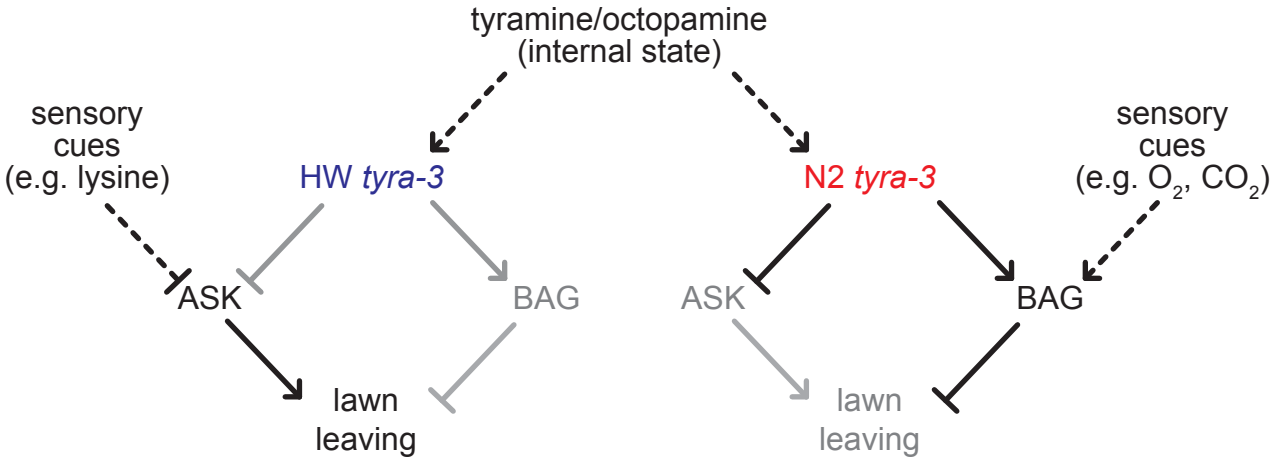
Supplementary Information

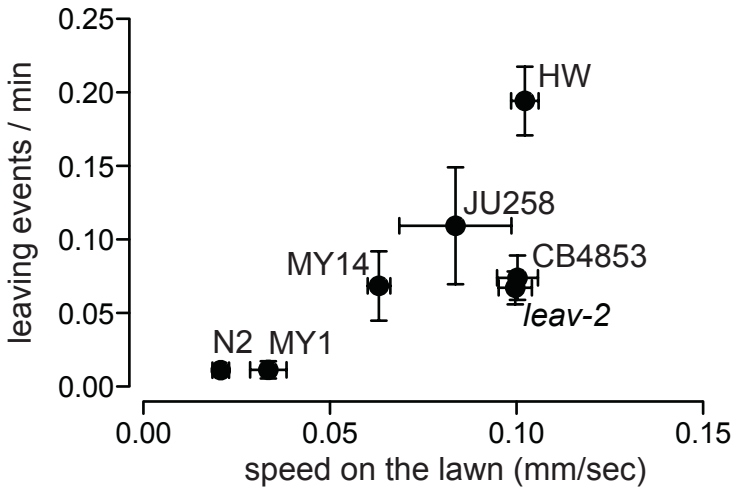
This file contains Legends to two Supplementary Movies, ten Supplementary Figures with their Legends, one Supplementary Table, Supplementary Methods, a list of Strains, and Supplementary References.

Supplementary Movie 1. Leaving assay showing six N2 adults, 20x actual speed. The border of the bacterial lawn is outlined for ease of visualization. Time stamp at upper left displays the actual time after placing the animals on the assay plate.

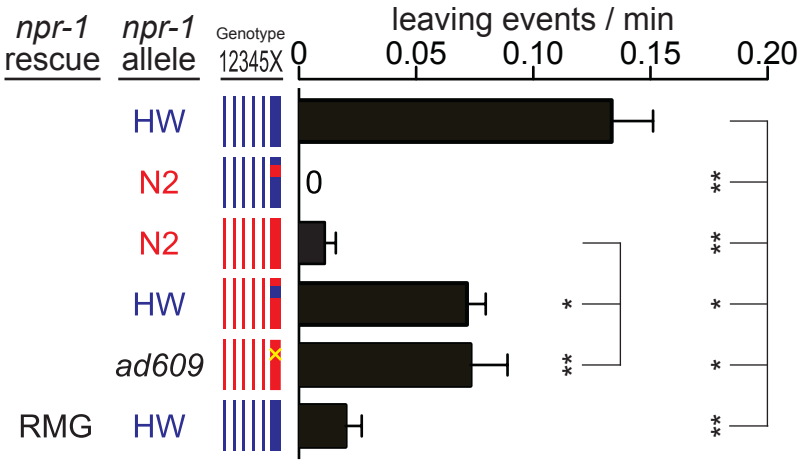
Supplementary Movie 2. Leaving assay showing six HW adults, 20x actual speed. Four animals are on the lawn and two off the lawn at the beginning of the assay. The border of the bacterial lawn is outlined for ease of visualization. Time stamp at upper left displays the actual time after placing the animals on the assay plate.



