SUPPLEMENTARY INFORMATION:

Figure 1: Alignment of Elongin C homologues from *H. sapiens*, *C. elegans* and *D. melanogaster*.

Figure 2: Embryos expressing PIE-1::GFP with genes in italics inactivated by RNAi. PIE-1::GFP is degraded only in somatic blastomeres in wild-type embryos, but is degraded in all cells in *par-1(RNAi)* embryos. Ubiquitous degradation in *par-1* mutants requires ZIF-1, MEX-5/6, and CUL-2. These experiments were done in parallel with those in Fig. 4 and control for the efficacy of the double RNAi experiments: in *par-1(RNAi)zif-1(RNAi)* and *par-1(RNAi)cul-2(RNAi)* doubles, PIE-1:GFP no longer segregates preferentially with the germline as in *par-1(RNAi)*, and is stabilized in all cells as in *zif-1(RNAi)* or *cul-2 (RNAi)*. Identical results were obtained with *par-1(ax53);zif-1(RNAi)*, *par-1(ax53);mex-5/6(RNAi)* and *par-1(ax53);cul-2(RNAi)* embryos expressing PIE-1:GFP.

mex-5 and *mex-6* are required for both PIE-1 asymmetry in germline blastomeres and degradation in somatic blastomeres ([4]; this work); as a result *mex-5(RNAi)mex-6(RNAi)* embryos resemble *par-1(RNAi)mex-5(RNAi)mex-6(RNAi)* embryos. Similar results were observed with *par-1(b274);mex-5(RNAi);mex-6(RNAi)* embryos in [4], consistent with *mex-5* and *mex-6* being epistatic to *par-1*.

Figure 3:

a. 4-cell embryos expressing GFP::PIE-1^{ZF1}. Embryos are partially depleted for CUL-3 or UBC-12 as indicated. Degradation of GFP::PIE-1^{ZF1} in somatic blastomeres is not blocked.

b. Fixed 12-cell wild-type and *cul-2(RNAi)* embryos expressing *pie-1*$_{prom}$:GFP::ZIF-1 and stained with anti-GFP antibody (Molecular Probes).