Supporting information for Bülow *et al.* (2002) *Proc. Natl. Acad. Sci. USA* **99** (9), 6346–6351. (10.1073/pnas.092128099)

Table 2. Forced expression of *kal-1* in various tissue

Tissue	Construct	*Is/Ex*	Cells examined	Reporter	Effect*
Panneuronal	*unc-119::kal-1*	*Is*	All neurons	*unc-119::gfp (otIs45)*	–
		Is/Ex	*kal-1*-expressing	*kal-1::gfp (otIs33)*	–
		Is/Ex	AIY interneurons	*ttx-3::gfp (mgIs18)*	++
		Is/Ex	AFD thermosensory	*gcy-8::gfp (oyIs17)*	+++
		Is	ASER chemosensory	*gcy-5::gfp (ntIs1)*	+†
		Is/Ex	Amphid sens. neurons	DiI	++
		Is	PVQ interneurons	*sra-6::gfp (oyIs14)*	+‡
		Is	DVB, PVT, etc.	*unc-47::gfp (otIs37)*	–
		Is	D-type motor neurons	*unc-47::gfp (oxIs12)*	+§
		Is	HSN	*tph-1::gfp (mgIs71)*	–
		Is	Touch neurons	*mec-18::gfp (uIs25)*	+(+)¶
Heat shock	*phsp16-2::kal-1*	*Is*	AIY interneurons	*ttx-3::gfp (mgIs18)*	–
AIY interneuron	*ttx-3::kal-1*	*Is/Ex*	AIY interneurons	*ttx-3::gfp (mgIs18)*	+++
		Ex	AFD thermosensory	*gcy-8::gfp (oyIs17)*	–
		Ex	AWC odorsensory	*str-2::gfp (kyIs140)*	–
		Ex	ASER chemosensory	*gcy-5::gfp (ntIs1)*	–
AFD sensory	*gcy-8::kal-1*	*Is/Ex*	AFD thermosensory	*gcy-8::gfp (oyIs17)*	++
		Is	AIY interneurons	*ttx-3::gfp (mgIs18)*	–
		Is	AWA odorsensory	*odr-10::gfp (kyIs37)*	–
		Is	AWC odorsensory	*str-2::gfp (kyIs140)*	
		Is	ASER chemosensory	*gcy-5::gfp (ntIs1)*	–
VNC motor neuron	*unc-4::kal-1*	*Ex*	VC motor neurons	*lin-11::gfp (nIs106)*	–
DVB motor neuron	*lim-6int3::kal-1*	*Ex*	DVB motor neuron	*unc-47::gfp (oxIs12)*	–
Touch neurons	*mec-7::kal-1*	*Ex*	Touch neurons	*mec-18::gfp (uIs25)*	+(+)¶

Enteric muscle	*pEMC::kal-1*	*Ex*	DVB motor neuron	*unc-47::gfp (oxIs12)*	–

Experiments were done with at least three extrachromosomal lines or integrated lines as indicated. *Is*, integrated line; *Ex*, extrachromosomal line. In bold are neurons that express the *kal-1*::*gfp* reporter; hence, heterologous expression of *kal-1* in these cells may classify as "overexpression" rather than "misexpression." Promoters and reporters are described in the text.

*Effect detected: no defect, –; minor defects (9–20%), +; substantial defects (21–69%), ++; strong defects (70–100%), +++.

[†]ASER showed 15% commissure misrouting defects.

[‡]Of the animals, 8% show strong defects (laterally meandering axon) that are never seen in wild type.

[§]Animals have a low penetrance (<10 %) defect in the D-type commissure [both with regard to the choice of side and to reaching the dorsal nerve cord (DNC)].

[¶]Of the animals, 25% showed axon misrouting and branching defects, e.g., the axon of PVM, which usually runs ventrally and joins the right fascicle, bifurcated at the VNC and sent one branch anteriorly (as in wild type) and one posteriorly. Alternatively, the axon left the fascicle in the right VNC again and traveled on a sublateral tract anteriorly.