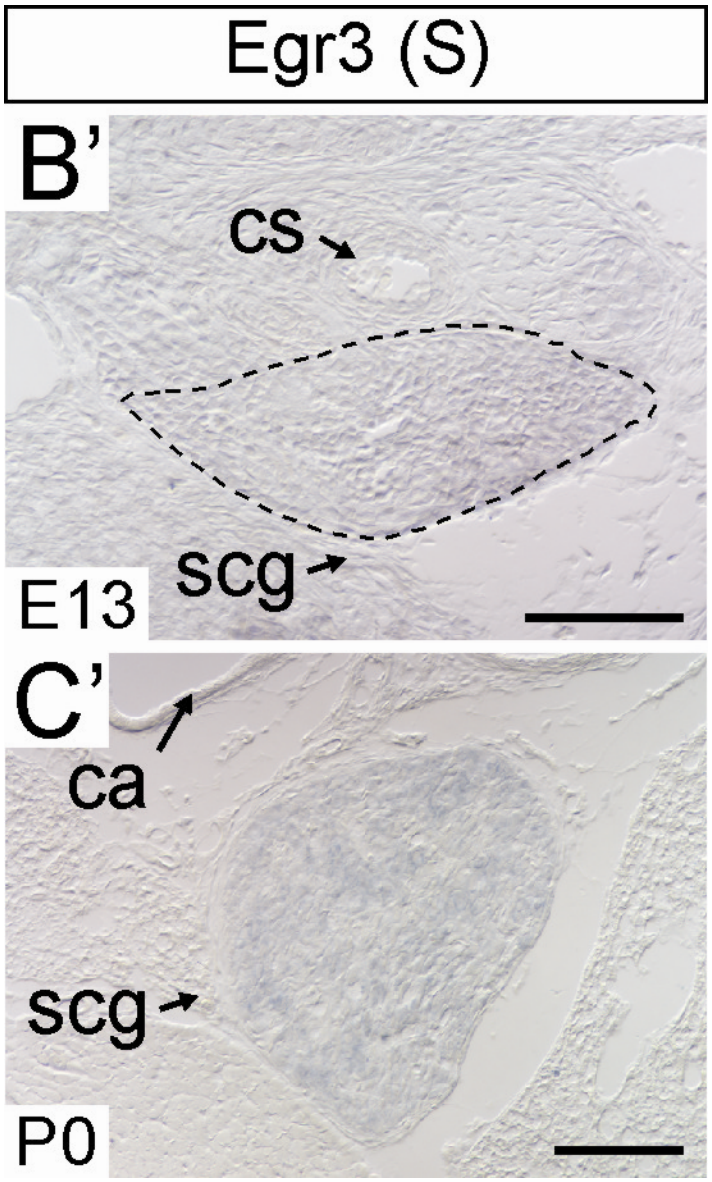

Figure S1. Egr3 is developmentally regulated in SCG neurons. **(B', C')** Tissue sections adjacent to those shown in Figure 1B and C hybridized with the sense Egr3 probe. No non-specific hybridization was observed. (cs = carotid sinus; ca = carotid artery; scg = superior cervical ganglion; scale bar = 100 μm)

