**Supplementary Materials and Methods**

**Strains**

*C. elegans* strains were grown at 20 °C as described, unless otherwise indicated (Brenner 1974). N2 (Bristol) was the reference wild type strain. Strains and mutations used in this study include:

CB4856 (Hawaiian) (Wicks *et al.* 2001)

CB767 *bli-3(e767) I* (Brenner 1974)

SP2275 *tsp-15(sv15) I* (Moribe *et al.* 2012)

CSM637 *skn-1(lax120) IV* (this study), derived from SPC167: *dvIs19 III; skn-1(lax120) IV* (Paek *et al.* 2012).

CSM296 *bli-3(mac37) I* (Xu *et al.* 2015)

CSM297 *bli-3(mac38) I* (Xu *et al.* 2015)

CSM627 *bli-3(mac40) I* (Xu *et al.* 2015) (backcrossed 4 times)

CSM300 *bli-3(mac41) I* (Xu *et al.* 2015)

CSM681 *tsp-15(mac33) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM682 *wdr-23(mac35) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM683 *wdr-23(mac32) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM797 *wdr-23(mac36) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM798 *wdr-23(mac42) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM799 *wdr-23(mac43) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM800 *wdr-23(mac44) I* (Xu *et al.* 2015) (backcrossed 6 times)

CSM298 *skn-1*(*mac39) IV* (Xu *et al.* 2015)

CSM424 *bli-3(mac48) I* (this study)

CSM426 *bli-3(mac50) I* (this study)

CSM825 *bli-3(mac52) I* (this study, backcrossed 6 times)

CSM430 *bli-3(mac54) I* (this study)

CSM432 *bli-3(mac56) I* (this study)

CSM433 *bli-3(mac57) I* (this study)

CSM434 *bli-3(mac58) I* (this study)

CSM435 *bli-3(mac59) I* (this study)

CSM436 *bli-3(mac60) I* (this study)

CSM437 *bli-3(mac61) I* (this study)

CSM438 *bli-3(mac62) I* (this study)

CSM439 *bli-3(mac63) I* (this study)

CSM440 *bli-3(mac64) I* (this study)

CSM441 *bli-3(mac65) I* (this study)

CSM826 *bli-3(mac66) I* (this study, backcrossed 6 times)

CSM827 *bli-3(mac68) I* (this study, backcrossed 6 times)

CSM445 *bli-3(mac69) I* (this study)

CSM425 *wdr-23(mac49) I* (this study)

CSM801 *wdr-23(mac51) I* (this study, backcrossed 6 times)

CSM446 *wdr-23(mac70) I* (this study)

CSM684 *skn-1(mac53) IV* (this study, backcrossed 6 times)

CSM752 *doxa-1(mac55) III* (this study, backcrossed 6 times)

CSM753 *doxa-1(mac67) III* (this study, backcrossed 6 times)

CSM663 *skn-1(mac411) IV* (this study)

CSM665 *skn-1(mac413) IV* (this study)

CSM667 *skn-1(mac415) IV* (this study)

CSM668 *skn-1(mac416) IV* (this study)

CSM669 *mac396* (this study)

CSM670 *mac397* (this study)

CSM679 *mac401* (this study)

CSM695 *mac403* (this study)

CSM664 *mac412* (this study)

CSM666 *mac414* (this study)

CSM638 *skn-1(zu135) IV/nT1[qIs51] (IV; V)* (Bowerman *et al.* 1992; Bishop and Guarente 2007)

CL2166 *dvIs19 [pAF15(gst-4::GFP::NLS)]* (Link and Johnson 2002)

CSM689 *bli-3(e767) I; skn-1(mac53) IV*

CSM898 *bli-3(e767) I; skn-1(lax120) IV*

CSM639 *bli-3(mac40) I; skn-1(lax120) IV*

CSM645 *bli-3(mac40) I; skn-1(mac53) IV*

CSM686 *bli-3(e767) wdr-23(mac32) I*

CSM848 *bli-3(e767) wdr-23(mac35) I*

CSM824 *bli-3(mac40) wdr-23(mac32) I*

CSM685 *bli-3(mac40) wdr-23(mac35) I*

CSM821 *bli-3(mac40) wdr-23(mac36) I*

CSM843 *bli-3(mac40) wdr-23(mac42) I*

CSM822 *bli-3(mac40) wdr-23(mac44) I*

CSM823 *bli-3(mac40) wdr-23(mac51) I*

CSM648 *tsp-15(sv15) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM1045 *tsp-15(mac33) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM649 *wdr-23(mac35) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM1044 *wdr-23(mac32) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM650 *bli-3(mac40) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM1046 *bli-3(mac52) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM1047 *bli-3(mac66) I; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM868 *doxa-1(mac55) III; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM1048 *doxa-1(mac67) III; skn-1(zu135) IV/nT1[qIs51] (IV; V)*

