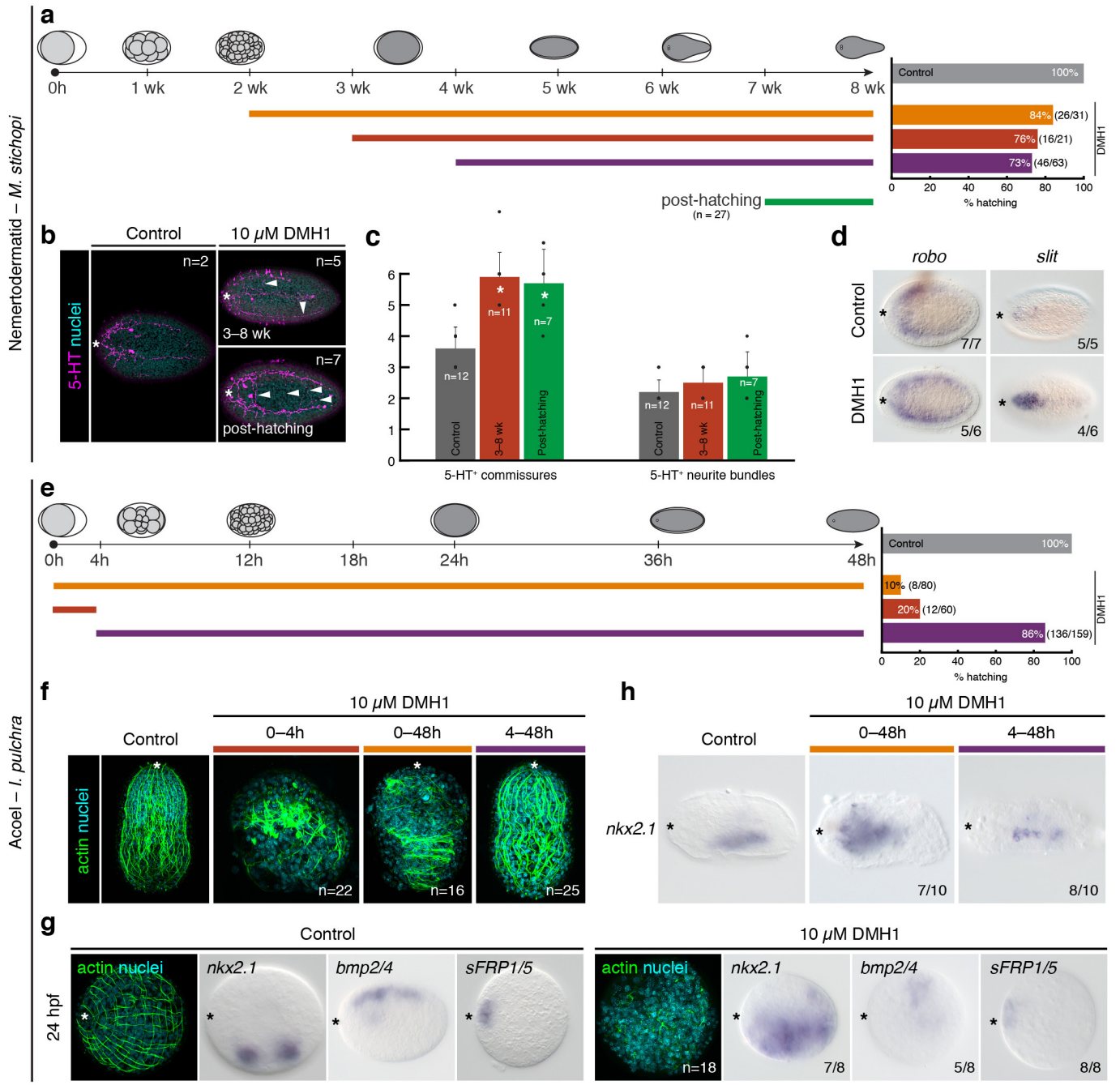

Extended Data Figure 6 | DMH1 treatments in *M. stichopi* and *I. pulchra*. **a**, Schematic overview of dorsomorphin homologue 1 (DMH1) treatments in *M. stichopi* and percentage of hatching embryos for each experimental condition. **b**, *M. stichopi* embryos incubated with DMH1 from 3 to 8 weeks and after hatching show more serotonergic commissures than control animals. **c**, The differences in the number of commissures are significant in both pre-hatching (asterisk; two-tailed *t*-test; $p<0.0001$) and post-hatching (asterisk; two-tailed *t*-test; $p<0.0014$) treated embryos. In contrast, the number of serotonin-positive neurite bundles is not significantly increased in any of the treatments. **d**, Despite the abnormal development of serotonergic axonal tracts, *slit* and *robo* genes are expressed similarly. The differences in signal intensity are due to technical variability. **e**, Schematic overview of DMH1 treatments in *I. pulchra* and the percentage of hatching embryos for each experimental condition. **f**, Morphological analyses of DMH1-treated embryos. Treatment in early stages affects normal development, whereas treatments from 4 h onwards do not significantly compromise embryogenesis. **g**, Embryos treated between 0 and 4 h post-fertilization and fixed at 24 h of development show expanded expression of the ventral marker *nkx2.1*, reduced expression of the dorsal gene *bmp2/4*, and unaffected expression of the anterior marker *sFRP1/5*. The embryo shows a disorganized morphology, as revealed by actin staining. **h**, The expression of the ventral marker *nkx2.1* is expanded in early treated embryos (0–48 h), but unaffected in embryos treated after 4 h of development. In **b**, **d**, **f**–**h**, the asterisk marks the anterior pole. In **b**, **d**, **f**, panels are dorsoventral views, and in **g** and **h** the panels are lateral views.