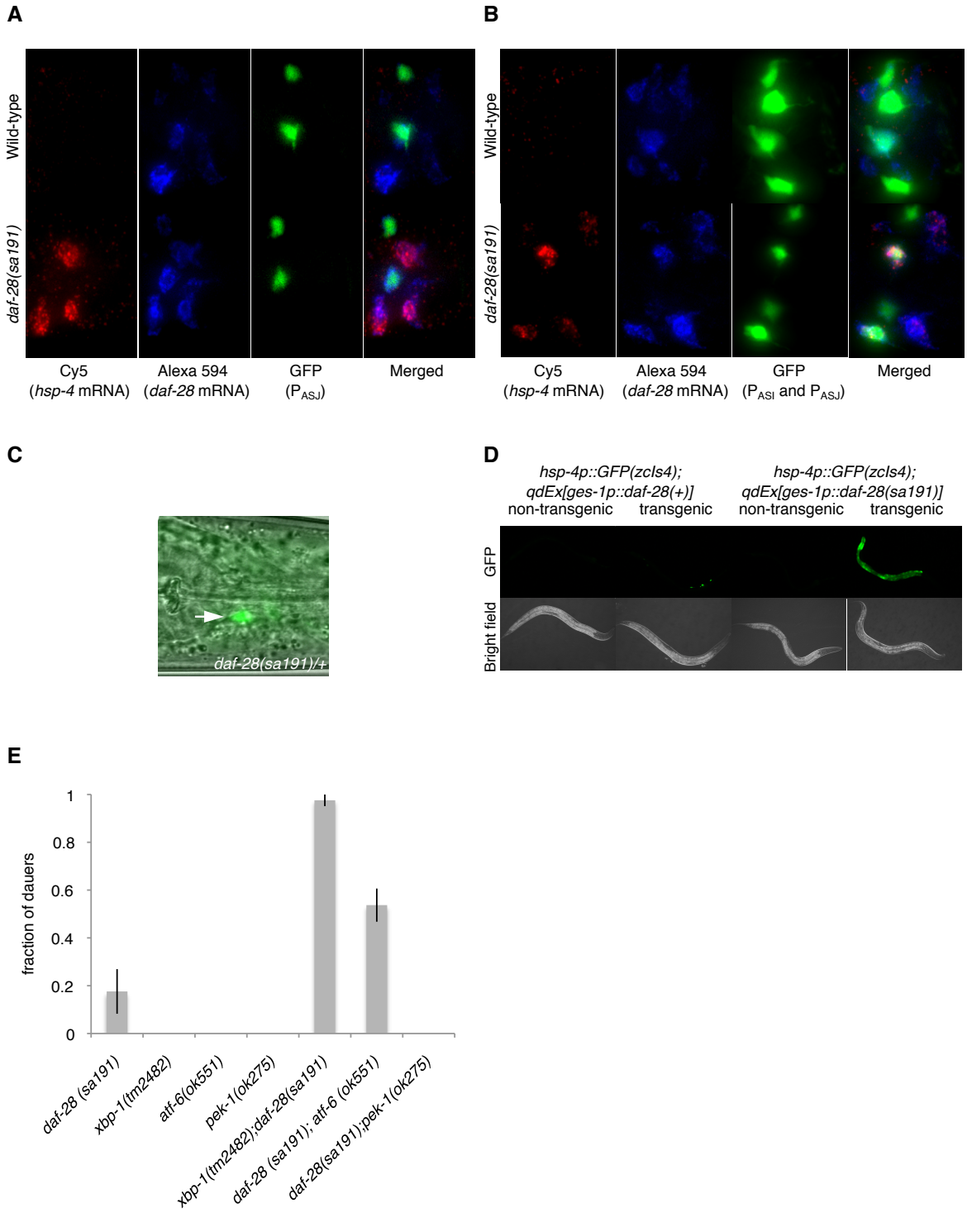
A
Wild-type
daf-28(sa191)
Cy5
(hsp-4 mRNA)
Alexa 594
(daf-28 mRNA)
GFP
(PASJ)
Merged
B
Wild-type
daf-28(sa191)
Cy5
(hsp-4 mRNA)
Alexa 594
(daf-28 mRNA)
GFP
(PASI and PASJ)
Merged
C
daf-28(sa191)/+
D
hsp-4p::GFP(zcIs4);
qdEx[ges-1p::daf-28(+)]
non-transgenic
transgenic
hsp-4p::GFP(zcIs4);
qdEx[ges-1p::daf-28(sa191)]
non-transgenic
transgenic
GFP
Bright field
E
fraction of dauers
1
0.8
0.6
0.4
0.2
0
daf-28 (sa191)
xbp-1(tm2482)
atf-6(ok551)
pek-1(ok275)
xbp-1(tm2482);daf-28(sa191)
daf-28 (sa191); atf-6 (ok551)
daf-28(sa191);pek-1(ok275)

