Table 1. No rescue of *sel-12/lin-12, glp-1* defects by *Ce-imp-2* RNAi

Strains	RNAi	Total worms	T, ˚C	Phenotype
sel-12(ar171) unc-1	*Ce-imp-2*	136	room	100% Egl
unc-32(e189) lin-12(n676n930)III		147	25	100% Egl
glp-1(e2142ts)		P_0 , 162	25	F_1 , 100% Emb

No rescue of *sel-12/lin-12, glp-1* defects by *Ce-imp-2* RNAi. Deficiency in *sel-12* alone (with a functional *hop-1* paralogous gene) does not induce the *lin-12, glp-1* phenotypes, but certain alleles produce an incompletely (e.g., *sel-12 (ar131*)) or completely (e.g., *sel-12 (ar171*) *unc-1*) penetrant recessive egg-laying defective phenotype (Egl) and large vulval protrusion. The loss-of-function of certain *lin-12* mutants is also associated with the Egl (egg-laying defect) phenotype and abnormal vulval development. We found no prominent Egl phenotype or protruding vulvae in N2 wild-type animals fed with *Ce-imp-2* dsRNA. However, we occasionally observed eggs that hatched within the mother and reduced brood size, resembling effects in *sel-12* mutants (1). We next used sensitized genetic backgrounds to look for interactions between *Ce-imp-2* and Notch-pathway components. To do this, we used RNAi to knock down *Ce-imp-2* activity in (*i*) *sel-12(ar171)unc-1(e538)* and *sel-12(ar131)* (presenilin loss-of-function mutants); (*ii*) *unc-32(e189) lin-12(n676n930) III* strain (temperature sensitive hypomorphic *lin-12* allele at 25˚C); (*iii*) *lin-12(n137)/unc-32(e189)III;him-5(e1467)V* (a *lin-12* hypermorphic strain); (*iv*) *glp-1(e2142ts)* (temperature-sensitive hypomorphic *glp-1* allele). None of these mutant strains exhibited enhancement or suppression of their corresponding phenotypes upon induction *Ce-imp-2* (RNAi). For example, in the *sel-12(ar171) unc-1* and *sel-12(ar131)* mutant strain, RNAi targeting the *sel-12* homolog *hop-1* induces an embryonic arrest and loss of anterior pharynx (2). No such defect in pharyngeal development was induced by *Ce-imp-2* (RNAi) in the *sel-12* mutants background. We found also that *Ce-imp-2* (RNAi) did not rescue Egl phenotype of *sel-12(ar171) unc-1*. Likewise, inhibition of *Ce-imp-2* in the *lin-12(n137)/unc-32(e189)III;him-5(e1467)V* hypermorphic strain did not reduce or enhance the multivulva phenotype of this strain. Finally, RNAi targeting

Ce-imp-2 in the hypomorphic *glp-1(2142ts)* genetic background did not reduce the rate of embryonic lethality (Emb) in this strain.

1. Lakowski, B., Eimer, S., Gobel, C., Bottcher, A., Wagler, B. & Baumeister, R. (2003) *Development* **130,** 2117-2128.

2. Li, X. & Greenwald, I. (1997) *Proc. Natl. Acad. Sci. USA* **94,** 12204-12209.