nrx-1(wy778);nlg-1(ok259) double null mutant males with NRX-1 expressed in DVB showed an increase in neurites, similar to *nlg-1* mutants (Extended Data Fig. 9g–i). Hence, restoration of NRX-1 expression in DVB with otherwise global loss of NRX-1 and NLG-1 recapitulates NLG-1 loss alone, suggesting that the *nlg-1* phenotype requires NRX-1 in DVB. GFP-tagged NRX-1 localized diffusely onto the membranes of soma and processes and did not appear to change between days 1 and 3 (Extended Data Fig. 9j). By contrast, expression of GFP-tagged NLG-1 decreased from days 1 to 3 in DVB-targeted muscles and neurons (Extended Data Fig. 8). Hence, NRX-1 appears to function cell-autonomously in DVB to promote DVB neurite outgrowth, whereas NLG-1 operates in postsynaptic partners of DVB to antagonize NRX-1-dependent growth. Decreases in NLG-1 expression may result in a reduction in the antagonistic relationship, thereby permitting more NRX-1-dependent neurite elaboration. Our demonstration of an antagonistic neurexin–neuroligin relationship that influences neurite outgrowth hints at a signalling process downstream of neurexin that is antagonized by neuroligin and is independent of neurexin's PDZ domain.

Finally, we tested whether manipulations that induce DVB neurites in males can also induce neurites in hermaphrodite DVB neurons. Activation of the anal depressor muscle (*gar-3b::ChR2::yfp*), loss of NLG-1, loss of NRX-1, or overexpression of NRX-1 in DVB had no effect on the axon morphology of hermaphrodite DVB neurons (Extended Data Fig. 10). Cell-autonomous sexual identity changes of either DVB or postsynaptic muscles using genetic manipulations of the sex-determination pathway also did not alter DVB morphology (see Methods). Thus, sexually dimorphic morphology and plasticity of the sex-shared DVB neuron seems to be non-autonomously instructed by male-specific circuit components.

Experience-dependent neuronal plasticity in the adult brain can include remodelling of dendrites and axons for behavioural adaptation or homeostatic maintenance of circuits. Our findings regarding male-specific DVB neurite outgrowth in *C. elegans* reveal the functional effect of morphological remodelling on circuits and behaviour. Through neurite outgrowth and rewiring of specific synapses, the DVB neuron undergoes a functional change that is likely to serve as an adaptive mechanism, perhaps translating experience into finer coordination of circuit activity and subsequent muscle contraction. These findings may have implications for the normal functions of neurexin and neuroligin in plasticity, and for the many human diseases associated with them.

Online Content Methods, along with any additional Extended Data display items and Source Data, are available in the online version of the paper; references unique to these sections appear only in the online paper.