Figure S1. The *daf-28 (sa191)* Mutation Causes ER Stress, Related to Figure 1

(A) Maximum Z projection of smFISH stacked images of pre-dauer larvae with indicated genotypes. The ASJ neurons are identified by expression of the translational fusion TRX-1::GFP [S1].

(B) Maximum Z projection of smFISH stacked images of pre-dauer larvae with indicated genotypes. The ASI and ASJ neurons are identified by GFP expression driven by the *daf-28* promoter [S2].

(C) Shown is fluorescence microscopy of the *daf-28(sa191)*/+ heterozygote that carries the *hsp-4p::GFP* transgene (green). The filled arrow indicates ASI.

(D) Shown is fluorescence microscopy of the animals with indicated genotypes that carry the *hsp-4p::GFP* transgene. *ges-1,* an intestine-specific promoter [S3], was used to drive ectopic expression of *daf-28*, both wild-type and *sa191* versions. *ofm-1p::GFP*, expressed in the coelomocytes, was used as a visible co-injection marker for transgenic animals. All images were recorded using the same exposure time.

Images shown represent five independent transgenic lines for *qdEx[ges-1p::daf-28(sa191)]*, all of which exhibit robust induction of *hsp-4p::GFP* expression in the intestine.

For *qdEx[ges-1p::daf-28(+)]*, images shown are representative of thirteen out of fourteen independent transgenic lines. One line carrying *qdEx[ges-1p::daf-28(+)]* exhibits modest and variable induction of *hsp-4p::GFP* expression in the intestine.

(E) Fractions of the *daf-28(sa191)* mutants with indicated *xbp-1, atf-6* and *pek-1* deficiencies that enter the dauer stage at 20 ºC. Similar results were obtained using another loss-of-function allele of *xbp-1, tm2457*. Plotted is mean ± SD. The number of trials and animals scored is documented in Table S3.

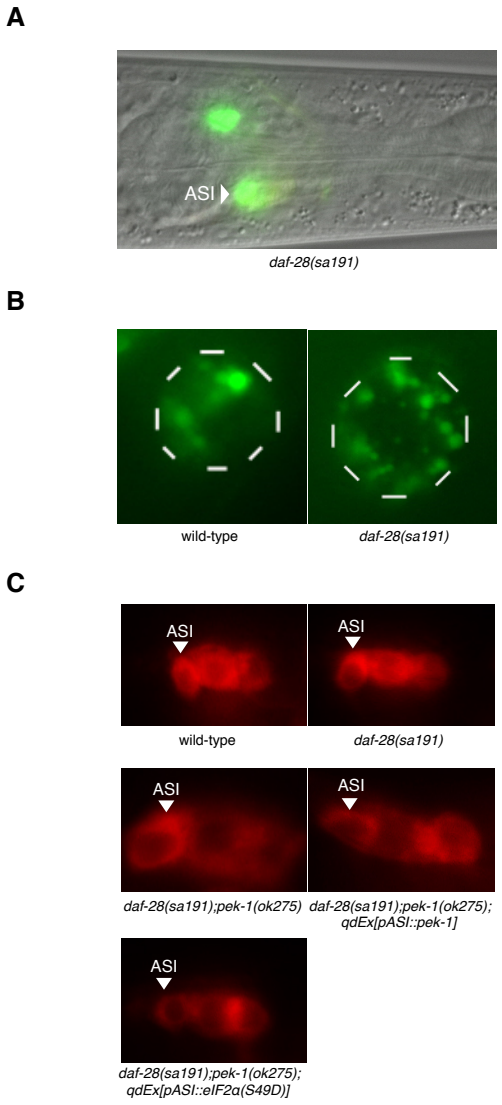

Figure S2. The *daf-28(sa191)* Mutation and Neuronal UPR Activation Does Not Compromise Cellular Survival and Function, Related to Figure 1

(A) Representative fluorescence microscopy of the anterior section of the *daf-28(sa191)* mutant carrying the *daf-28p::GFP* transgene. Note apparent expression of the transcriptional reporter (green).

