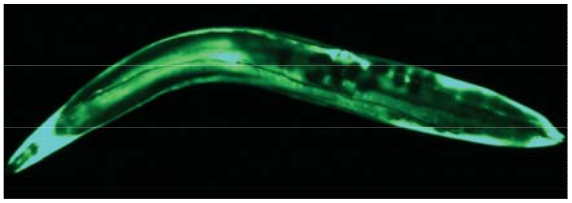
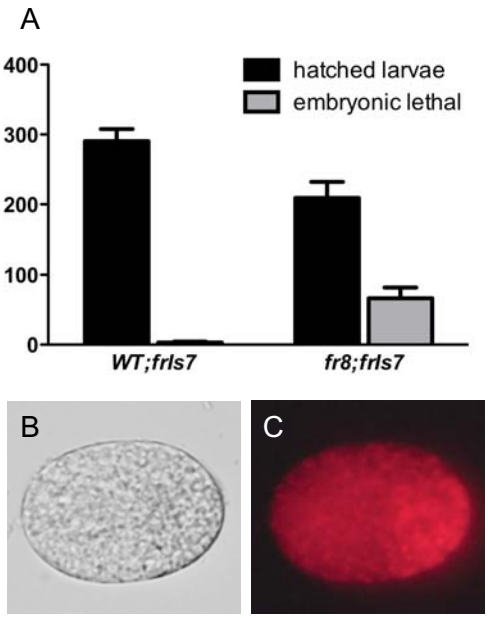
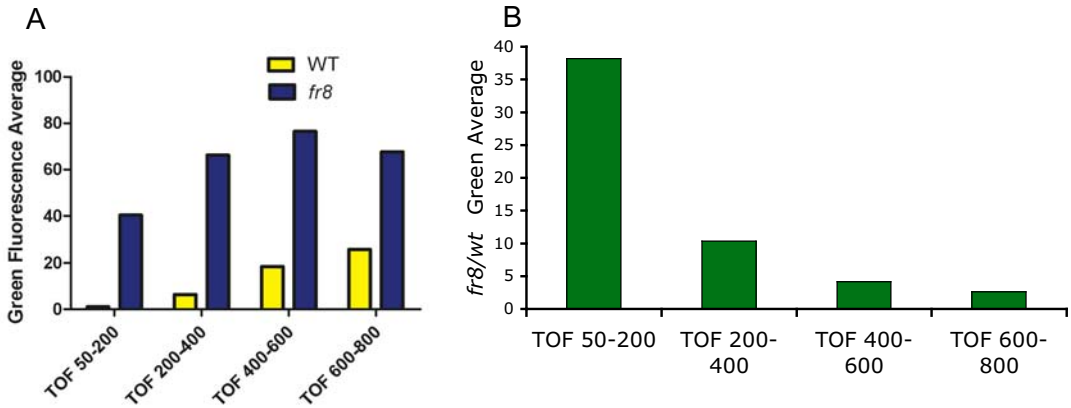


Supplementary Fig. 1. The *cnc-2* AMP gene is expressed constitutively in *fr8* mutant worms. Uninfected *fr8* worms carrying a p*cnc-2*::GFP transgenic reporter express high levels of GFP in the epidermis under normal culture conditions. In a wild-type background, the reporter gene is not expressed under the same conditions (Zugasti O, Ewbank JJ. Neuroimmune regulation of antimicrobial peptide expression by a noncanonical TGF-beta signaling pathway in *Caenorhabditis elegans* epidermis. Nature immunology 2009; 10:249-56).

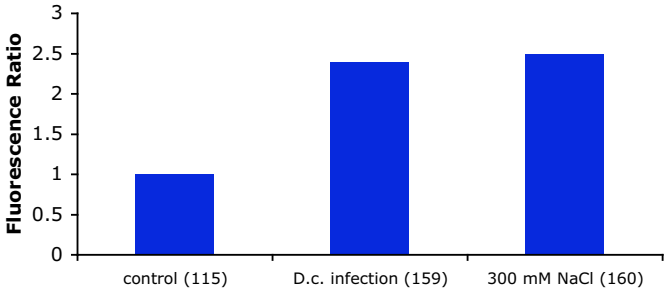


Supplementary Fig. 2. Embryonic arrest and osmosensivity in *fr8* mutants. (A) 60 wild-type and *fr8* mutant young adult worms (both containing the integrated *frls7* reporter transgene) were left to lay eggs for one hour at 20°C. The number of eggs were then counted, and 18 h later the number of hatched and un-hatched eggs counted. Whereas in wild-type almost 100 % of the laid eggs hatch, a quarter of *fr8* eggs fail to hatch. The error bars show standard deviations from three independent experiments. (B) Unhatched *fr8* embryo 16h after laying, (C) The same embryo shows uptake of Nile Red dye, unlike wild-type embryos which do not take up the dye. A modification of the protocol in Rappleye et al., was used, replacing Nile Red for Nile Blue.

