**Table S1.** **Detrimental effects of KGB-1 activation on resistance to *Pseudomonas aeruginosa* infection depend on *mir-71***

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Strain a** | **Median survival time (days c)** | **Median survival fold change d** | **n** | **Log-Rank p-value d** |
| **EV control** | ***vhp-1*****RNAi** | **EV control** | ***vhp-1*****RNAi** |
| *wt* ***b*** | 7.2 | 5.5 | 0.76 | 87 | 73 | <0.0001 |
| *daf-12*(*rh61rh412)* | 5.3 | 4.1 | 0.77 | 66 | 62 | <0.0001 |
| *kri-1*(*ok1251*) | 4.9 | 3.2 | 0.65 | 94 | 59 | <0.0001 |
| *mir-71*(*n4115*) | 2.5 | 3.2 | 1.29**d** | 79 | 93 | <0.0001**d** |
| *wt* | 6.3 | 4.2 | 0.66 | 86 | 87 | <0.0001 |
| *daf-12*(*rh61rh412)* | 4.8 | 2.8 | 0.58 | 85 | 91 | <0.0001 |
| *kri-1*(*ok1251*) | 3.0 | 2.7 | 0.91 | 74 | 73 | 0.0002 |
| *mir-71*(*n4115*) | 2.3 | 3.0 | 1.29 | 89 | 92 | 0.0074 |
| *wt* | 4.5 | 3.4 | 0.76 | 40 | 37 | <0.0001 |
| *daf-12*(*rh61rh412)* | 5.4 | 3.2 | 0.59 | 48 | 40 | <0.0001 |
| *kri-1*(*ok1251*) | 2.6 | 1.7 | 0.65 | 50 | 51 | <0.0001 |
| *mir-71*(*n4115*) | 1.8 | 2.2 | 1.20 | 50 | 50 | 0.016 |
| *wt* | 4.8 | 2.8 | 0.59 | 107 | 124 | <0.0001 |
| *daf-12*(*rh61rh412)* | 4.1 | 4.1 | 1.00 | 42 | 36 | n.s. |
| *kri-1*(*ok1251*) | 4.1 | 3.2 | 0.78 | 134 | 125 | <0.0001 |
| *mir-71*(*n4115*) | 1.4 | 2.0 | 1.37 | 144 | 126 | <0.0001 |

**a** All strains were rendered sterile by *cdc-25.1* RNAi treatment during development to disrupt germline proliferation (see Methods).

**b** Each shade-separated block represents an independent experiment.

**c** Days are counted starting at day 2 of adulthood, following *vhp-1* knock-down or exposure to empty vector (EV).

**d** Red values denote instances where *vhp-1* RNAi has a positive effect on survival. Previous results have demonstrated (in *kgb-1* mutants) that this is due to activation of the p38 ortholog, PMK-1, a protective (age-invariably) MAPK also negatively regulated by VHP-1 (Twumasi-Boateng et al., 2012). In the absence of the detrimental effects of activated KGB-1, this protection become a dominant outcome of *vhp-1* RNAi.