Figure S1  DPY-21 regulates dauer arrest. (A) *dpy-21(null)* suppresses the dauer-constitutive phenotype of *akt-1* mutant animals at 27° (6.3% dauer arrest in *akt-1 dpy-21* mutants compared to 92.5% arrest in *akt-1*, P <0.0001). Data are pooled from four replicate experiments with at least 700 animals scored per genotype. (B) *dpy-21* RNAi suppresses the dauer-constitutive phenotype of the TGFβ-like pathway mutant *daf-8(e1393)* (11.9% dauer arrest in *daf-8* mutants exposed to *dpy-21* RNAi compared to 33.7% arrest in animals exposed to control vector, p = 0.0289). Data are from a single representative experiment with at least 450 animals scored per condition. In contrast, *dpy-21(null)* enhances dauer arrest of *daf-8* mutant animals (49.6% dauer arrest in *daf-8; dpy-21* mutants compared to 24.5% arrest in *daf-8*, p = 0.0004). Data are pooled from three replicate experiments with at least 950 animals scored per genotype. (C) *dpy-21(null)* suppresses the dauer-constitutive phenotype of *daf-9(k182)* and *daf-36(k114)* mutants raised on plates without supplemental cholesterol at 27° (60.7% dauer arrest in *dpy-21;daf-9* mutants compared to 79.9% arrest in *daf-9*, p = 0.0269; 82.6% dauer arrest in *daf-36 dpy-21* mutants compared to 98.4% arrest in *daf-36*, p = 0.0004). Data are pooled from four replicate experiments with at least 450 animals scored per genotype. Error bars indicate SEM. Raw data and statistics are presented in Table S1.