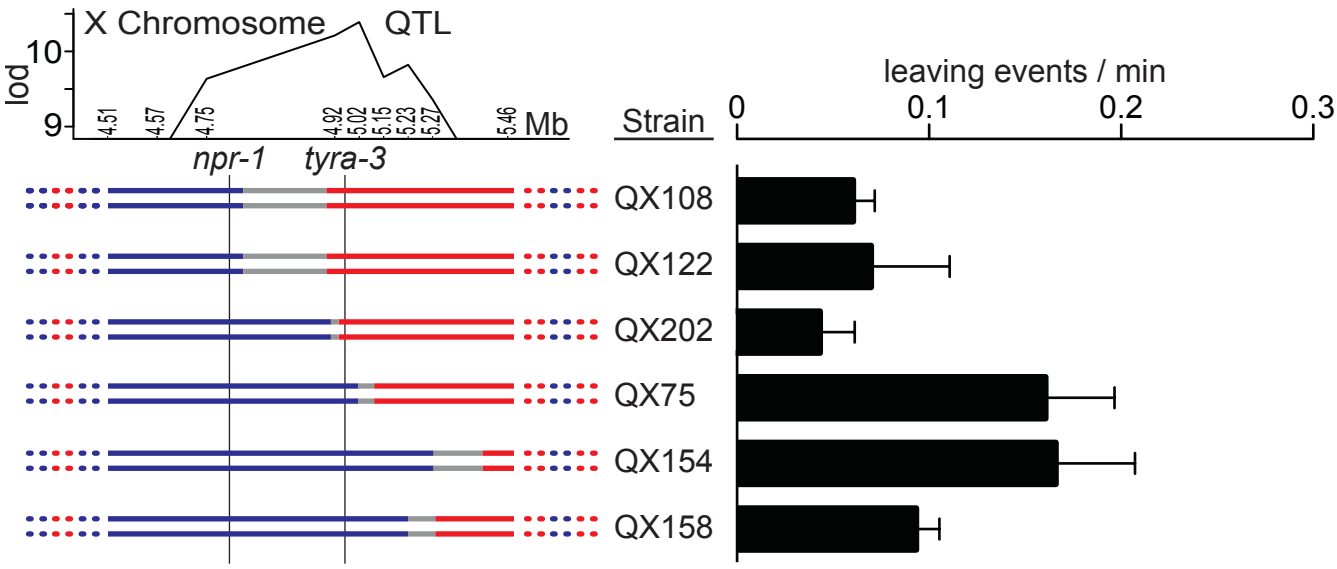
Suppl. Fig. 1. *tyra-3* polymorphism affects an exploration-exploitation decision. The *tyra-3* gene senses catecholamines and modifies the function of ASK and BAG chemosensory neurons, which promote or inhibit lawn-leaving, respectively. The N2 high-activity allele of *tyra-3* suppresses the function of ASK neurons and enhances the function of BAG neurons compared to the HW low-activity allele of *tyra-3*. Both functions of the N2 *tyra-3* allele suppress lawn-leaving. Solid lines indicate activities identified in this study; dashed lines indicate results from prior studies.



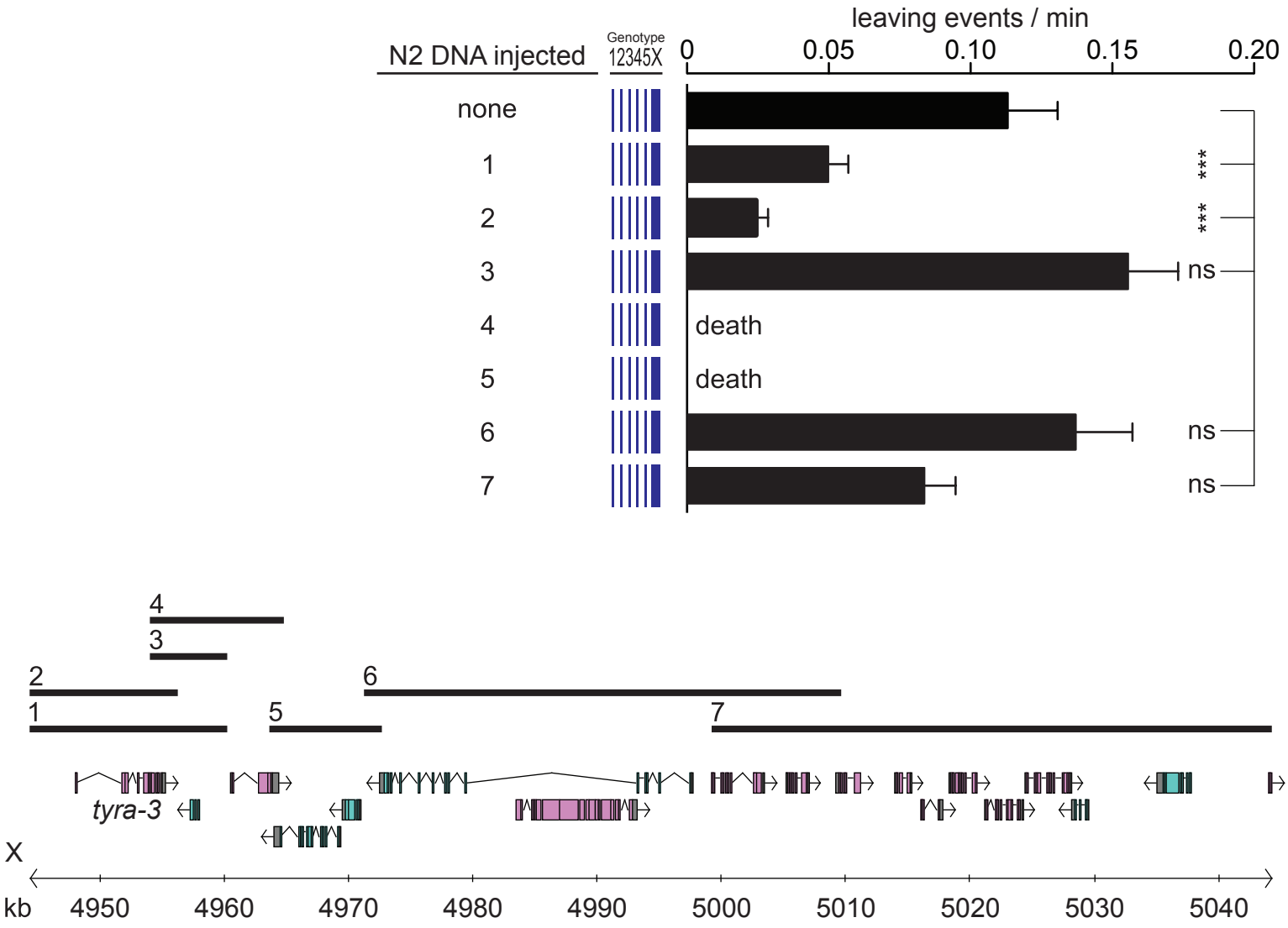
Suppl. Fig. 2. Locomotion speed on a food patch correlates with leaving rate. Correlation between locomotion speed on a small bacterial lawn and leaving rate in wild-type strains examined in Fig. 1. The *leav-2* strain (Fig. 2a), which has an N2 allele of *tyra-3* in HW background, is shown for comparison.