(B) Representative fluorescence microscopy of the coelomocytes of the animals carrying the *daf-28p::DAF-28::GFP* transgene [S4] with indicated genetic backgrounds. Coelomocytes are specialized in taking up material from the pseudocoelomic fluid. Since *daf-28* is not expressed in the coelomocytes, the accumulation of DAF-28::GFP (note green puncta) could only be attributed to uptake from the pseudocoelom, where DAF-28::GFP, produced and secreted from the chemosensory neurons is deposited. The patterns depicted are representative of three independent experiments. Post-dauer animals were used in the experiments for consistency in coelomocyte identification.

(C) Fluorescence microscopy of the dye-filled amphid chemosensory neurons of the animals with indicated genotypes. The patterns depicted are representative of developmental stages both pre- and post-dauer entry at the restrictive temperature 25ºC. For transgenic animals, images shown represent observations from three independent lines.

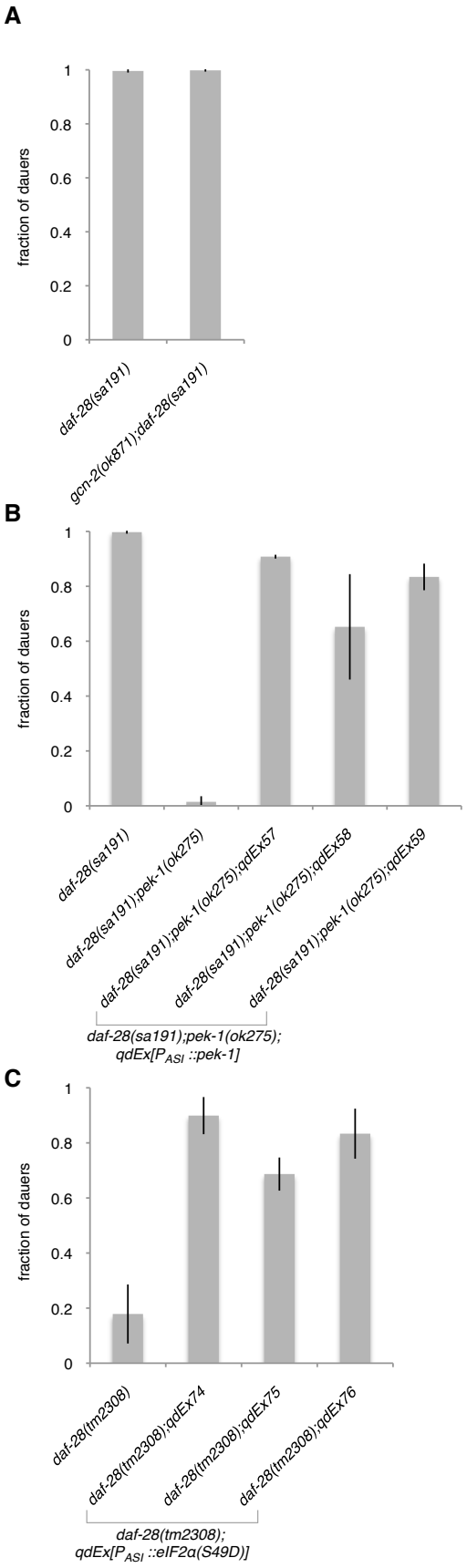

Figure S3. PEK-1 Phosphorylates eIF2α in the ASI Neuron Pair to Promote Entry into Dauer Diapause , Related to Figures 2 and 3

(A) Fractions of the animals with indicated genotypes that enter the dauer stage at 25 ºC. Unlike PEK-1, GCN-2, a *C. elegans* ortholog of the eIF2α kinase GCN2 that functions independently of ER stress, is not required for the constitutive entry into dauer in the *sa191* animals. These observations rule out the possibility that general dysregulation of eIF2α phosphorylation promotes dauer entry, and pointing to an ER stress-specific role of PEK-1 in promoting the dauer developmental decision.