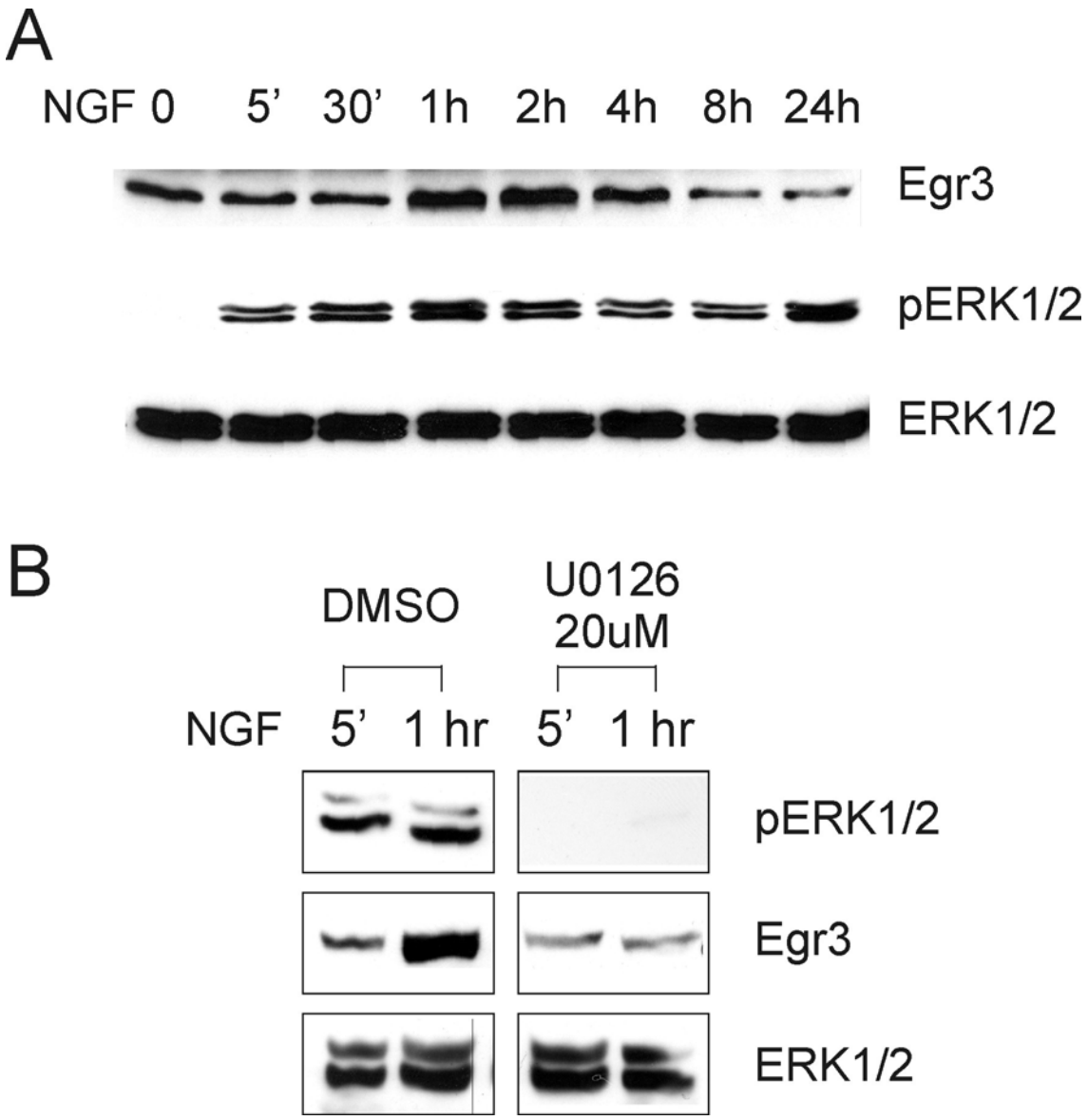

Figure S2. MEK-dependent Egr3 protein induction after NGF-treatment of SH-SY5Y/TrkA human neuroblastoma cells. **(A)** Egr3 protein was induced in SH-SY5Y/TrkA cells after 100 ng/mL NGF treatment. Egr3 protein levels peaked 1-2 hours after stimulation and returned to basal level by 8 hours post stimulation. ERK phosphorylation (pERK1/2) was detectable 5 minutes after NGF treatment and remained detectable for at least 24 hours. **(B)** NGF-mediated ERK phosphorylation and Egr3 induction, but not basal Egr3 expression, were completely abrogated by the MEK inhibitor U0126.