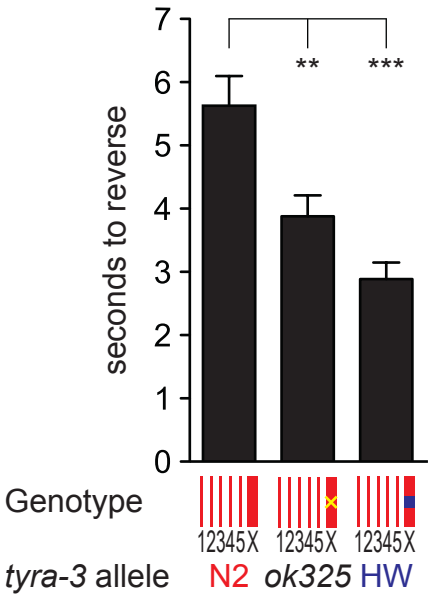
Suppl. Fig. 3. *npr-1* affects leaving behaviour.
Near-isogenic lines replace *npr-1* in each strain with the other strain's allele. The N2 allele of *npr-1* has high activity compared to the HW allele; *npr-1 (ad609)* is an EMS-induced loss-of-function allele of *npr-1*. Transgenic RMG expression of N2 *npr-1* in HW animals was achieved using an intersectional Cre-Lox strategy with two transgenes [1]. Error bars indicate s.e.m. * $P < 0.05$, ** $P < 0.01$ by ANOVA with Dunnett test to correct for multiple comparisons.



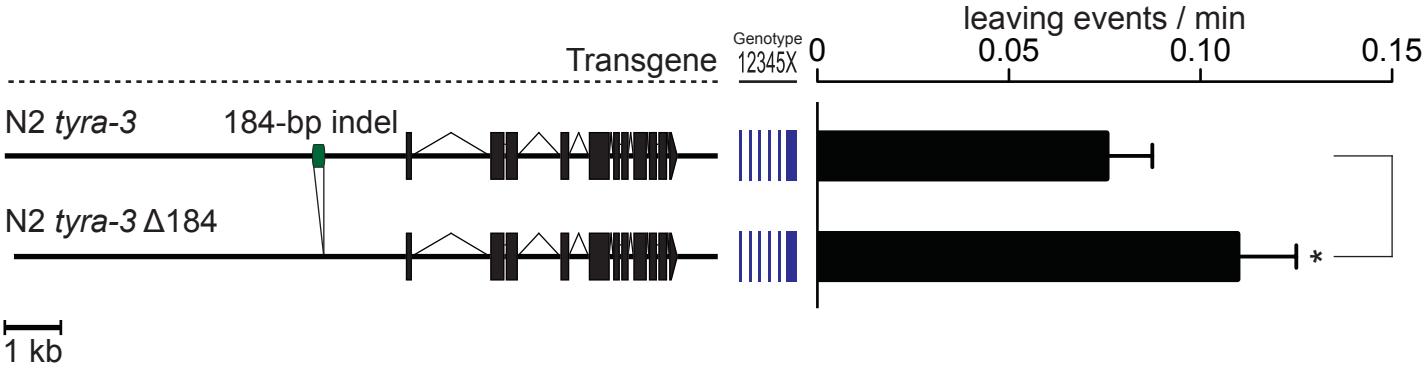
Suppl. Fig. 4. Phenotype-genotype correlations in RIAILs.
Leaving behaviour of recombinant inbred advanced intercross lines (RIAILs) with HW *npr-1* and a breakpoint to the left of 5.75 Mb; these six RIAILs were used to define a potential location for the QTL in *leav-2* strain. Blue denotes HW DNA, red denotes N2 DNA, and grey denotes breakpoints. QX108, QX122, and QX202 have lower leaving rates than QX75, QX154, and QX158. The QX158 strain appears to have a lower leaving rate than QX75 and QX154, but after backcrossing the X chromosome into the HW strain its leaving rates were indistinguishable from HW.



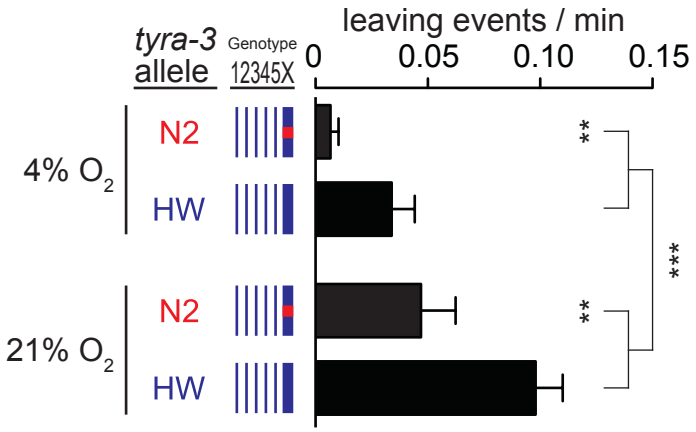
Suppl. Fig. 5. *tyra-3* is the gene affected by the *leav-2* QTL.
Leaving rates of transgenic HW animals injected with N2 DNA covering segments of the inferred position of the *leav-2* QTL. Three independent transgenic lines were tested for each segment; two segments caused lethality upon injection and could not be scored. Error bars indicate s.e.m. *** P < 0.001 by ANOVA with Dunnett test to correct for multiple comparisons.



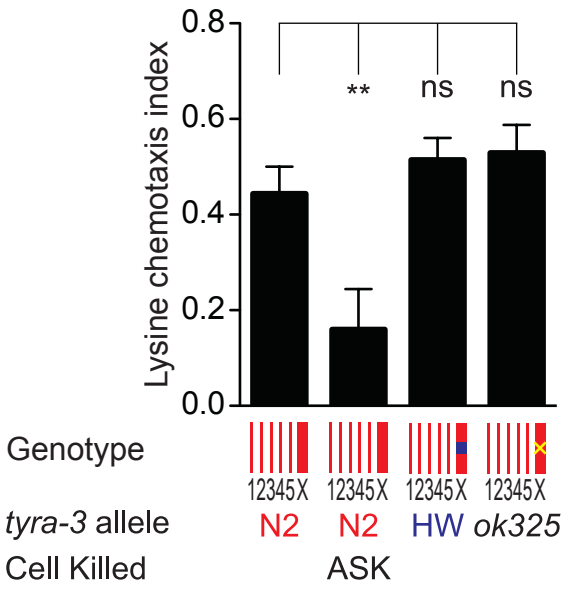
Suppl. Fig. 6. The HW allele of *tyra-3* has reduced activity in an avoidance assay. Animals were scored for avoidance of a point source of 30% octanol, off food after 40 minutes of starvation; rapid onset of reversals indicate a stronger response. Animals with a *tyra-3(ok325)* null allele or a HW *tyra-3* allele responded more rapidly than N2 animals. Error bars indicate s.e.m. ** $P < 0.01$, *** $P < 0.001$ by ANOVA with Dunnett test to correct for multiple comparisons.