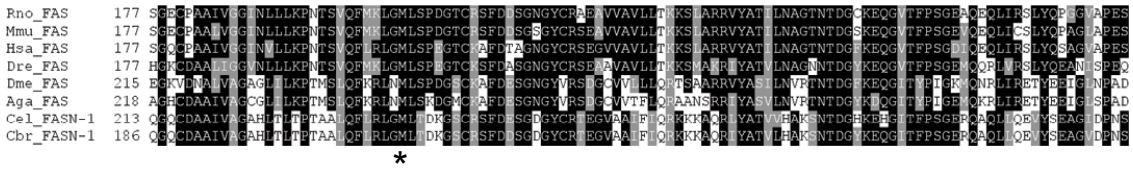
Rappleye CA, Tagawa A, Le Bot N, Ahringer J, Aroian RV. Involvement of fatty acid pathways and cortical interaction of the pronuclear complex in *Caenorhabditis elegans* embryonic polarity. BMC Dev Biol 2003; 3:8.



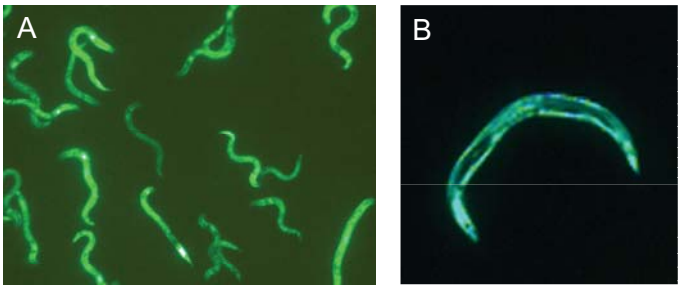
Supplementary Fig. 3. Quantitative fluorescence analysis of *fr8*. (A) The Time of Flight (TOF) is a measure of the length and therefore the age of the worms. Worms of different ages were placed in bins based on their TOF. The y axis denotes the average green fluorescence for the wild-type and *fr8* strains. (B) The ratio of *fr8/wt* for the green fluorescence average declines with age (measured for each TOF bin). The graph is an alternative representation of the data in (A). The data in (A) and (B) is that shown in Figure 1D.



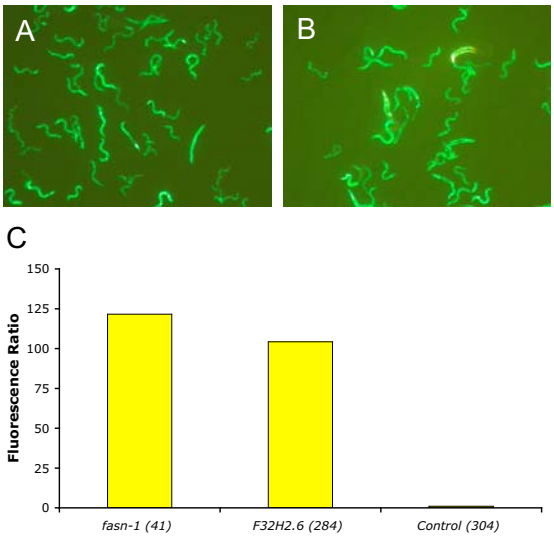
Supplementary Fig. 4. Fungal infection and osmotic shock induce p*nlp-29*::GFP in *fr8; frls7* worms. Increase of GFP expression in the *fr8* mutant background 24 h after fungal infection (D.c. infection) and 6 h of osmotic shock (300 mM NaCl). The numbers of worms analyzed is shown in parenthesis. The results shown are representative of two independent experiments.



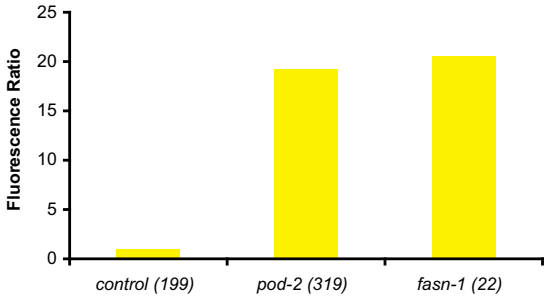
Supplementary Fig. 5. ClustalW alignment of FAS proteins. Part of the alignment is shown. The star under the conserved methionine corresponds to the site of the mutation in *fasn-1(fr8)*. Abbreviations (with accession numbers in brackets) are Rno: *Rattus norvegicus (*NP_059028.1); Mmu: *Mus musculus (*NP_032014.3); Hsa: *Homo sapiens* (NP_004095.4); Dre: *Danio rerio* (XP_687387.2); Dme: *Drosophila melanogaster* (NP_608748.1); Aga: *Anopheles gambie (*XP_319941.4); Cel: *Caenorhabditis elegans (*NP_492417.2); Cbr: *Caenorhabditis briggsae (*XP_001668410)



