**Table S2. RNAi screen of *C. elegans* nucleoporins**

| *C. elegans* gene | Vertebrate Nup | Percentage broods with abnormal GFP::PGL-1 (*n*) | Abnormal K76 staining | Embryonic lethality (%) |
|---|---|---|---|---|
| *npp-1* | Nup54 | 28 (13) | No | 95 |
| *npp-2* | Nup85 | 33 (6) | No | 31 |
| *npp-3* | Nup205 | 33 (6) | No | 100 |
| *npp-4* | Nup45/58 | 0 (5) | No | 84 |
| *npp-5* | Nup107 | 20 (5) | No | (reported 0*) |
| *npp-6* | Nup160 | 18 (17) | No | 98 |
| *npp-7* | Nup153 | 44 (32) | YES | 81 |
| *npp-8* | Nup155 | 78 (23) | YES | 85 |
| *npp-9* | Nup358 | 78 (83) | YES† | 80 |
| *npp-10* | Nup98/96 | 100 (22) | YES | 95 |
| *npp-11* | Nup62 | 0 (6) | No | 88 |
| *npp-12* | Gp120 | 0 (10) | | (reported 0) |
| *npp-13* | Nup93 | 25 (12) | No | 58 |
| *npp-14* | Nup214 | 0 (16) | | (reported 0) |
| *npp-15* | Nup133 | 0 (6) | | (reported 0) |
| *npp-16* | Nup50 | 0 (7) | | (reported 0) |
| *npp-17* | RAE1 | 0 (6) | | (reported 0 |
| *npp-18* | Seh1 | 0 (7) | | (reported 0) |
| *npp-19* | Nup35 | 28 (31) | No | 84 |
| *npp-20* | Sec13R | 0 (8) | | 70 |
| *npp-21* | Tpr | 0 (6) | | (lethal only in *rrf-3*) |
| *npp-22* | TMEM48 | 0 (6) | | (reported 0) |
| *C09G9.2* | Nup43 | 0 (10) | | (reported 0) |
| *mel-28* | ELYS | 0 (23) | | 68 |
| | | | | |
| *nxf-1* | | 0 (8) | No | 97 |
| *ama-1* | | 0 (8) | No | 92 |
| *rps-6* | | 0 (3) | | 50 |
| *lmn-1* | | 0 (12) | No | 95 |
| Vector | | 0 (14) | No | 0 |

JH2107 (GFP::PGL-1) was subjected to the indicated RNAi for 22 hours by feeding and the hermaphrodites were scored for embryos with abnormal GFP::PGL-1. Percentages are the numbers of broods with one or more embryos with abnormal GFP::PGL-1. All positives and several negatives were subjected to a secondary RNAi screen using wild-type worms and endogenous PGL-1 scored by immunostaining (K76 antibody) (Strome and Wood, 1982). The efficacy of the RNAi treatments was evaluated by scoring embryonic lethality; values (including those of no lethality) were similar to those reported previously (Galy et al., 2003). Identities of vertebrate homologs are according to previous reports (Galy et al., 2003; Franz et al., 2005).

*As in Galy et al. (Galy et al., 2003).

†Defects might be secondary to general cytological abnormalities (see Fig. 1F).