Suppl. Fig. 7. The 184 bp *tyra-3* noncoding indel affects the leaving rate.
Leaving rates in HW animals with an N2 *tyra-3* transgene or a similar transgene bearing an engineered 184 bp deletion. To control for variation between different transgenes, five independent transgenic lines per transgene were tested and combined. Error bars indicate s.e.m. * $P < 0.05$ by t-test.

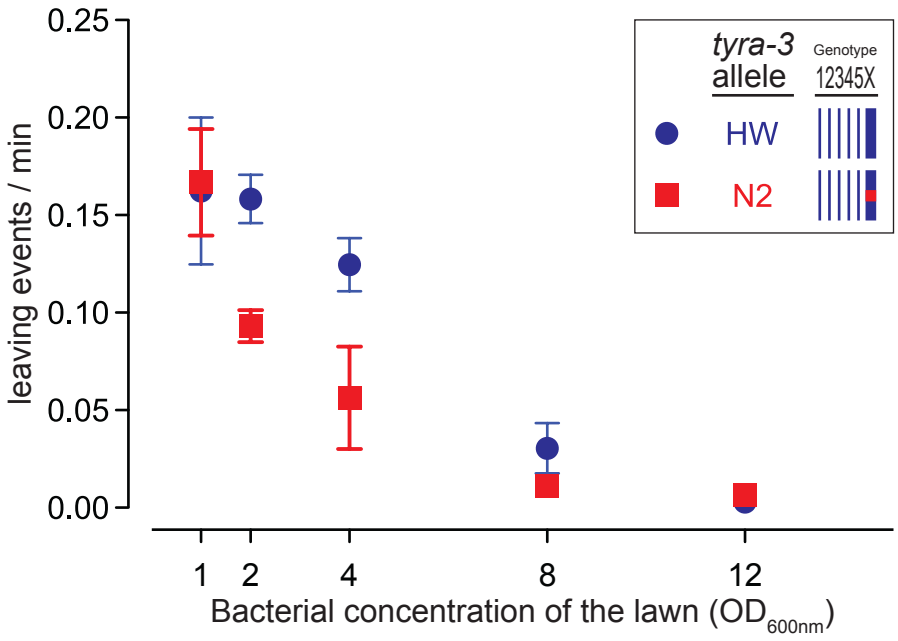


Suppl. Fig. 8. Lowering O_2 levels reduces leaving rates.
HW animals with either N2 or HW alleles of *tyra-3* have lower leaving rates at 4% O_2 than at 21% O_2. Assays were conducted in a flow chamber with oxygen concentrations controlled by external tanks of mixed gases. Two-way ANOVA showed significant effects of both the *tyra-3* genotype and the O_2 concentration, with no significant interaction between them. Error bars indicate s.e.m.
** $P < 0.01$, *** $P < 0.001$ by two-way ANOVA.

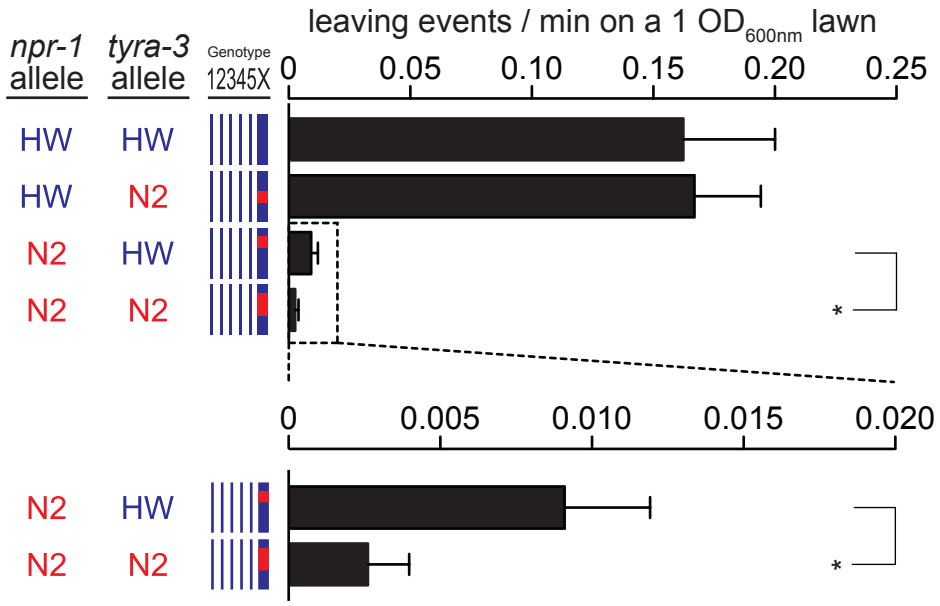


Suppl. Fig. 9. *tyra-3* does not affect lysine chemotaxis.
Killing ASK reduces chemotaxis to 5 mM L-lysine, but a *tyra-3(ok325)* null allele or a HW *tyra-3* allele did not affect the behaviour. Chemotaxis was scored by adding lysine to two quadrants of agar on a small plate, placing washed animals in the center of the plate, and examining their distribution after 5 minutes; chemotaxis index = [(animals on lysine)-(animals not on lysine)]/(total number of animals). Error bars indicate s.e.m. ** $P < 0.01$ by ANOVA with Dunnett test to correct for multiple comparisons.

a



b



Suppl. Fig. 10. Density of the bacterial lawn affects leaving rates and genetic interactions between *npr-1* and *tyra-3*.
a) Leaving rates of both N2 *tyra-3* and HW *tyra-3* strains (HW *npr-1* background) are higher on thinner lawns. b) N2 *tyra-3* further decreases the leaving rate of N2 *npr-1* on OD_{600nm}=1 lawns, but not on OD_{600nm}=2 lawns (Fig. 2a). Error bars indicate s.e.m. * $P < 0.05$ by t-test.