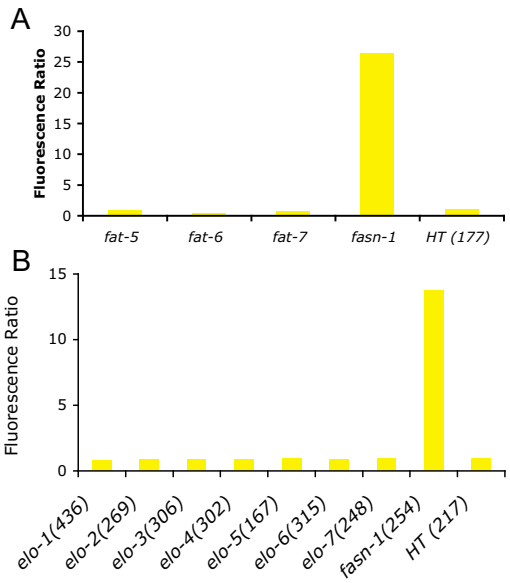
Supplementary Fig. 6. *fasn-1*(RNAi) induces AMPs of different classes. *fasn-1*(RNAi) of uninfected transgenic worms carrying a p*nlp-29*::GFP (A) or p*cnc-2*::GFP reporter (B) in the wild-type background show constitutive expression of GFP, in the absence of an infection. Under normal conditions both strains would have a very low level of expression of GFP.



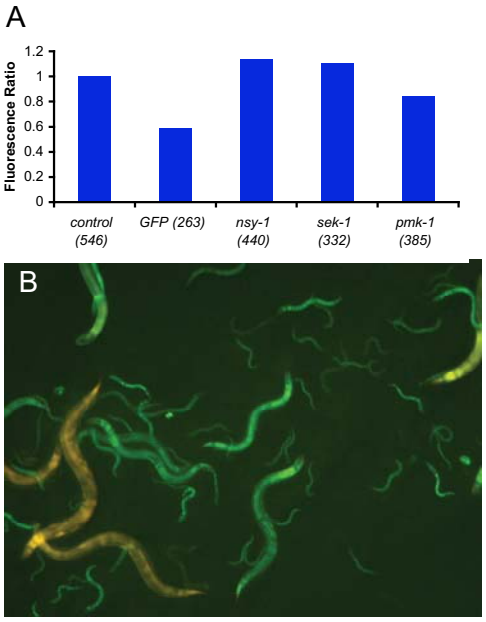
Supplementary Fig. 7. RNAi of *F32H2.6* provokes a Peni phenotype. RNAi knockdown of *fasn-1* (A) and of the N-terminal *fasn-1* like gene *F32H2.6* (B) leads to induction of p*nlp-29*::GFP. (C) quantification of the reporter gene induction. The numbers of worms used is shown in parenthesis. The results shown are representative of three independent experiments.



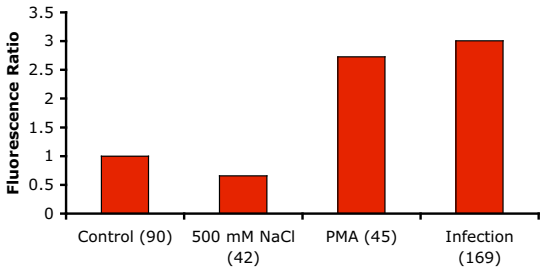
Supplementary Fig. 8. Abrogation of the ACC gene *pod-2* provokes a Peni phenotype. RNAi mediated gene silencing of the *C. elegans* ACC ortholog *pod-2* leads to a Peni phenotype similar to that obtained with *fasn-1*. The numbers of worms used is shown in parenthesis. This figure is representative of three independent experiments.



Supplementary Fig. 9. RNAi of SCD and elongation genes.
(A) Knockdown of the SCD genes *fat-5*, *fat-6* and *fat-7* does not lead to increased expression of the p*nlp-29*::GFP reporter compared empty vector (HT) RNAi controls, and unlike *fasn-1(RNAi)*.
(B) No induction of the p*nlp-29*::GFP reporter after RNAi of *elo* genes. The numbers of worms used is shown in parenthesis. The data are representative of two independent experiments.



Supplementary Fig. 10. Regulation of p*nlp-29*::GFP in *fasn-1(fr8)* is independent of the PKC - p38 MAPK pathway. (A) Quantification following RNAi of the indicated genes in the *fasn-1(fr8);frIs7* strain. The numbers of worms used is shown in parenthesis. The data is a representative of two independent experiments. (B) *fasn-1(fr8); tir-1(tm3036);frIs7* worms show high expression of the reporter p*nlp-29*::GFP and are green. Just as GFP expression decreases with age in adult *fasn-1(fr8);frIs7* worms, so too did it decrease in the double mutant background, so that the old adults on the left of the image appear more red due to the constitutive expression of the p*col-12*::dsRed reporter.



Supplemetary Fig. 11. Extra copies of *fasn-1* block *nlp-29* induction upon osmotic shock
Quantification following exposure to salt (500 mM NaCl), PMA or *D. coniospora* infection of a *wt*;*frIs7* strain carrying an additional transgene (*frEx288*) containing wild-type *fasn-1*. The numbers of worms used is shown in parenthesis. The data are representative of two independent experiments.