CSM850 *macEx450[dpy-7p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM851 *macEx451[dpy-7p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM852 *macEx452[nhx-2p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM853 *macEx453[nhx-2p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM854 *macEx454[nhx-2p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM855 *macEx455[nhx-2p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM856 *macEx456[nhx-2p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM857 *macEx457[nhx-2p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM858 *macEx458[ges-1p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM859 *macEx459[ges-1p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM860 *macEx460[ges-1p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM861 *macEx461[ges-1p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM862 *macEx462[ges-1p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM863 *macEx463[ges-1p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM864 *macEx464[skn-1ap::skn-1a(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM865 *macEx465[skn-1ap::skn-1a(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM866 *macEx466[skn-1ap::skn-1a(WT) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM869 *macEx467[skn-1ap::skn-1a(mac53) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM870 *macEx468[skn-1ap::skn-1a(mac53) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM871 *macEx469[skn-1ap::skn-1a(mac53) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM872 *macEx470[skn-1cp::skn-1a(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM873 *macEx471[skn-1cp::skn-1a(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM874 *macEx472[skn-1cp::skn-1a(WT) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM875 *macEx473[skn-1cp::skn-1a(mac53) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM876 *macEx474[skn-1cp::skn-1a(mac53) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM877 *macEx475[skn-1cp::skn-1a(mac53) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM885 *macEx477[skn-1cp::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM886 *macEx478[skn-1cp::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM887 *macEx479[skn-1cp::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM888 *macEx480[myo-3p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM889 *macEx481[myo-3p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM890 *macEx482[myo-3p::skn-1c(WT) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM891 *macEx483[myo-3p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #1*

CSM892 *macEx484[myo-3p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #2*

CSM893 *macEx485[myo-3p::skn-1c(mac53) cDNA::3’UTR; myo-2p::mcherry] #3*

CSM899 *tsp-15(mac33) I; macEx490[myo-3p::GFP] #1*

CSM900 *tsp-15(mac33) I; macEx491[myo-3p::GFP] #2*

CSM901 *tsp-15(mac33) I; macEx492[tsp-15p::tsp-15 cDNA::3’UTR; myo-3p::GFP] #1*

CSM902 *tsp-15(mac33) I; macEx493[tsp-15p::tsp-15 cDNA::3’UTR; myo-3p::GFP] #2*

CSM928 *tsp-15(mac33)* I; *macEx504[dpy-7p::tsp-15 cDNA::3'UTR; myo-3p::GFP]* #*1*

CSM929 *tsp-15(mac33)* I; *macEx505[dpy-7p::tsp-15 cDNA::3'UTR; myo-3p::GFP] #2*

CSM903 *wdr-23(mac32) I; macEx494[myo-3p::GFP] #1*

CSM904 *wdr-23(mac32) I; macEx495[myo-3p::GFP] #2*

CSM905 *wdr-23(mac32) I; macEx496[wdr-23ap::wdr-23a cDNA::3’UTR; myo-3p::GFP] #1*

CSM906 *wdr-23(mac32) I; macEx497[wdr-23ap::wdr-23a cDNA::3’UTR; myo-3p::GFP] #2*

CSM973 *wdr-23(mac32) I; macEx529[wdr-23bp::wdr-23b cDNA::3’UTR; myo-3p::GFP] #1*

CSM974 *wdr-23(mac32) I; macEx530[wdr-23bp::wdr-23b cDNA::3’UTR; myo-3p::GFP] #2*