Supplementary Table 1

Position on X Chr. (WS219)	Wormbase reference	N2 (Bristol)[1]	MY1	MY14	CB4853	JU258	CB4856 (HW)
4937008	T	T	T	C	C	C	C
4937279	G	G	G	C	C	C	C
4937525	A	A	A	C	C	C	C
4938557-4938560	4 bp[2]	Del	Del	Del	Del	Del	Del
4939032-4939033[3]	-	-	-	Ins A[4]	-	-	-
4940383	C	C	C	C	T	C	C
4940524-4940525	2 bp	2 bp	2 bp	2 bp	2 bp	Del	2 bp
4940538	A	A	A	Del	Del	A	Del
4940540	A	A	A	Del	Del	T	Del
4940740	T	T	T	C	C	C	C
4941601	A	A	A	G	G	G	G
4941668	A	A	A	A	A	C	A
4941684	A	A	A	T	T	T	T
4941752-4941753	-	-	-	Ins A	Ins A	Ins A	Ins A
4941946	A	A	A	A	A	Del	Del
4942122-4942141	20 bp	20 bp	20 bp	Del	Del	Del	Del
4942248	T	T	T	G	G	G	G
4942471	A	A	A	A	Del	A	Del
4942486	G	G	G	A	A	A	A
4942500-4942503	4 bp	4 bp	4 bp	Del	Del	4 bp	4 bp
4942565-4942573	9 bp	9 bp	9 bp	9 bp	Del	9 bp	9 bp
4942815	T	T	T	A	A	A	A
4942836	G	G	G	A	A	A	A
4943047	G	G	G	G	G	A	G
4943084	G	G	G	G	G	A	G
4943188	G	G	G	G	C	G	G
4943344	C	C	C	T	C	T	T
4944083	C	C	C	T	T	T	T
4944482	A	A	A	A	C	A	A
4944611	A	A	A	A	A	A	Del
4944629	G	G	G	A	G	G	G
4944776	A	A	A	T	T	T	T
4945282	T	T	T	G	T	T	T
4945772-4945773[5]	-	-	-	Ins A	Ins A	Ins A	Ins A
4946063	A	A	A	A	T	A	A
4946367	T	T	T	T	T	C	T
4947025	T	T	T	T	A	T	T
4947026	G	G	G	G	A	G	G
4947027-4947028	-	-	-	-	Ins (5 bp)	-	-
4947028	C	C	C	C	T	C	C
4947029	G	G	G	G	C	G	G
4948269	T	T	T	A	A	A	A
4948487	A	A	A	A	A	A	Del
4948578-4948579	-	-	-	-	Ins T	-	-

Supplementary Table 1, continued

Position on X Chr. (WS219)	Wormbase reference	N2 (Bristol)	MY1	MY14	CB4853	JU258	CB4856 (HW)
4948644	A	A	A	**G**	**G**	**G**	**G**
4948657	T	T	T	**C**	T	**C**	**C**
4948658-4948841	184 bp	184 bp	184 bp	**Del**	184 bp	**Del**	**Del**
4948784	C	C	C	C	**G**	C	C
4948801	T	T	T	T	**C**	T	T
4948804-4948805	-	-	-	-	**Ins T**	-	-
4948807-4948808	2 bp	2 bp	2 bp	2 bp	**Del**	2 bp	2 bp
4949069	C	C	C	**T**	**T**	**T**	**T**
4949663[(6)]	A	A	A	**G**	**G**	**G**	**G**
4950349[(7)]	A	A	A	**G**	**G**	**G**	**G**
4950685	T	T	T	**C**	**C**	**C**	**C**
4951209	A	A	A	**G**	A	**G**	**G**
4951596	G	G	G	**A**	**A**	**A**	**A**
4952048	T	T	T	**T**	**T**	**T**	**T**
4952532	T	T	T	T	**C**	T	T
4952780	A	A	A	A	**G**	A	A
4955677	G	G	G	**T**	G	**T**	**T**

Alleles in **gray** are identical to the Wormbase N2 reference genome.
Alleles in **black** are different from the Wormbase N2 reference genome.
(1) N2 was resequenced and compared to the N2 Wormbase reference.
(2) These 4 bp are deleted in the resequenced N2 and all 'wild type' strains, indicating a discrepancy with the Wormbase reference.

(3) Insertions (Ins) lie between the two positions.

(4) The nucleotide of single-bp insertions is specified. Longer insertions are referred to by their length, indicated in parentheses.

(5-6) The alleles between this positions (including those marked by [5] and [6]) are covered by the 4.9 kb promoter used in this study.
(7) Non-synonymous substitution. 'A' allele codes for glutamate and 'G' allele codes for glycine.

Supplementary Methods

Nematode Growth

Strains were grown and maintained under standard conditions at 22-23 ˚C on nematode growth medium (NGM) 2% agar plates [2]. All animals used for behavioural assays were grown on plates seeded with dense *E. coli* HB101 lawns.

Analysis of Behaviour in the Leaving Assay

6 cm NGM agar plates were seeded with 70 mL (conditioning plate) or with 10 mL (assay plate) of a fresh overnight culture of *E. coli* HB101 diluted in LB to OD_{600nm}=2.0. 90 min after seeding the plates, ten young adult hermaphrodites were picked onto the conditioning plate. This step allowed animals to acclimate to the low-density lawn used for test conditions; omitting the conditioning step resulted in significantly higher leaving rates. 30 min after being placed on the conditioning plates, seven of the animals were transferred onto the lawn of the assay plate. The handling during transfer increased leaving rates, consistent with an escape response induced by mechanical stimuli [3], and some animals were lost in the first hour on the assay plate by crawling up the sides of the plate. Leaving rates decreased with time on the assay plate and were stable after 1 hr, so the 30 min leaving assay began 1 hr after placing the seven animals on the assay plate. The combined time the seven animals spent inside the lawn during the 30 min assay was 171 min for N2 and 101 min for HW animals, on average. In agreement with previous studies [4], leaving behaviour in both N2 and HW was

influenced by bacterial density (Supplementary Fig. 10a and data not shown). For movies of N2 and HW lawn-leaving behaviour see Supplementary Movies 1 and 2.

