 20 hours. Cells were processed for immunofluorescence and imaged using an automated microscope (ImagXpress Micro). There were no Gfp^{+} cells in the UV inactivated sample.

Shelly_Supplementary Figure 4



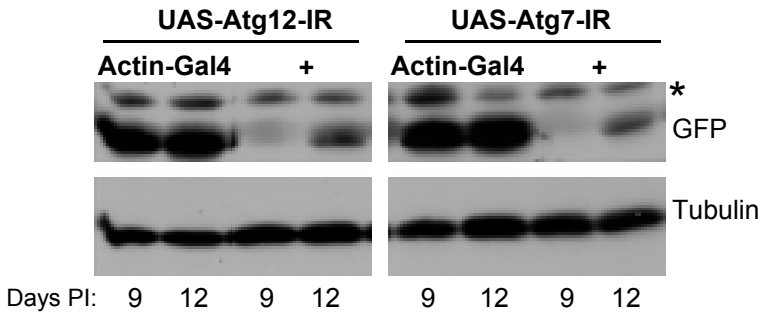
Supplementary Figure 4: Purified viral components reveal that VSV G is the PAMP. **A**. Purified VSV RNP but not VSV RNA is infectious when transfected into cells. Schneider cells were co-transfected with Flag-tagged RAN and either VSV RNA (left) or VSV RNP (right). Cells were processed for immunofluorescence with anti-FLAG (red), anti-VSV M (green) and nuclei (blue) and imaged. There were no VSV-M$^+$ cells in the viral RNA transfected sample while >95% of the Flag$^+$ (transfected) cells were VSV-M$^+$. **B**. Cells treated with VPs from VSV G transfected 293s but not blebs from mock-transfected cells are VSV G$^+$ and induce GFP-LC3 punctae. Cells were transfected with GFP-LC3 and then treated with VPs and processed for fluorescence with GFP-LC3 (green), anti-VSV G (red) and nuclei (blue).

Shelly_Supplementary Figure 5



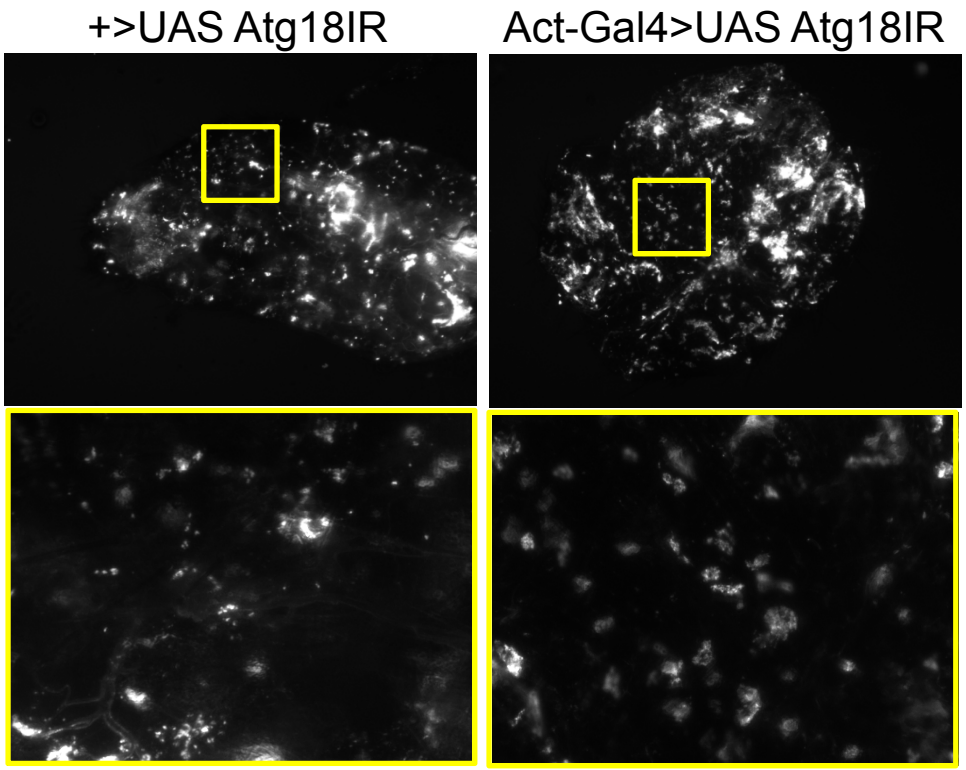
Supplementary Figure 5: Depletion of autophagy in adult flies specifically impairs VSV susceptiblity. Flies of the indicated genotypes were challenged with **A**. PBS or **B**. Drosophila C Virus (DCV) or **C**. VSV. **D**. VSV and monitored daily for mortality. . Log rank test reveals that loss of Atg7 significantly increases susceptibility ($p<0.05$). A representative experiment is shown. Each challenge was repeated at least three times.

