Table S1. Oligonucleotides used in this study (f indicates forward primer and r indicates reverse primer).

Genes	Sequence
asp-1	f: 5'-CGTCCTTGACCTTCAACTCG-3'
	r: 5'-ACGGATGAAGGTGTCTCCAA-3'
C15C8.3	f: 5'-ATCGGAGCACCAAAGAGTGT-3'
	r: 5'-AAGAATGCACCCGTAAGTGG-3'
cpr-1	f: 5'- AAAGTACCTCGGAGGACACG-3'
	r: 5'-ATTCCGCATTGATCATCTCC-3'
cpr-2	f: 5'-CAGAACGACCTACACCAACG-3'
	r: 5'-CGGTTCTTGGAACAGGGTAT-3'
cpr-4	f: 5'-GGAACTCCATACTGGCTTGTTG-3'
	r: 5'-CGTTGGTTCCACGGATGATAC-3'
cpr-5	f: 5'-GACAACGGAACCCCATACTG-3'
	r: 5'-CTCGATTCCACACTCGTTGA-3'
thn-2	f: 5'-CCTACGGAACTCCAGACAAGTG-3'
	r: 5'-CGTCGTAGGCATAGGAATAAGC-3'
lys-5	f: 5'-TCCCAGAATTTATCATTCATC G-3'
	r: 5'-TGGCATTCTTGACATTTTGC-3'
F53A9.8	f: 5'-GCGCTAAAACTCAACACCAA-3'
	r: 5'-ATGTCCTTCATGGGAGTCGT-3'
clec-52	f: 5'-ATGGAGGAGATTTGGCTTCA-3'
	r: 5'-CCTGTCCAATCCTTGTCCTT-3'
clec-60	f: 5'-ACGGGCAAGTTATTGGAGAG-3'
	r: 5'-ACACGGTATTGAATCCACGA-3'
clec-71	f: 5'-CGGTATCGAGCAAGACTCA-3'
	r: 5'-GCATTGACGGCATATATTGG-3'
fmo-2	f: 5'-AAGCTGGAGACACGAGGATT-3'
	r: 5'-GGAGTTAAGCATAGCTTGAGGAA-3'
ilys-3	f: 5'-GCGAATGATCTTAGCTGTGC-3'
	r: 5'-CCAGTTCCAGCACATTGACT-3'
F53A9.8	f: 5'-GCGCTAAAACTCAACACCAA-3'
	r: 5'-ATGTCCTTCATGGGAGTCGT-3'
T24B8.5	f: 5'-GTCGACTCAAGACCATCATGC-3'
	r: 5'-GAGTATCGGTAACGCAGACACC-3'
C32H11.1	f: 5'-TTTTGGAAACTACACCCAAGG-3'
	r: 5'-TGTTGGATCATTCGAGGGTA-3'
C32H11.12	f: 5'-GTGTCCAACACAACCTGCAT-3'
	r: 5'-CATGTTTCCATTCACCTGGA-3'
K08D8.5	f: 5'-ATGCTACCGAATGCTTTTCC-3'
	r: 5'-TCCTTGGGTGTAGTTTCCAA-3'
C17H12.8	f: 5'-CCTCCAACTCCATCATCTGAA-3'
	r: 5'-TTTGTGGGTGATGTTTCTCG-3'
F35E12.5	f: 5'-ATTGTCCCCAACACAGAAGC-3'
	r: 5'-GGTAGTCATTGGAGCCGAAA-3'
sod-3	f: 5'-AAATGTCCGCCCAGACTATG-3'
	r: 5'-TGGCAAATCTCTCGCTGA-3'
sod-5	f: 5'-GGTGAAGAGACCGAATTC-3'

	r: 5’-CTTGATCTTCGCAACTCC-3’
mtl-1	f: 5’-TGCAGTCTCCCTTACATCCA-3’
	r: 5’-TGCAGTGGAGACAAGTGTTG-3’
hsp16.2	f: 5’-TATGGCTCTGATGGAACG-3’
	r: 5’-GATTGATAGCGTACGACC-3’
old-1	f: 5’-AAGTGATGTGTGCAGTCG-3’
	r: 5’-GAGAGCCAAATCACGATG-3’
gst-10	f: 5’-GACTGGAGCAATTATGCGTCA-3’
	r: 5’-TCGAAGAACATGTCGAGGAAG-3’
tor-1	f: 5’-AGAATCCTCTGATCTGTGATTGTCA-3’
	r: 5’-GTAGATCCTTCTTGCTGAGACTTTG-3’
pik-1	f: 5’-CTTATTGTGAACTTCTCGAAGCAAC-3’
	r: 5’-GTTAAACTCTGTCGAAGTCGTTCTC-3’
trf-1	f: 5’-AGTCGTTTGCACTTTTGAGTCTTC-3’
	r: 5’-GTCTTGTTACAAAATGAGCAGACAG-3’
ibk-1	f: 5’-GTTCACAGTCCCTTCTCTGAAAGTA-3’
	r: 5’-GCTGAAGTAATGAAATTGTGTCTCC-3’
tir-1	f: 5’-CCTATAAACTCGCTCAACAAGTACC-3’
	r: 5’-CATCATGCTTCAGATCATTCTCAGT-3’
snb-1	f: 5’-CCGGATAAGACCATCTTGACG-3’
	r: 5’-GACGACTTCATCAACCTGAGC-3’