(B) Results from a second ASI-specific promoter, *str*-3p, are shown here (referred to as P_{ASI}). The *daf-28(sa191);pek-1(ok275)* strain in this figure represents non-transgenic animals from all three lines. Both *str-3p* and *gpa-4p* were characterized and validated in previous studies [S5-S7].

(C) Fractions of the animals with indicated genotypes that enter the dauer stage at 25 ºC. The *daf-28(tm2308)* strain in this figure represents non-transgenic controls from all three lines. Results from the ASI-specific *gpa-4* promoter are shown in this figure (referred to as P_{ASI}).

Plotted is mean ± SD. The number of trials and animals scored is documented in Table S3.

Table S1. Data for Dauer Formation Assays, Related to Figure 1 and Discussion

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	97.8 ± 3.9	19	1017
daf-16(mgDf47);daf-3(mgDf90)	0 ± 0	10	690
daf-16(mgDf47);daf-28(sa191);daf-3(mgDf90)	38.4 ± 10.4*	8	371

Note: The *daf-16(mgDf47);daf-28(sa191);daf-3(mgDf90)* animals only formed partial dauers (*), the features of which were described in [S8]. A previous study showed that *daf-16;daf-28(sa191)* mutants only formed partial dauers, and the fraction of partial dauers was significantly lower than the fraction of *bona fide* dauers formed by the *daf-28(sa191)* mutant [S9].

Table S2. Data for Dauer Formation Assays, Related to Figures 1, 2 and 3

Figure 1A

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
N2	0 ± 0	7	221
daf-28(gk411072)	0.6 ± 0.5	6	657
daf-28(tm2308)	6.3 ± 4.8	19	887
daf-28(gk411072)/daf-28(tm2308)	0.4 ± 1.3	10	183
daf-28 (sa191)/daf-28 (gk411072)	72.5 ± 1.0	5	77
daf-28 (sa191)/daf-28 (tm2308)	83.7 ± 6.0	9	399
daf-28(sa191)	97.5 ± 4.0	16	857

Note: We observed minimal or no Daf-c phenotype in animals carrying two putative null alleles of *daf-28*, the *daf-28(tm2308)* [S10] and *daf-28(gk411072)* alleles that were not available at the time of prior studies, indicating that the robust Daf-c phenotype of the *sa191* mutant cannot be attributed to loss of function of the insulin-encoding gene *daf-28*. In addition, the trans-heterozygote between the *sa191* mutation and each of the *daf-28* null alleles exhibited constitutive entry into dauer similar to the *sa191* homozygote, corroborating the reported gain-of-function nature of the *sa191* mutation.

Figure 2A

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	99.7 ± 0.5	8	665
daf-28(sa191);ire-1(v33)	99.6 ± 1.1	7	264
daf-28(sa191);xbp-1(tm2482)	99.6 ± 1.0	7	357
daf-28(sa191);atf-6(ok551)	99.1 ± 1.4	8	595

Figure 2B

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	99.7 ± 0.5	8	665
daf-28(sa191);pek-1(ok275)	1.9 ± 2.8	9	891
daf-28(sa191);pek-1(tm629)	0.9 ± 1.2	6	456

Figure 2C

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
N2	0 ± 0	8	264
pek-1(ok275)	0 ± 0	7	537
daf-2(e1368)	96.8 ± 3.5	7	325
daf-2(e1368);pek-1(ok275)	100 ± 0	4	237
daf-7(ok3125)	100 ± 0	2	97
daf-7(ok3125);pek-1(ok275)	97.8 ± 1.5	6	411

Figure 2D

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	99.7 ± 0.5	8	665
daf-28(sa191);pek-1(ok275)	4.4 ± 3.45	12	542
daf-28(sa191);pek-1(ok275);qdEx48	82.8 ± 3.9	4	103
daf-28(sa191);pek-1(ok275);qdEx49	85.9 ± 6.4	4	194
daf-28(sa191);pek-1(ok275);qdEx50	91.9 ± 5.0	4	278

Figure 3A

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	94.1 ± 5.1	13	399
daf-28(sa191);qdEx51	73.1 ± 3.6	4	141
daf-28(sa191);qdEx52	67.9 ± 7.6	4	169
daf-28(sa191);qdEx53	54.8 ± 11.4	4	119

Figure 3B

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191);pek-1(ok275)	2.4 ± 2.0	14	1037
daf-28(sa191);pek-1(ok275);qdEx54	69.9 ± 1.4	5	254
daf-28(sa191);pek-1(ok275);qdEx55	91.5 ± 8.0	5	117