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1. Fu, M. & Zuo, Y. Experience-dependent structural plasticity in the cortex. *Trends Neurosci.* **34,** 177–187 (2011).
2. Holtmaat, A. & Svoboda, K. Experience-dependent structural synaptic plasticity in the mammalian brain. *Nat. Rev. Neurosci.* **10,** 647–658 (2009).
3. Vitureira, N. & Goda, Y. Cell biology in neuroscience: the interplay between Hebbian and homeostatic synaptic plasticity. *J. Cell Biol.* **203,** 175–186 (2013).
4. Yin, J. & Yuan, Q. Structural homeostasis in the nervous system: a balancing act for wiring plasticity and stability. *Front. Cell. Neurosci.* **8,** 439 (2015).
5. Lee, W. C. *et al.* Dynamic remodeling of dendritic arbors in GABAergic interneurons of adult visual cortex. *PLoS Biol.* **4,** e29 (2006).
6. Marik, S. A., Yamahachi, H., Meyer zum Alten Borgloh, S. & Gilbert, C. D. Large-scale axonal reorganization of inhibitory neurons following retinal lesions. *J. Neurosci.* **34,** 1625–1632 (2014).
7. Keck, T. *et al.* Loss of sensory input causes rapid structural changes of inhibitory neurons in adult mouse visual cortex. *Neuron* **71,** 869–882 (2011).
8. Jarrell, T. A. *et al.* The connectome of a decision-making neural network. *Science* **337,** 437–444 (2012).
9. White, J. G., Southgate, E., Thomson, J. N. & Brenner, S. The structure of the nervous system of the nematode *Caenorhabditis elegans. Phil. Trans. R. Soc. Lond. B* **314,** 1–340 (1986).
10. Reiner, D. J. & Thomas, J. H. Reversal of a muscle response to GABA during *C. elegans* male development. *J. Neurosci.* **15,** 6094–6102 (1995).
11. LeBoeuf, B. & Garcia, L. R. *Caenorhabditis elegans* male copulation circuitry incorporates sex-shared defecation components to promote intromission and sperm transfer. *G3* **7,** 647–662 (2017).
12. Pokala, N., Liu, Q., Gordus, A. & Bargmann, C. I. Inducible and titratable silencing of *Caenorhabditis elegans* neurons *in vivo* with histamine-gated chloride channels. *Proc. Natl Acad. Sci. USA* **111,** 2770–2775 (2014).
13. Garcia, L. R., Mehta, P. & Sternberg, P. W. Regulation of distinct muscle behaviors controls the *C. elegans* male's copulatory spicules during mating. *Cell* **107,** 777–788 (2001).
14. Hobert, O., Tessmar, K. & Ruvkun, G. The *Caenorhabditis elegans lim-6* LIM homeobox gene regulates neurite outgrowth and function of particular GABAergic neurons. *Development* **126,** 1547–1562 (1999).
15. Feinberg, E. H. *et al.* GFP reconstitution across synaptic partners (GRASP) defines cell contacts and synapses in living nervous systems. *Neuron* **57,** 353–363 (2008).
16. Liu, Y. *et al.* A cholinergic-regulated circuit coordinates the maintenance and bi-stable states of a sensory-motor behavior during *Caenorhabditis elegans* male copulation. *PLoS Genet.* **7,** e1001326 (2011).
17. Liu, Y., LeBoeuf, B. & Garcia, L. R. $G\alpha_q$-coupled muscarinic acetylcholine receptors enhance nicotinic acetylcholine receptor signaling in *Caenorhabditis elegans* mating behavior. *J. Neurosci.* **27,** 1411–1421 (2007).
18. Jobson, M. A. *et al.* Spillover transmission is mediated by the excitatory GABA receptor LGC-35 in *C. elegans. J. Neurosci.* **35,** 2803–2816 (2015).
19. Garcia, L. R. & Sternberg, P. W. *Caenorhabditis elegans* UNC-103 ERG-like potassium channel regulates contractile behaviors of sex muscles in males before and during mating. *J. Neurosci.* **23,** 2696–2705 (2003).
20. Guo, X., Navetta, A., Gualberto, D. G. & Garcia, L. R. Behavioral decay in aging male *C. elegans* correlates with increased cell excitability. *Neurobiol. Aging* **33,** 1483.e5–1483.e23 (2012).
21. LeBoeuf, B. & Garcia, L. R. Cell excitability necessary for male mating behavior in *Caenorhabditis elegans* is coordinated by interactions between big current and ether-a-go-go family K^+ channels. *Genetics* **190,** 1025–1041 (2012).
22. Barr, M. M. *et al.* The *Caenorhabditis elegans* autosomal dominant polycystic kidney disease gene homologs *lov-1* and *pkd-2* act in the same pathway. *Curr. Biol.* **11,** 1341–1346 (2001).
23. Hobert, O., Moerman, D. G., Clark, K. A., Beckerle, M. C. & Ruvkun, G. A conserved LIM protein that affects muscular adherens junction integrity and mechanosensory function in *Caenorhabditis elegans. J. Cell Biol.* **144,** 45–57 (1999).
24. Bang, M. L. & Owczarek, S. A matter of balance: role of neurexin and neuroligin at the synapse. *Neurochem. Res.* **38,** 1174–1189 (2013).
25. Maćkowiak, M., Mordalska, P. & Wędzony, K. Neuroligins, synapse balance and neuropsychiatric disorders. *Pharmacol. Rep.* **66,** 830–835 (2014).
26. Pizzarelli, R. & Cherubini, E. Alterations of GABAergic signaling in autism spectrum disorders. *Neural Plast.* **2011,** 297153 (2011).
27. Chih, B., Engelman, H. & Scheiffele, P. Control of excitatory and inhibitory synapse formation by neuroligins. *Science* **307,** 1324–1328 (2005).
28. Maro, G. S. *et al.* MADD-4/punctin and neurexin organize *C. elegans* GABAergic postsynapses through neuroligin. *Neuron* **86,** 1420–1432 (2015).
29. Haklai-Topper, L. *et al.* The neurexin superfamily of *Caenorhabditis elegans*. *Gene Expr. Patterns* **11,** 144–150 (2011).

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