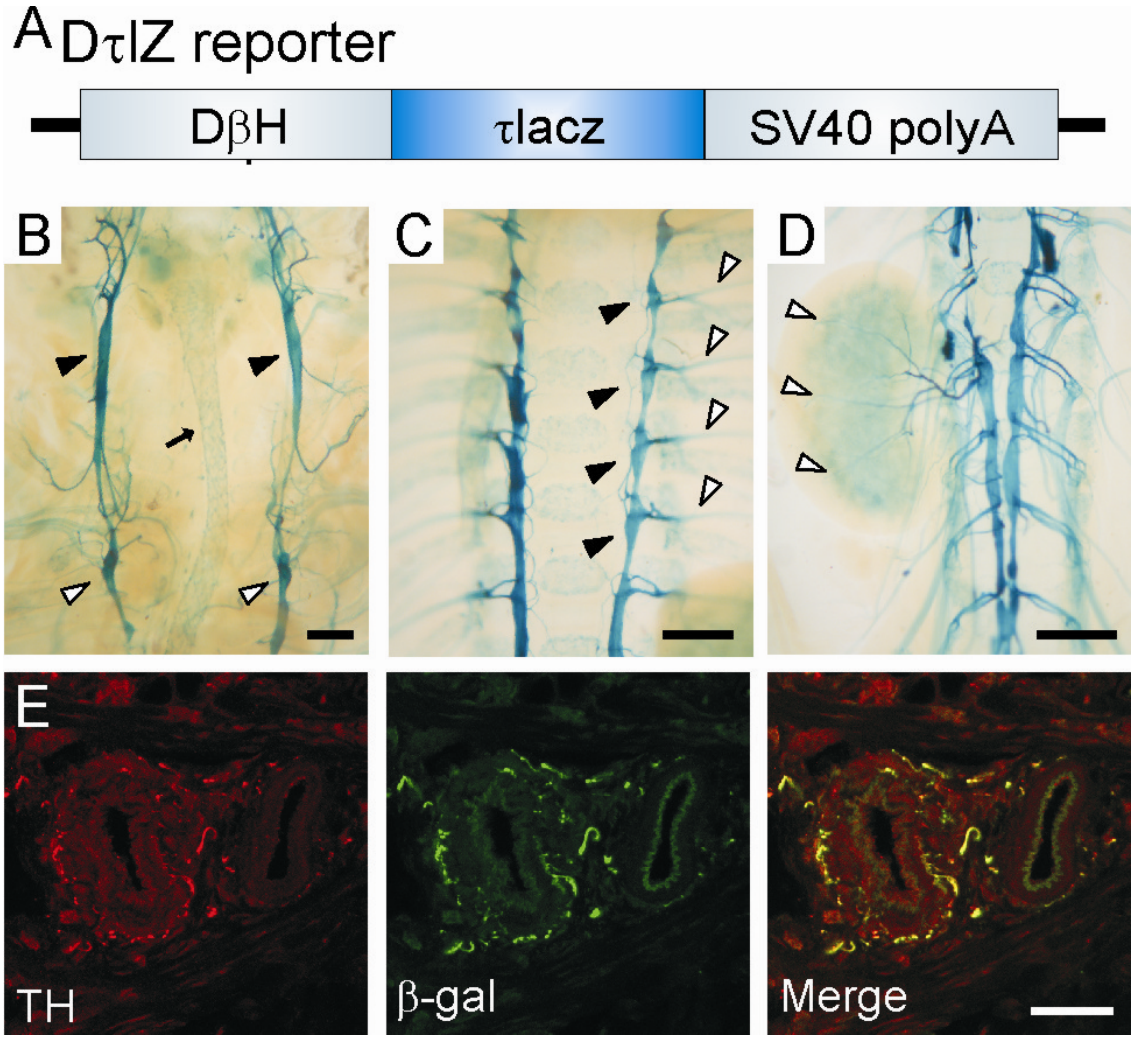

Figure S3. Generating dopamine β-hydroxylase-τlacZ (DτlZ) sympathetic neuron reporter mice. **(A)** In DτlZ transgenic mice the τlacZ fusion protein was expressed under the control of the human dopamine β-hydroxylase promoter. Whole mount lacZ histochemistry from transgenic mice showed robust expression of the τlacZ transgene in all sympathetic neurons and their axons. **(B)** The superior cervical ganglia (black arrowheads), stellate ganglia (white arrowheads) and their processes were well visualized using whole mount lacZ histochemistry. In addition, delicate sympathetic terminal axons innervating target organs were also well visualized, such as those innervating the esophagus shown here (arrow). **(C)** The thoracic sympathetic chain ganglia (black arrowheads) and their axons (white arrowheads) which merge into intercostal nerves were also well visualized in DτlZ^{+} transgenic mice. **(D)** In many organs such as the kidney shown here, sympathetic innervation within the organ parenchyma could be visualized with high resolution (white arrowheads). **(E)** That lacZ was a reliable surrogate