Table S3. Data for Dauer Formation Assays, Related to Figures 3, S1, S2 and S3 and Discussion

Related to Figure 3

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	98.3 ± 2.4	10	536
daf-28(sa191);qdEx60	98.1 ± 1.3	4	139
daf-28(sa191);qdEx61	98.4 ± 1.4	3	135
daf-28(sa191);qdEx62	98.3 ± 2.9	3	54
daf-28(sa191);pek-1(ok275)	0.1 ± 0.4	12	612
daf-28(sa191);pek-1(ok275);qdEx71	0.4 ± 0.8	4	196
daf-28(sa191);pek-1(ok275);qdEx72	0 ± 0	4	118
daf-28(sa191);pek-1(ok275);qdEx73	0 ± 0	4	156

Note: *qdEx60*, *qdEx61*, *qdEx62*, *qdEx71*, *qdEx72* and *qdEx73* represent *qdEx[gpa-4p::eIF2α(+)::unc-54 3′UTR]*.

Figure S1E

Genotype	Mean percentage of dauers ± standard deviation (percentage) at 20 ºC	Number of trials	Number of total (pooled) animals
daf-28(sa191)	17.6 ± 9.3	7	511
xbp-1(tm2482)	0 ± 0	4	228
atf-6(ok551)	0 ± 0	4	280
pek-1(ok275)	0 ± 0	4	274
xbp-1(tm2482);daf-28(sa191)	97.6 ± 2.4	5	228
daf-28(sa191);atf-6(ok551)	53.7 ± 6.9	6	280
daf-28(sa191);pek-1(ok275)	0 ± 0	6	304

Figure S3A

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	99.6 ± 0.6	8	659
gcn-2(ok871);daf-28(sa191)	99.8 ± 0.5	6	427

Figure S3B

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	99.7 ± 0.5	8	665
daf-28(sa191);pek-1(ok275)	1.4 ± 2.0	12	591
daf-28(sa191);pek-1(ok275);qdEx57	90.8 ± 0.7	4	121
daf-28(sa191);pek-1(ok275);qdEx58	65.2 ± 19.2	4	102
daf-28(sa191);pek-1(ok275);qdEx59	83.4 ± 4.8	4	246

Figure S3C

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(tm2308)	17.9 ± 10.7	14	412
daf-28(tm2308);qdEx74	89.9 ± 6.7	5	154
daf-28(tm2308);qdEx75	68.7 ± 6.0	4	146
daf-28(tm2308);qdEx76	83.3 ± 9.1	5	122

Related to Figure S2 and Discussion

Genotype	Mean percentage of dauers ± standard deviation (percentage)	Number of trials	Number of total (pooled) animals
daf-28(sa191)	99.6 ± 0.6	6	493
daf-28(sa191);daf-3(mgDf90)	99.8 ± 0.5	6	564

Note: DAF-3/SMAD is required for chemosensory neuron ablation to mediate dauer formation [S11]. The result is consistent with a previous genetic study [S9] using a different allele of *daf-3*, namely *e1376*.

Supplemental Experimental Procedures

***Caenorhabditis elegans* Strains**

Additional strains were used in Figures S1, S2 and S3 and Tables S1, S2 and S3: GR1455 *mgIs40[daf-28p::GFP]*, ZD803 *daf-28(sa191);mgIs40[daf-28p::GFP],* OE3010 *ofEx4[trx-1p::TRX-1::GFP]*, ZD1065 *daf-28(sa191);ofEx4[trx-1p::TRX-1::GFP]*, ZD747 *svIs69[daf-28p::DAF-28::GFP]*, ZD805 *daf-28(sa191);svIs69[daf-28p::DAF-28::GFP]*, ZD1066-1068 *zcIs4[hsp-4p::GFP];qdEx80-82[ges-1p::daf-28(+)::unc-54 3′UTR]*, ZD1069-1071 *zcIs4[hsp-4p::GFP];qdEx80-82[ges-1p::daf-28(sa191)::unc-54 3′UTR]* ZD815 *gcn-2(ok871);daf-28(sa191)*, ZD954-956 *daf-28(sa191);pek-1(ok275);qdEx57-59[str-3p::pek-1::unc-54 3′UTR]*, ZD933, 1021 and 1023 *daf-28(tm2308);qdEx74-76[gpa-4p::eIF2α(S49D)::unc-54 3′UTR]*, ZD967-969 *daf-28(sa191);qdEx60-62[gpa-4p::eIF2α(WT)::unc-54 3′UTR]*, ZD995-997 *daf-28(sa191);pek-1(ok275);qdEx71-73[gpa-4p::eIF2α(WT)::unc-54 3′UTR],* GR1311 *daf-3(mgDf90)*, ZD822 *daf-28(sa191);daf-3(mgDf90),* ZD939 *daf-16(mgDf47);daf-28(sa191);daf-3(mgDf90)* and ZD941 *daf-16(mgDf47);daf-3(mgDf90)*.