CSM907 *doxa-1(mac55) III; macEx498[myo-3p::GFP] #1*

CSM908 *doxa-1(mac55) III; macEx499[myo-3p::GFP] #2*

CSM909 *doxa-1(mac55) III; macEx500[doxa-1p::doxa-1 cDNA::3’UTR; myo-3p::GFP] #1*

CSM910 *doxa-1(mac55) III; macEx501[doxa-1p::doxa-1 cDNA::3’UTR; myo-3p::GFP] #2*

**Genetic screens and mapping of mutations**

For F2 screens, synchronized L4 wild-type animals (P0) were mutagenized with EMS (ethyl methanesulfonate) as described (Brenner 1974). F1 progeny were allowed to grow to young adults in NGM plates, and were transferred to plates containing 5 mM NaI. After eight days, F2 progeny were examined under a dissecting microscope for surviving adult animals. In Screen 1, we obtained 12 independent F2 isolates from ~5000 F1 (~10000 haploid genomes) animals. In Screen 2, we obtained 23 isolates from ~75,000 F1 animals.

For the F1 screen, EMS-mutagenized P0 animals at the L4 stage were grown on regular NGM plates overnight and then transferred to plates with 5 mM NaI. After 6 to 8 days, surviving F1 adult progeny were picked under a dissecting microscope and retested in 5 mM NaI. We found that one P0 adult could generate approximately 100 to 150 F1 progeny under the screening condition. We screened the F1 progeny of ~10,000 P0 animals (~100 P0 per 10 cm plate), the total number of which was estimated to be ~1000,000.

We mapped the mutations to chromosomes using the single nucleotide polymorphisms (SNP) method (Wicks *et al.* 2001) and previously described SNPs (Davis *et al.* 2005). Mutations mapped on the same chromosome were tested by genetic complementation for the survival of *trans* heterozygous animals in 5 mM NaI. We previously reported the cloning of *bli-3* and *tsp-15* from Screen 1. For the rest of mutations from Screen 1, *mac32* was mapped to a 0.2 mapping unit region on Chr. I, to the right of SNP *WBVar00154404* (SNP D2030: 16772 S = AG, genetic location: 2.17; GenBank accession no. Z73906) and the left of SNP *WBVar00154618* (SNP D2005: 31734 S = AG, genetic location: 2.37; GenBank accession no. Z79752). *mac39* was mapped to a 0.4 mapping unit region on Chr. IV, to the right of SNP *WBVar00189035* (SNP B0273: 29164 S=AG, genetic location: 1.79; GenBank accession no. FO080145) and the left of SNP *WBVar00189183* (SNP C31H1: 4110 S=CT, genetic location: 2.29; GenBank accession no. FO080496).

Mutations isolated from Screen 2 were first examined for recessive or dominant effects on the survival. Recessive mutants were tested in complementation tests with *bli-3(lf)*, *tsp-15(lf)* and *wdr-23(lf)* mutants isolated in Screen 1. Dominant mutants were examined by sequencing for *skn-1* mutations. Mutations in the *bli-3*, *tsp-15* and *wdr-23* complementation groups were determined by sequencing target gene exons and splice sites.

*doxa-1* mutations (*mac55, mac56, mac67*) were initially thought to belong to the *bli-3* group based on complementation tests. However sequencing failed to identify nucleotide changes on *bli-3* exons or splice sites. We suspect that a gene interacting with *bli-3* might be affected by these mutations and the non-complementation between these mutations and *bli-3(lf)* might result from compound haploid insufficiency in both genes. We determined the sequence of *doxa-1* in these mutants and identified a distinct mutation in each of them.

**Plasmids**

The 5.7 kb *EcoR*I genomic fragment that rescues *skn-1* mutants encodes a 533 amino-acid SKN-1 protein (SKN-1 isoform c) (Bowerman *et al.* 1992). The upstream fragment of the ATG start codon was used as *skn-1c* endogenous promoter. To construct the *skn-1cp::skn-1c cDNA::3’UTR* plasmid, the full-length *skn-1c* cDNA, the *skn-1c* promoter (0.9 kb upstream of the *skn-1c* start codon) and a 3’ UTR fragment (0.7 kb downstream of the *skn-1* stop codon) were amplified by PCR and subcloned to the pPD95\_79 vector (kindly provided by Andrew Fire) using In-fusion HD Cloning Kit (Clontech) at *Hind*III and *BamH*I sites following the instructions of the manufacturer.