reporter for sympathetic neurons and their axons in DτlZ^{+} transgenic mice was confirmed by tyrosine hydroxylase (TH, left) and β-galactosidase (β-gal, center) double labeling immunofluorescence which demonstrated 100% colocalization (merged image, right) between TH and the τlacZ reporter transgene (exemplary sympathetic innervation to a small arteriole in the tail from a DτlZ^{+} transgenic mouse shown here). (Magnification bar, B-D = 500μm and E = 50μm)

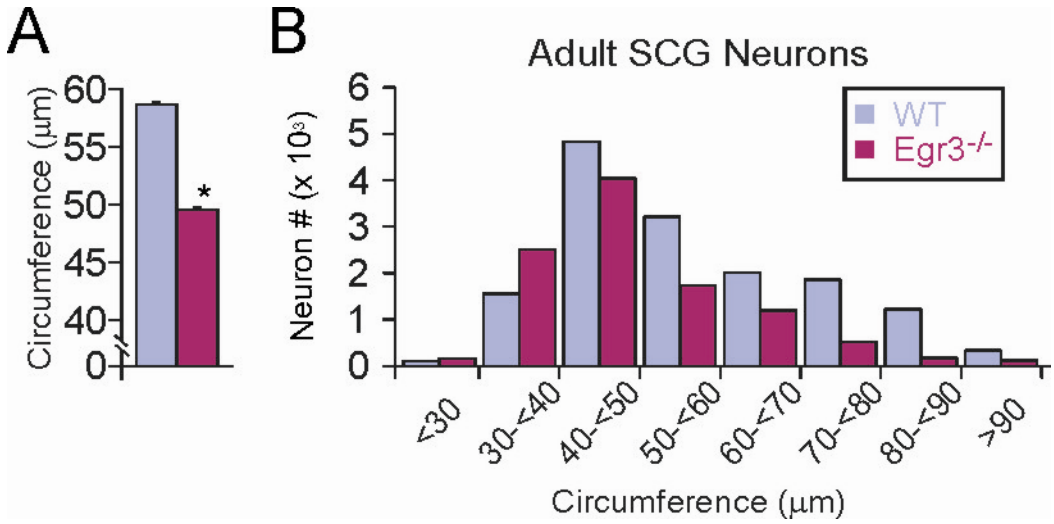

Figure S4. Sympathetic neuron atrophy in surviving SCG neurons in Egr3$^{-/-}$ mice. **(A)** The diameter of surviving adult SCG neurons was significantly decreased in Egr3$^{-/-}$ mice relative to wild type (** = $p < 0.01$, Student's t test). **(B)** Although about 1/3 of sympathetic neurons were lost in the SCG in Egr3$^{-/-}$ mice, the decrease in large diameter SCG neurons was accompanied by an increase in small diameter neurons, suggesting that at least part of the decrease in neuron diameter is due to atrophy. Thus, there was a left shift in the diameter-frequency histogram of SCG neurons from Egr3$^{-/-}$ relative to wild type mice.