QTL Analysis

The initial RIAIL analysis identified a QTL on chromosome II in addition to the QTL on X (Fig. 1d); an alternate analysis of the RIAIL leaving data revealed a potential QTL with genome-wide significance on chromosome IV. As neither HW chromosome II nor chromosome IV, nor both together, could reconstitute the full HW behaviour when combined with HW *npr-1* and HW *tyra-3*, the autosomal QTLs were not pursued further.

Identification of the Minimal Genetic Region for the *leav-2* QTL

The genetic region from 4.78-5.75 MB defined by the *leav-2* strain in Fig. 2 encompassed 158 genes. The location of the potential leaving-suppressing variant was inferred from the analysis of breakpoints within the QTL in individual RIAILs, as follows. The 1.5-lod score confidence interval defined by the RIAILs spans from ~4.6 to 5.3 Mb (Supplementary Fig. 4). Thus, the causal variant in *leav-2* was hypothesized to lie between 4.78 and 5.3 Mb, a region containing only 85 genes. Six RIAILs that had HW *npr-1* had a breakpoint to the left of 5.75 Mb. Three of the strains (QX108, QX122, and QX202) had N2 sequence to the right of 4.93 Mb and low leaving rates (Supplementary Fig. 4). The other three

(QX75, QX154, QX158) had N2 sequence to the right of 5.03 Mb and higher leaving rates. These observations suggested that the N2 allele that suppresses leaving may lie between 4.93 and 5.03 Mb, a region that contains 17 genes (Supplementary Fig. 5).

Identification of *tyra-3* as the Gene Affected by the *leav-2* QTL

PCR products that overlap by at least 1 kb, covering the region from 4.93 to 5.03 Mb on the X chromosome, were amplified from N2 genomic DNA using the following primers and injected into HW animals (at 5 ng/μL) in groups:

Product a (*tyra-3*). Forward cctgctcttttctggaggtg, reverse gccgcaaaaacagagaaaac

Product b. Forward ttttccttttttagatctccatgtc, reverse tgaaggaaccgtattttccaa

Product c. Forward ttttccttttttagatctccatgtc, reverse aaagcggatcaagaattcca

Product d. Forward ccaccatgtacccaggaatc, reverse ccttcctcgagtcaagttgc

Product e. Forward agaacaaccccgagacacac, reverse tggagttttccaccgatttc

Product f. Forward ccaatcacctgccctttcta, reverse tgtggacgatgagttggtgt

Product g. Forward cgactcaaaggtgcaagaca, reverse gaagttcggctgaaaagcac

Product h. Forward aacctttcagccaccgtatg, reverse acgcgttcaagcacttttct

Product i. Forward gcaatttccatcctcatcgt, reverse ttcaacttccagtcggaacc

Product j. Forward gtgctcacaaaatcgcagaa, reverse gctcgagacattttcgaagg

Product k. Forward cgacaatgatggacacaagg, reverse agaagccgaagaaggaggac

Product l. Forward aacaaaattggctcgtgacc, reverse aacttttgttcccggatgtg

DNA pools tested by injection were pool 1=a+b; 2,=a; 3,=b; 4=c; 5=d; 6=e+f+g+h; 7=h+i+j+k+l. Three transgenic lines were tested per DNA pool.

Generation of Near-Isogenic Lines

Near-isogenic lines were created by backcrossing a chromosomal region or allele into the desired genetic background as described below. Desired segments were then inbred to homozygosity. For introgressions into the HW (CB4856) background, crosses were set up to avoid problems with the incompatibility locus between N2 and HW on chromosome I [5].

Marker positions are based on Wormbase release WS219; *npr-1* is at 4,769,595 (indel) and *tyra-3* is at 4,948,658 (indel).

QX1092 *npr-1 (CB4856>N2) [qqIR3]* X: QX202, a RIAIL containing CB4856 *npr-1* and N2 *tyra-3*, was crossed to *lon-2* males (in an N2 background) for 10 generations, picking non-Lon hermaphrodites each generation (*lon-2* is tightly linked to *npr-1).* The introgression breakpoints are, on the left, between 3,921,083 (marker haw101674) and 4,060,839 (marker pkP6146), and on the right, between 4,892,213 (marker pkP6106) and 4,937,279 (marker haw102792). There is additional CB4856 sequence with a left breakpoint between 6,073,091 (marker haw103987) and 6,278,584 (indel) and a right breakpoint between 6,278,584 (indel) and 6,581,237 (marker pkP6154).

CX11400 *leav-1 (N2>CB4856) [kyIR9]* X: CB4856 males were crossed to QX9, a RIAIL containing N2 *npr-1*, and a recombinant F2 between *npr-1* and an indel

marker at 4,948,658 was selected. This recombinant bearing N2 *npr-1* and CB4856 sequence to its right was backcrossed to CB4856 males nine more times, selecting hermaphrodites with an N2 allele of *npr-1* each generation. The introgression breakpoints are, on the left, between 4,649,200 (marker uCE6-872) and 4,745,912 (marker snp_C39E6[1]), and on the right, between 4,768,758 (marker snp_C39E6[4]) and 4,797,631 (marker uCE6-877).

CX10927 *leav-2 (N2>CB4856) [kyIR2]* X: QX122, a RIAIL containing CB4856 *npr-1* and N2 sequence to its right, was crossed to CB4856 males for 10 generations, selecting hermaphrodites with an N2 allele at 4,948,658 (indel) each generation. The introgression breakpoints are, on the left, between 4,769,595 (indel) and 4,797,631 (marker uCE6-877), and on the right, between 5,744,794 (indel) and 5,759,074 (marker uCE6-952).