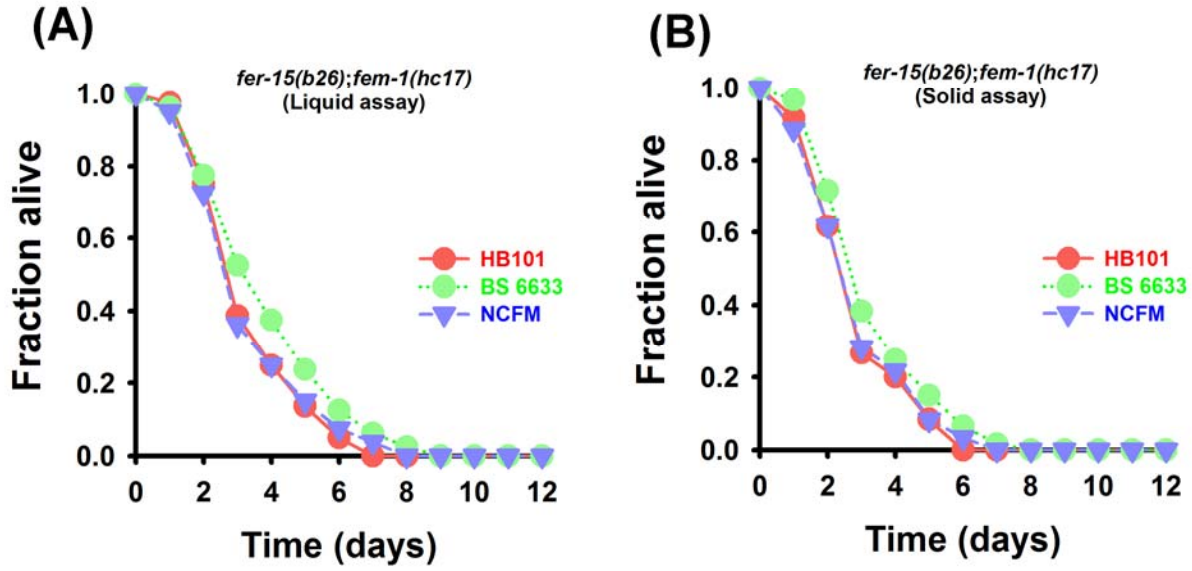

Figure S1. Heat-killed *L. acidophilus* NCFM and *B. subtilis* ATCC 6633 have no effect in the survival of *C. elegans* nematodes infected with *E. faecalis* MMH594. (A) Liquid killing assay (n=40 per well) and (B) solid killing assay (n=30 per plate) of *C. elegans fer-15;fem-1* worms infected by *E. faecalis* MMH594 following conditioning with heat-killed *L. acidophilus* NCFM or *B. subtilis* ATCC 6633 cells for 24 h (liquid killing assay [p=0.7702 and p=0.0623 compared with worms feeding on HB101, respectively] and Solid killing assay [p=0.7872 and p=0.0813 compared with worms feeding on HB101, respectively]).

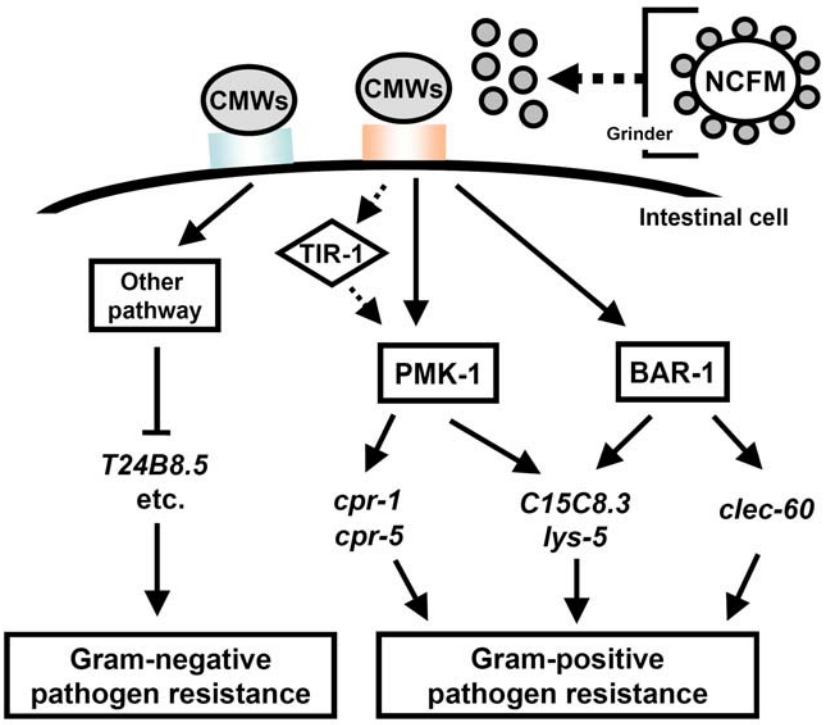

Figure S2. Proposed model of the conditioning mechanism associated with *C. elegans* exposure to NCFM probiotic bacteria. Our working hypothesis is that when intact probiotic cells are digested by the worm grinder, cell wall molecules (CWMs) up-regulate specific host immune responses. These probiotic ligands may stimulate immune response genes resulting in lower infection levels and prolonged survival during Gram-positive infections via the TIR-1, PMK-1 and BAR-1 pathways. → indicates induction, ⊥ indicates repression.