To construct the *dpy-7p::skn-1c* *cDNA::3’UTR* plasmid, a *dpy-7* promoter (430 bp upstream of the *dpy-7* start codon) (Gilleard *et al.* 1997) was amplified by PCR and subcloned to the pPD95\_79 vector using *Hind*III and *BamH*I sites. The *skn-1c* DNA and 3’ UTR fragment were subcloned to the pPD95\_79-*dpy-7p* backbone using *Xma*I and *Kpn*I sites.

To construct the *nhx-2p::skn-1c* *cDNA::3’UTR* plasmid, an *nhx-2* promoter (4.1 kb upstream of the *nhx-2* start codon) (Nehrke 2003) was amplified by PCR and subcloned to pPD95\_79 using *Xba*I and *Xma*I sites. The *skn-1c* DNA and 3’ UTR fragment were subcloned to the pPD95\_79-*nhx-2p* backbone using *Xma*I and *Kpn*I sites. To construct the the *ges-1p::skn-1c* *cDNA::3’UTR* plasmid, a 3.3 kb *ges-1* promoter (Egan *et al.* 1995) was amplified using PCR and used to replace the *nhx-2* promoter using *Xba*I and *Xma*I sites.

To construct the *myo-3p::skn-1c* *cDNA::3’UTR* plasmid, a 2.3 kb *myo-3* promoter (Okkema *et al.* 1993) was released from pPD93\_97 using *Hind*III and *BamH*I sites and subcloned to pPD95\_79-*dpy-7p::skn-1c* *cDNA::3’UTR* plasmid to replace the *dpy-7* promoter.

The *skn-1ap::skn-1a::3’UTR* transgene was constructed by two steps. First, the full-length *skn-1a* cDNA, a 5’ UTR fragment (955 bp upstream of the *skn-1a* start codon) and a 3’ UTR fragment (0.7 kb downstream of the *skn-1* stop codon) were amplified by PCR and subcloned to the pPD95\_79 vector with In-fusion HD Cloning Kit (Clontech) using *Xma*I and *Kpn*I sites. Second, a 6.3 kb fragment (3078 bp *bec-1* gDNA and a 3.2 kb fragment upstream of the *bec-1* start codon) was amplified and subcloned using *Hind*III and *Xma*I sites to generate the *skn-1ap::skn-1a* *cDNA::3’UTR* construct.

To generate the *skn-1cp::skn-1a* *cDNA::3’UTR* construct, the full-length *skn-1a* cDNA and the 0.7 kb 3’ UTR fragment were subcloned to the pPD95\_79 vector using *Xma*I and *Kpn*I sites. Next, the *skn-1c* promoter was subcloned to pPD95\_79-*skn-1a cDNA::3’UTR* using *Hind*III and *BamH*I sites.

The *mac53* mutation was introduced to the *skn-1* transgenic constructs using the Q5 Site-Directed Mutagenesis Kit (New England Biolabs) respectively.

To generate the *tsp-15* rescue construct, the 777 bp *tsp-15* cDNA and a 3’UTR fragment (0.9 kb downstream of the *tsp-15* stop codon) were amplified by PCR and subcloned to the pPD95\_79 vector using *BamH*I and *BstB*I sites with the Gibson Assembly Master Mix (NEB). The 3.6 kb *tsp-15* promoter or the 430 bp *dpy-7* promoter was amplified and subcloned to pPD95\_79-*tsp-15 cDNA::3’UTR* using *Hind*III and *BamH*I sites.

To generate the *doxa-1* rescue construct, the full-length *doxa-1* cDNA and a 3’UTR fragment (0.9 kb downstream of the *doxa-1* stop codon) were amplified and subcloned to the pPD95\_79 vector using *BamH*I and *Age*I sites with the Gibson Assembly Master Mix (NEB). The 3.1 kb *doxa-1* promoter was amplified and subcloned to pPD95\_79-*doxa-1 cDNA::3’UTR* using *Hind*III and *BamH*I sites.