The strain carrying *daf-28(tm2308)* allele was kindly provided by G. Ruvkun. The strain carrying *pek-1(ok275)* was kindly provided by M. Crowder. The strain carrying *svIs69* was kindly provided by S. Tuck. Unless otherwise noted, the ZD strains have been outcrossed at least three times. ZD940 was outcrossed twice. Strains carrying the *pek-1(tm629)* and *atf-6(ok551)* alleles were outcrossed once to the *daf-28(sa191)* strain. In addition to using two independent alleles of *pek-1,* the transheterozygote between the *pek-1(ok275)* and *pek-1(tm629)* alleles was generated in the *sa191* background to confirm the suppression of the Daf-c phenotype by *pek-1* loss-of-function mutations. Double and triple mutants were generated and genotyped using standard methods (relying on PCR or visible phenotypes, when

possible). Experiments involving transheterozygotes were performed by crossing the two indicated strains. The mated P_0 hermaphrodites were allowed to lay eggs under the same condition as the original strains, and the F_1 cross progeny was confirmed and scored as L4 or dauer. The *daf-28(sa191)/daf-28(gk411072)* and *daf-28(tm2308)/daf-28(gk411072)* heterozygotes were derived from crosses involving the strain VC30082, carrying the *daf-28(gk411072)* allele.

Constructs and Generation of Transgenic Lines

The *gpa-4p::pek-1::unc-54 3′UTR* construct includes 2.9 kb of *gpa-4* promoter, 4.2 kb of *pek-1* genomic region (including short introns) and 0.7 kb of *unc-54* 3′UTR. The *gpa-4* promoter region was amplified by using 5′ primer 5′-ATCACACCGTCGTGAGCTA-3′ and 3′ primer 5′-CTATATAAT ACACACTCATTGTTGAAAAGTGTTCACAAAATG-3′ (contains an overhang complementary to the *pek-1* genomic region for subsequent PCR fusion). The *pek-1* genomic region was amplified by using 5′ primer 5′-ATGAGTGTGTATTATATAG-3′ and 3′ primer 5′-AGGCACGGGCGCGAGATGTTAT TGGAGAAATTTATGAG-3′ (contains an overhang complementary to the *unc-54* 3′UTR region for subsequent PCR fusion). The *unc-54* 3′UTR region was amplified by using 5′ primer 5′-CATCTCGCGCCCGTGCCT-3′ and 3′ primer 5′-AAGGGCCCGTACGGCCGACTAGTAGG-3′. Nested primers were used in subsequent PCR fusion, as previously described [S12]. The *str-3p::pek-1::unc-54 3′UTR* construct includes 2.9 kb of *str-3* promoter, 4.2 kb of *pek-1* genomic region (including short introns) and 0.7 kb of *unc-54* 3′UTR. The *str-3* promoter region was amplified by using 5′primer 5′-TTCAGAAGGCAGATGCAAAA-3′ and 3′ primer 5′-TAAAACTATATAATACACACTCATGT TCCTTTTGAAATTGAGGCAGT-3′ (contains an overhang complementary to the *pek-1* genomic region for subsequent PCR fusion). The *str-3p::pek-1::unc-54 3′UTR* construct was injected at 40 ng/μl.

