

Supplemental Table 1: Additional *C. elegans* homologs of human disease genes that may be useful for this laboratory exercise.

<b>Gene (allele) STRAIN</b>	<b>Molecular function of gene product</b>	<b><i>C. elegans</i> deletion phenotype from Wormbase</b>	<b><i>C. elegans</i> feeding RNAi phenotype from Wormbase</b>	<b>Human homolog (Genbank accession number)</b>	<b>Associated human disease (OMIM number)</b>	<b>Special treatment conditions to visualize phenotypes</b>
<i>eya-1</i> ( <i>ok654</i> ) SA146	Protein tyrosine phosphatase, Transcriptional regulation	Early larval arrest, higher penetrance above/below 20°C, Uncoordinated, Dumpy, Egg-laying defective, Multivulval	Locomotion variant, Dumpy	EYA1 (Q99502)	Bor syndrome (600257)	Temperature affects phenotype penetrance for deletion allele.
<i>him-6</i> ( <i>ok412</i> ) VC193	ATP-dependent DNA helicase	Germline mortal	Locomotion variant	BLM (U39817)	Bloom syndrome (210900)	Grow <i>him-6(ok412)</i> worms at 25°C to see germline mortal phenotype.
<i>itsn-1</i> ( <i>ok268</i> ) VC201	ATP-binding; Role at synapses	Aldicarb hypersensitive	Oocyte morphology variant	NCF1 (M55067)	Chronic granulomatous disease (306400)	For RNAi, use <i>rrf-3(pk1426)</i> worms [strain: NL2099, these are hypersensitive to RNAi]. Weaker touch response to aldicarb, see S. Rose, et al. PMID 17942601.
<i>ogt-1</i> ( <i>ok430</i> ) RB653	Protein N-acetylglucosaminyl transferase activity, Protein serine/threonine phosphatase activity	Drug resistant, Phermone-induced dauer formation defective	Dumpy	OGT (8473)	Maps to a region associated with neurologic diseases, X-linked (300255)	Deletion requires treatment with PUGNAc or daumone, see J. Lee, et al. PMID 19940149.

<i>pdr-1</i> ( <i>lg103</i> ), no strain in CGC or try (gk488) VC1024	E3 ubiquitin protein ligase	Developmental defects, Early larval lethality (see Springer, PMID 16204351)	Embryonic lethal	parkin (AB009973)	Parkinson disease juvenile 2 (600116)	Deletion requires treatment with an ER stressor, see Springer et al. PMID 16204351; contact authors for strain or VC1024 may have an ER stressor phenotype (untested).
<i>sma-1</i> ( <i>ral8</i> )	Beta Heavy- spectrin	Reduced body length (by 44%), Abnormal pharynx, Small brood size	Reduced body length, Slow growth, Body shape defects, Small brood size	SPTB (J05500.1)	Hereditary spherocytosis and elliptocytosis (182870)	None needed.
<i>wrn-1</i> ( <i>gk99</i> ) VC174	ATP-dependent 3'-5' DNA helicase activity	Hypersensitivity to ionizing radiation	Head and other morphology defects	WRN (L76937)	Werner syndrome (277700)	Ionizing radiation, see SJ Lee et al. PMID 20062519.
<i>wsp-1</i> ( <i>gm324</i> ) NG324	Actin regulator, Interacts with Arp2/3	Embryonic lethality (25%), Reduced brood size	Embryonic lethal, Larval arrest, Locomotion variant	WASP (U12707)	Wiskott–Aldrich syndrome (301000)	None needed.
<i>xnp-1</i> ( <i>tm678</i> ) IG256	ATP-dependent DNA helicase	Sterile at 25 <sup>0</sup> C	Gonad defects, Small brood size	nibrin (AF051334)	Nijmegen breakage syndrome (251260)	Deletion sterile at 25 <sup>0</sup> C; for RNAi see AM Bender, et al. PMID 15328017.

## Legend for Supplemental Table 1

Some of the genes utilized in the described laboratory module have relatively mild phenotypes. Therefore, we continued to search for more genes that may be tried in the future. This table presents nine genes that could potentially be used effectively in this lab exercise, based on information in Wormbase and the literature. The genes listed here were derived from Culetto et al. (2000), which contains a table of one hundred *C. elegans* genes that are homologues of positionally cloned genes mutated in human disease. An additional thirty-eight genes from the scientific literature were also examined.

Due to the nature of the experiments performed in this laboratory exercise, only the nine genes listed in this table appear to be good candidates for use (in addition to the four in use already). Genes were eliminated from consideration based on the following issues:

- 1) the gene had no reported deletion phenotype
- 2) no deletion allele (in some cases, point mutations are available; however, point mutations are more difficult to detect by PCR, and would require revision of the current protocol.)
- 3) no *C. elegans* strains available even though a deletion allele has been described
- 4) the deletion is lethal or sterile and so cannot be maintained as a homozygote
- 5) deletion too large to easily characterize by PCR (>3000 bp)
- 6) deletion too small to easily detect by PCR (<200 bp)
- 7) no RNAi by feeding phenotype listed in Wormbase

The genes listed in this table do not have these limitations; however, most will require an additional treatment to bring out a phenotype, such as using an RNAi hypersensitive strain, or a chemical treatment. Interestingly, several of the genes that fit the selection criteria are DNA helicases (yellow rows) and could make for a DNA helicase-themed lab exercise.