To generate the *wdr-23* rescue construct, the full-length 1716 bp *wdr-23* isoform a cDNA or the 1593 bp *wdr-23* isoform b cDNA and a 3’UTR fragment (0.4 kb downstream of the *wdr-23* stop codon) were amplified and subcloned to the pPD95\_79 vector using *BamH*I and *Age*I sites with the Gibson Assembly Master Mix (NEB) respectively. A *wdr-23a* promoter (2.1 kb upstream of the *wdr-23a* start codon) was amplified and subcloned to the pPD95\_79-*wdr-23a cDNA::3’UTR* using *Hind*III and *BamH*I sites. A *wdr-23b* promoter (2.8 kb upstream of the *wdr-23b* start codon), which contains the first exon of isoform a, was amplified and subcloned to the pPD95\_79-*wdr-23b cDNA::3’UTR* using *Hind*III and *BamH*I sites, in which the start codon of isoform a was mutated to ‘GTG’.

All plasmids were verified by restriction digestions and sequencing.

**Transgene experiments**

Germline transgene experiments were performed as described (Mello *et al.* 1991).

For *skn-1* phenocopy experiments, the transgenic mixtures normally contained 2.5 ng/μl pCFJ90 (*myo-2p::mCherry*) plasmid (Frokjaer-Jensen *et al.* 2008) as co-injection marker and 20 ng/μl of the transgene of interest, except for the *skn-1ap::skn-1a::3’UTR* transgene, the concentration of which was 30 ng/μl.

For survival assay, wild-type P0 adults injected with transgenes were picked to NGM plates containing 5 mM NaI and mCherry-positive F1 larval progeny were scored on day 4 and mCherry-positive adults were counted on day 8.

To examine the effects of transgenes on *gst-4p::GFP* expression, F1 progeny of *dvIs19* P0 animals injected with transgenes were picked to new plates and the pictures of adults were taken using a digital camera attached to an Olympus SZX16 dissecting microscope immediately after treatment with NaN3.

For *tsp-15*, *doxa-1* and *wdr-23* rescue experiments, the transgenic mixtures contained 50 ng/μl (*tsp-15* or *doxa-1*) or 100 ng/μl (*wdr-23*) of the transgenes of interest, with 20 ng/μl pPD95\_86 (*myo-3p::GFP*) as co-injection marker. The corresponding transgene mixture was injected to *tsp-15(mac33lf)*, *doxa-1(mac55lf)* or *wdr-23(mac32lf)* adults. For survival test of stable transgenic lines, 10 transgenic adults were allowed to lay eggs on regular or 5 mM NaI NGM plates for 24 hrs and removed. The number of transgenic F1 was counted 24 hrs later and the adults were counted in the following days.

**Transcriptome analyses**

Synchronized animals at the L1 larval stage were allowed to recover on OP50-seeded NGM agar plates with or without 5 mM NaI for 8 hrs and subsequently washed three times with H2O. RNA was extracted using TRIzol (Invitrogen) and chloroform-isopropanol purification and treated with DNase I (NEB). RNA-Seq was performed by Annoroad Gene Technology Corporation (Beijing).

DESeq2 v1.6.3 was used for differential gene expression analysis between two samples with biological replicates under the theoretical basis obeying the hypothesis of negative binomial distribution for the value of count. DESeq2 estimates the expression level of each gene in each sample by the linear regression, then calculates the *p*-value with the Wald test. *p*-value was corrected by the BH procedure. Genes with *q* ≤ 0.05 and |log2\_ratio| ≥ 1 are identified as differentially expressed genes (DEGs).

According to the expression level of differentially expressed genes in each sample, the Euclidean distance was calculated with |log2\_FPKM|, and the Hierarchical Cluster method was used to generated the heatmap.

The GO (Gene Ontology, http://geneontology.org/) enrichment of DEGs was implemented by the hypergeometric test, in which *p*-value is calculated and adjusted by FDR (False Discovery Rate) as *q*-value, and data background is gene expression of the whole genome. GO terms with *q* < 0.05 were considered to be significantly enriched.

KEGG (Kyoto Encyclopedia of Genes and Genomes, http://www.kegg.jp/) is a database resource containing a collection of manually drawn pathway maps representing the molecular interaction and reaction networks. The KEGG enrichment of DEGs was implemented by the hypergeometric test, in which *p*-value was adjusted by Benjamini and Hochberg multiple comparisons as *q*-value. KEGG terms with *q* < 0.05 were considered to be significantly enriched.

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