The *gpa-4p::eIF2α::unc-54 3′UTR* construct includes 2.9 kb of *gpa-4* promoter, 1.1 kb of *eIF2α* cDNA (wild-type, phosphomimetic and unphosphorylatable versions [S13] kindly provided by S.Takagi) and 0.7 kb of *unc-54* 3′UTR. The *gpa-4* promoter and *unc-54* 3′UTR regions were amplified as described above. The 3′ primers used to amplify *gpa-4p* contain an overhang complementary to the *eIF2α* cDNA region for subsequent PCR fusion. An indicated version of *eIF2α* was amplified by using 5′ primer 5′-ATGAAATGCCGTTTCTACGAG-3′ and 3′ primer 5′-AGGCACGGGCGCGAGATGTT AATCATCCTCCTCATCACTGT-3′ (contains an overhang complementary to the *unc-54* 3′UTR region for subsequent PCR fusion). Nested primers were used in subsequent PCR fusion, as previously described.

The *ges-1p::daf-28::unc-54 3′UTR* construct includes 1.5 kb of *ges-1* promoter, 0.7 kb of *daf-28* genomic region (including an intron) and 0.7 kb of *unc-54* 3′UTR. The *ges-1* promoter region was amplified by using 5′ primer 5′-CTTCGGGCGCTACCAATAAG-3′ and 3′ primer 5′-GATGGCGATG AGCTTGCAGTTCATCTGAATTCAAAGATAAGATATGT-3′ (contains an overhang complementary to the *daf-28* genomic region for subsequent PCR fusion). The *daf-28* genomic region (wild-type and *sa191* versions) was amplified by using 5′ primer 5′-TCTCCTCTCAACAACT ATCTCAACA-3′ and 3′ primer 5′-AGTCAGAGGCACGGGCGCGAGATGTTAAAGAAGCAAACGTGGGCAA-3′ (contains an overhang complementary to the *unc-54* 3′UTR region for subsequent PCR fusion). Nested primers were used in subsequent PCR fusion, as previously described. The *ges-1p::daf-28::unc-54 3′UTR* construct was injected at 50 ng/μl.

Dauer Assay

For dauer assays involving transgenic strains, gravid extrachromosomal-array-carrying hermaphrodites were used for egg lay, and both non-transgenic and transgenic offspring were scored as

dauer or L4. Fractions of dauers were then derived for both non-transgenic and transgenic cohorts. Paired Student's t-test analyses between transgenic and non-transgenic dauer fractions from each trial were conducted for all lines. At least three independent lines were tested for each transgenic construct.

Microscopy

DiI stock was diluted in M9 to the final concentration of 10 μg/ml. Worms raised at 22.5 ºC or 25ºC were transferred to the solution and incubated at room temperature for two hours before imaging. Rhodamine filter was used for imaging and the fluorescence signals were recorded without saturation. For the purpose of neuron identification by dye filling, both pre-dauer and post-dauer animals were mounted. Dauer animals were not used for this purpose because some amphid neurons are altered in shape and position in the dauer stage [S14].

Single Molecule Fluorescent *in situ* Hybridization (smFISH)

smFISH was performed as described previously [S15]. Pre-dauer larvae grown at 25ºC were used in the experiments. *hsp-4* probes were coupled to Cy5 and *daf-28* probes were coupled to Alexa Fluor 594. The oligonucleotide probes were designed based on the coding sequences of *hsp-4* and *daf-28,* using Stellaris FISH probe designer. To ensure specificity, *hsp-4* probes were designed to target the deleted region of the *hsp-4(gk514)* allele, and the probes yielded detectable signals in wild-type animals, but not in the *hsp-4(gk514)* mutant (data not shown). The same strategy could not be adopted for *daf-28* probe design because the deleted region of the only deletion allele of *daf-28* available (*tm2308*) is too small. The designed *daf-28* probes allowed us to detect *daf-28* mRNA in the ASI and ASJ neurons, corroborating observations from previous studies using GFP transcriptional and translational reporters [S2, S4]. Note, however, that *daf-28* mRNA expression levels are lower in ASJ (Figures S1A and S1B).

Hybridized larvae were imaged using a Nikon Ti-E inverted fluorescence microscope equipped with a 100x oil-immersion objective and a Photometrics Pixis 1024 CCD camera using MetaMorph software and appropriate optical filters for Cy5, Alexa Fluor 594 and GFP. At least 10-20 animals per genotype were examined, and the observed *daf-28* and *hsp-4* mRNA expression patterns were derived from at least three independent experiments. ImageJ was used to perform maximum Z projection of stacked images. For image presentation, only linear adjustment (brightness and contrast) was applied equally to images from different genotypes to optimize signal-to-noise ratios for each channel.

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