Shelly_Supplementary Figure 6



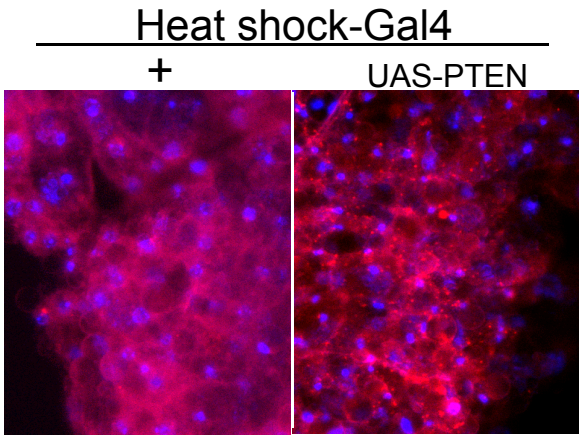
Supplementary Figure 6: Adult flies expressing ubiquitous and high level dsRNA against either Atg12 (Actin-Gal4; UAS-Atg12IR) or Atg7 (Actin-Gal4; UAS-Atg7IR) or their respective sibling controls (+; UAS-Atg12IR or +; UAS-Atg7IR) were challenged with VSV and monitored over time for viral replication as measured by immunoblot against virally produced GFP and normalized to a cellular control protein. There is a significant increase in VSV replication in flies depleted for autophagy genes measured by protein production. * indicates nonspecific background band.

Shelly_Supplementary Figure 7



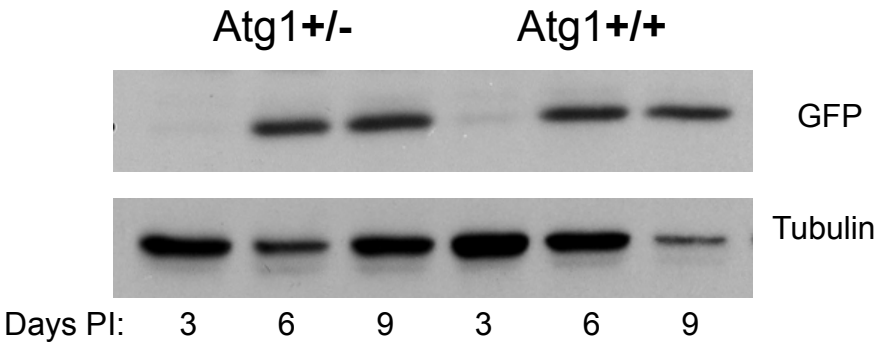
Supplementary Figure 7: Atg18 depleted flies have wild type numbers of hemocytes that are functional as measured by phagocytic activity. Adult flies were injected with 0.2um red fluorescent beads. Three days later the flies were processed for fluorescence microscopy. Hemocytes are the only phagocytic cell which take up fluorescent beads, which can be readily visualized through the cuticle and are distributed throughout the abdomen. Top panels at low magnification and the indicated inset is shown at higher magnification to show individual cells that have taken up multiple beads. Areas of increased saturation in top panels are likely aggregated hemocytes.

Shelly_Supplementary Figure 8



Supplementary Figure 8: Flies carrying a heat-shock inducible Gal4 were crossed to UAS-PTEN or control at room temperature. Progeny were collected and heat shocked at 37C for one hour the day before and the day of staining. Autophagy was monitored using Lysotracker staining to visualize the autophagosomes and counterstained with Hoescht 33342 to observe the nuclei. There are increased acidic compartments in the fat body cells of well-fed flies expressing the negative regulator PTEN compared to the diffuse dim staining in the control fat body cells.

Shelly_Supplementary Figure 9



Supplementary Figure 9: Flies of the indicated genotypes were challenged with VSV and monitored by immunoblot at the indicated time post infection.