CX13272 *npr-1 tyra-3 (N2>CB4856) [kyIR91]* X: QX32, a RIAIL containing N2 *npr-1* and *tyra-3*, was crossed to CB4856 males. Male F1s were backcrossed to CB4856 hermaphrodites, F2s were selfed, and F3s that kept N2 *npr-1* and *tyra-3* were crossed to CB4856 males again. This cycle was repeated four more times. The introgression breakpoints are, on the left, between 4,060,839 (marker pkP6146) and 4,279,605 (marker uCE6-854), and on the right, between 5,153,187 (marker uCE6-890) and 5,234,763 (marker uCE6-904).

CX11950 *tyra-3 (ok325>CB4856) [kyIR25]* X: CB4856 males were crossed to CX11839 (*tyra-3* [*ok325*]) and a recombinant F2 between *npr-1* and *tyra-3 (ok325)* was identified. This recombinant bearing CB4856 *npr-1* and *tyra-3 (ok325)* was backcrossed to CB4856 males eight more times, selecting

hermaphrodites with an *ok325* allele of *tyra-3* each generation. The introgression breakpoints are, on the left, between 4,919,592 (marker haw102765) and 4,919,769 (marker haw102766), and on the right, between 5,360,624 (indel) and 5,414,461 (marker uCE6-929).

CX13271 *tyra-3 (CB4856>N2) [kyIR90]* X: QX75, a RIAIL containing CB4856 *npr-1* and *tyra-3,* was backcrossed repeatedly to N2-derived strains, while recombining *dpy-3* and *lon-2* markers on and off the X chromosome to eliminate linked sequences from CB4856. The introgression breakpoints are, on the left, between 4,866,708 (indel) and 4,919,592 (marker haw102765), and on the right, between 5,033,445 (marker uCE6-886) and 5,152,492 (marker uCE6-888).

QX1157 *npr-1 tyra-3 (CB4856>N2) [qqIR2]* X: QX125, a RIAIL containing CB4856 *npr-1* and *tyra-3*, was backcrossed to *lon-2* males (in an N2 background) for 20 generations, picking non-Lon hermaphrodites each generation. The introgression breakpoints are, on the left, between 4,507,511 (marker uCE6-865) and 4,637,513 (marker pkP6149), and on the right, between 10,265,260 (indel) and 11,142,289 (marker pkP6114).

tyra-3 Population Genetic Summary Statistics

Population genetic summary statistics for the gapless sites in the alignment of the *tyra-3* genomic region were calculated using *libseq* [6]. SNP variation (17644.3 non-coding and synonymous sites, $p = 10.53 \times 10^{-4}$, $Q_w = 9.99 \times 10^{-4}$) is typical for *C. elegans* genes [7,8]. The allele frequency spectrum is also consistent with

neutral equilibrium (Tajima's *D* = 0.368, p = 0.6). The SNP data provided no evidence for intragenic recombination (R_{min} = 0).

Transgenes

The *Pflp-21::LoxP stop LoxP::npr-1 SL2 GFP* and *Pncs-1::nCre* constructs are described in Macosko et al, 2009 [1].

The 11 kb *tyra-3* genomic transgene was amplified using Expand Long Range dNTPack (Roche) with primers cctgctcttttctggaggtg and gccgcaaaaacagagaaaac

Ptyra-3b was amplified using primers: tcaacctaaccactaactaaggg and cGatgaagcaagatgtcaggt, which overlaps the coding region by 4 bp. The ATG start codon is mutated to ATC (mutation is uppercase in primer). This product was cloned into pSM.

ADL Psri-51 ends: gactgtaaaatcgataagca…ccactgccaccgggcagaac

ASK Psra-9 ends: gcatgctatattccaccaaa...tgtgcatcaatcatagaaca

BAG Pflp-17 ends: ccttgaagcttttcctctga…gcaaaactttatttttccag

CEP, ADE, PDE Pdat-1 ends: atctctgaaatgtttctagt…aatctcaacaatttttagcc

tyra-3b cDNA was cloned by RT-PCR. The ends are atggctatttggtggtggtt… agcaatcgacaatattctaa. The product was cloned into pSM with NheI and KpnI.

Promoter-GFP fusions were performed as described [9], with the same promoter end sequences as in *Ptyra-3b*.

The 12.6 kb *tyra-3* genomic transgene was cloned into pSM. The ends are agttggtacaaaaagcttac…gttctcagggtgattgtgtt. The 184 bp deletion was engineered by site-directed mutagenesis.

Strains

'Wild-type' strains

Strain	Origin	Haplotype [10]
N2	Bristol, England	1
CB4856	Hawaii, USA	41
MY1	Lingen, Germany	29
MY14	Mecklenbeck, Germany	40
JU258	Madeira, Portugal	39
CB4853	Altadena, California, USA	19

N2-HW RIAILs

QX10, QX11, QX12, QX13, QX14, QX24, QX31, QX32, QX34, QX37, QX38, QX39, QX42, QX43, QX45, QX47, QX49, QX52, QX55, QX56, QX57, QX58, QX61, QX62, QX64, QX65, QX68, QX70, QX71, QX73, QX74, QX75, QX77, QX82, QX86, QX91, QX98, QX107, QX108, QX121, QX122, QX124, QX125, QX126, QX127, QX128, QX129, QX131, QX132, QX133, QX134, QX144, QX149, QX151, QX152, QX154, QX157, QX158, QX165, QX167, QX168, QX169, QX171, QX174, QX175, QX176, QX178, QX179, QX181, QX182,

QX185, QX187, QX190, QX191, QX192, QX193, QX195, QX196, QX198, QX199, QX200, QX202, QX205, QX208, QX209, QX210, QX212, QX213, QX221, QX223, QX233

Near-isogenic lines in a HW genetic background:

CX11400 *kyIR9 [leav-1 X:~4.70-~4.78Mb, N2>CB4856]*

CX10927 *kyIR2 [leav-2 X:~4.78-~5.75Mb, N2>CB4856]*

CX13272 *kyIR91 [X:~4.17-~5.19Mb, N2>CB4856]*

CX11950 *kyIR25 [tyra-3 (ok325) X:~4.92-~5.39Mb, CX11839>CB4856]*

Near-isogenic lines in an N2 background:

QX1092 *qqIR3 [X: ~3.99-~4.91Mb, CB4856>N2]*

CX13271 *kyIR90 [X:~4.89-~5.09Mb, CB4856>N2]*

QX1157 *qqIR2 [X: ~4.57-~10.70Mb, CB4856>N2]*

Transgenic strains

CX12787, CX12788 *kyEx3586, kyEx3589 [Pflp-21::LoxP stop LoxP::npr-1 SL2 GFP @50ng/mL, Pelt-2::mCherry @2ng/mL]*

