



Figure S2. DEXseq analysis identified three genes in the *cnd-1(ju29)* RNA-seq dataset with significantly different coding blocks. (A) DEXseq analysis of *aak-2*. Transcripts from an intron 2 transcriptional start site (asterisk) are about two-fold higher in *cnd-1* mutants when compared to wild type suggesting that CND-1 normally represses this transcript variant. (B) The G protein-coupled receptor *srw-85* is expressed at a significantly lower level in *cnd-1(ju29)* mutants (asterisk). (C) Illustration of the *srw-85* locus and reporter gene used to identify its expression pattern. (D-G) *srw-85p::GFP* is expressed in the ciliated ASJ chemosensory neurons and co-labels with the lipophilic dye DiD. (I) DEXseq analysis of the *ptrn-1* locus shows significantly lower transcript levels in the 3' UTR of this gene (asterisk), which was caused by the 7kb genomic deletion, *ken2*. (J) Integrated Genome

Viewer screenshot of the *ptrn-1* locus from the three wild type and *cnd-1(ju29)* RNA-seq datasets. Red box shows the missing transcript in the *ptrn-1* 3' untranslated region caused by the *ken2* deletion identified in the *cnd-1(ju29)* samples. Arrows show location of PCR primers used to probe this region. (K) PCR amplification of wild type, *cnd-1(ju29)* and *cnd-1(gk718)* genomic DNA using the F1 + R1 and F1 + R2 primers annotated that target the *ptrn-1* 3' end. Note that the F1 + R2 amplicon is missing in *cnd-1(ju29)* mutants but present in the *cnd-1(gk718)* allele. (L) www.wormbase.org screenshot of the *ptrn-1* gene and surrounding genomic region showing genes affected by the *ken2* deletion. F35B3.1 and F35B3.4 appear as significantly down-regulated in our *cnd-1(ju29)* transcriptome and have essentially zero transcript (Table 1 and Table S2).