CX12789-CX12790 *kyEx3590-kyEx3591 [Pncs-1::nCre @20ng/mL, Pofm-1::dsRed @10ng/mL]*

CX10576, CX10577, CX10595, CX10596 *kyEx2635, kyEx2636, kyEx2651, kyEx2652 [11kb tyra-3 HW-genomic fragment @5ng/mL; Pelt-2::GFP @4.5ng/mL]*

CX10457-CX10459 *kyEx2536-kyEx2538 [11kb N2-tyra-3 genomic fragment @5ng/mL; Pelt-2::GFP @4.5ng/mL]*

CX10619-CX10621 *kyEx2667-kyEx2669 [11kb HW-tyra-3 genomic fragment @1ng/mL; Pelt-2::GFP @4.5ng/mL]*

CX10622-CX10624 *kyEx2670-kyEx2672 [11kb N2-tyra-3 genomic fragment @1ng/mL; Pelt-2::GFP @4.5g/mL]*

CX11362, CX11365 *kyEx3025, kyEx3028 [HW-Ptyra-3b::HW-tyra-3b::SL2 GFP @5ng/mL]*

CX11367-CX11368 *kyEx3030-kyEx3031 [HW-Ptyra-3b::N2-tyra-3b::SL2 GFP @5ng/mL]*

CX11363-CX11364 *kyEx3026-kyEx3027 [N2-Ptyra-3b::N2-tyra-3b::SL2 GFP @5ng/mL]*

CX11366, CX11369 *kyEx3029, kyEx3032 [N2-Ptyra-3b::HW-tyra-3b::SL2 GFP @5ng/mL]*

CX10790-CX10791 *kyEx2762-kyEx2763 [N2-Ptyra-3b::GFP @20ng/mL; Pelt-2::GFP @4.5ng/mL]*

CX10789, CX10792 *kyEx2761, kyEx2764 [HW-Ptyra-3b::GFP @20ng/µL; Pelt-2::GFP @4.5ng/mL]*

CX13452-CX13456 *kyEx4030-kyEx4034 [12.6kb N2-tyra-3 genomic fragment @5ng/mL; Pelt-2::GFP @4.5g/mL]*

CX13447-CX13451 *kyEx4025-kyEx4034 [12.6kb N2-tyra-3 genomic fragment Δ184 @5ng/mL; Pelt-2::GFP @4.5g/mL]*

CX13112-CX13114 *kyEx3778-kyEx3780 [Psri-51::N2-tyra-3b::SL2 GFP @50ng/µL, Pelt-2::mCherry @2ng/µL]*

CX11495-CX11497 *kyEx3063-kyEx3065 [Psra-9::N2-tyra-3b::SL2 GFP @40ng/mL, Pelt-2::GFP @4.5ng/mL]*

CX13118-CX13120 *kyEx3784-kyEx3786 [Pflp-17::N2-tyra-3b::SL2 GFP @1ng/µL, Pelt-2::mCherry @2ng/µL]*

CX13115-CX13117 *kyEx3781-kyEx3783 [Pdat-1::N2-tyra-3b::SL2 GFP @25ng/µL, Pelt-2::mCherry @2ng/µL]*

QS4 *qrIs2 (Psra-9::mCaspase 1, Pelt-2::GFP)*

CX13355 *kyIR92 [kyIs536 (Pglb-5::p12 hCaspase 3::SL2 GFP, Pelt-2::mCherry), CX11697>CB4856]; kyIR93 [kyIs538 (Pflp-17::p17 hCaspase 3::SL2 GFP, Pelt-2::GFP), CX11697>CB4856]*

CX11674-CX11675 *kyIR14-kyIR15 [kySi47,kySi46 (Cbr-unc-119(+)::N2-Ptyra-3b::N2-tyra-3b::SL2 GFP) II, N2>CB4856]*

CX11673, CX11676 *kyIR13,kyIR16 [kySi41,kySi43(Cbr-unc-119(+)::HW-Ptyra-3b::N2-tyra-3b::SL2 GFP) II, N2>CB4856]*

Mutant strains

DA609 *npr-1 (ad609) X*

CX11839 *tyra-3 (ok325) X*, outcrossed 4X to N2

References

1 Macosko, E. Z. *et al.* A hub-and-spoke circuit drives pheromone attraction and social behaviour in C. elegans. *Nature* **458**, 1171-1175 (2009).

2 Brenner, S. The genetics of Caenorhabditis elegans. *Genetics.* **77**, 71-94 (1974).

3 Zhao, B., Khare, P., Feldman, L. & Dent, J. A. Reversal frequency in Caenorhabditis elegans represents an integrated response to the state of the animal and its environment. *J Neurosci* **23**, 5319-5328 (2003).

4 Harvey, S. C. Non-dauer larval dispersal in Caenorhabditis elegans. *J Exp Zool B Mol Dev Evol* **312B**, 224-230 (2009).

5 Seidel, H. S., Rockman, M. V. & Kruglyak, L. Widespread genetic incompatibility in C. elegans maintained by balancing selection. *Science* **319**, 589-594 (2008).

6 Thornton, K. Libsequence: a C++ class library for evolutionary genetic analysis. *Bioinformatics* **19**, 2325-2327 (2003).

7 Cutter, A. D. Nucleotide polymorphism and linkage disequilibrium in wild populations of the partial selfer *Caenorhabditis elegans*. *Genetics.* **172**, 171-184 (2006).

8 Dolgin, E. S., Felix, M. A. & Cutter, A. D. Hakuna Nematoda: genetic and phenotypic diversity in African isolates of Caenorhabditis elegans and C. briggsae. *Heredity* **100**, 304-315 (2008).

9 Hobert, O. PCR fusion-based approach to create reporter gene constructs for expression analysis in transgenic *C. elegans*. *Biotechnique* **32**, 728-730 (2002).

10 Rockman, M. V. & Kruglyak, L. Recombinational landscape and population genomics of Caenorhabditis elegans. *PLoS Genet* **5**, e1000419 (2009).