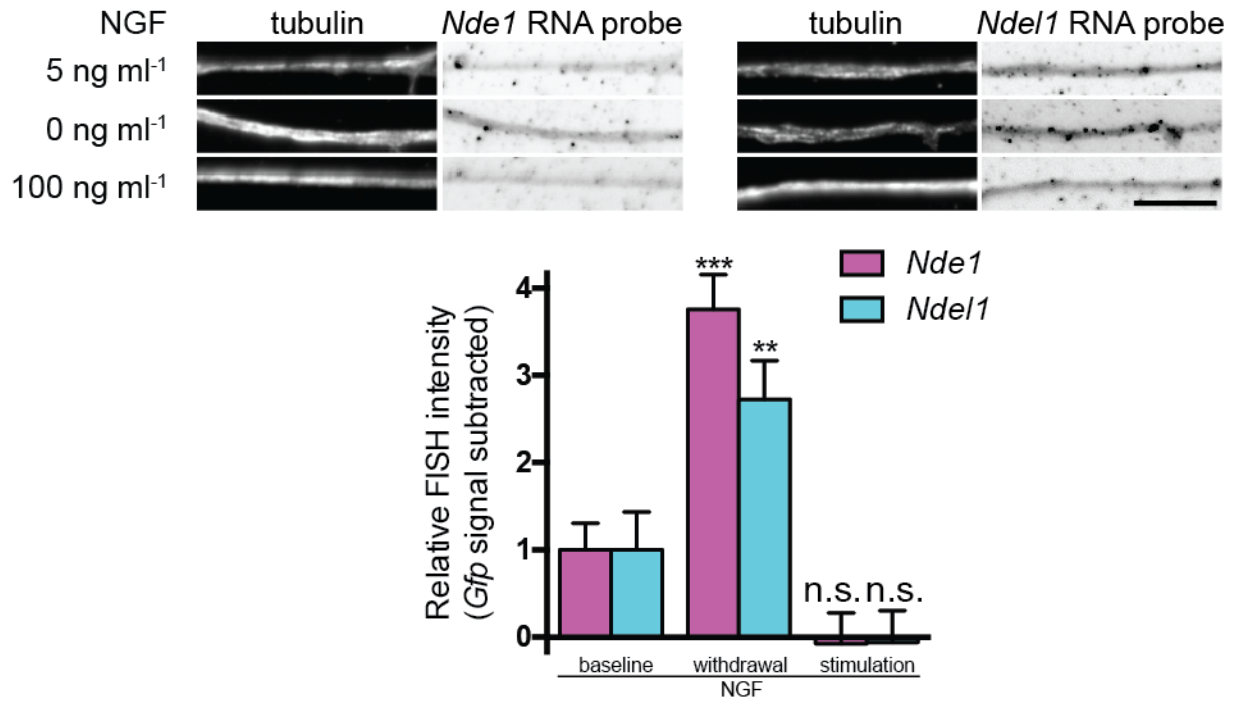


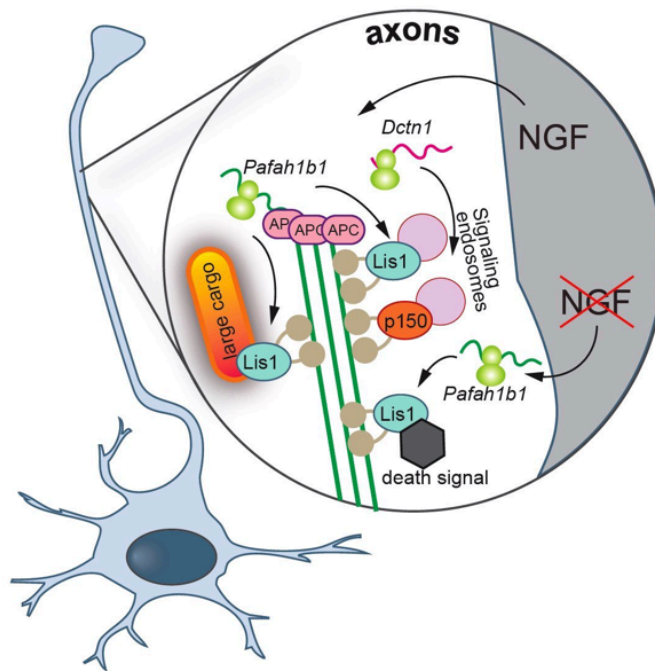
**Supplementary Figure 1. Validation of siRNAs**

C6 cells were transfected with each two siRNAs targeting *Pafah1b1* (A), *Dctn1* (B) and a non-targeting control siRNA for 72 hours before immunoblot analysis.



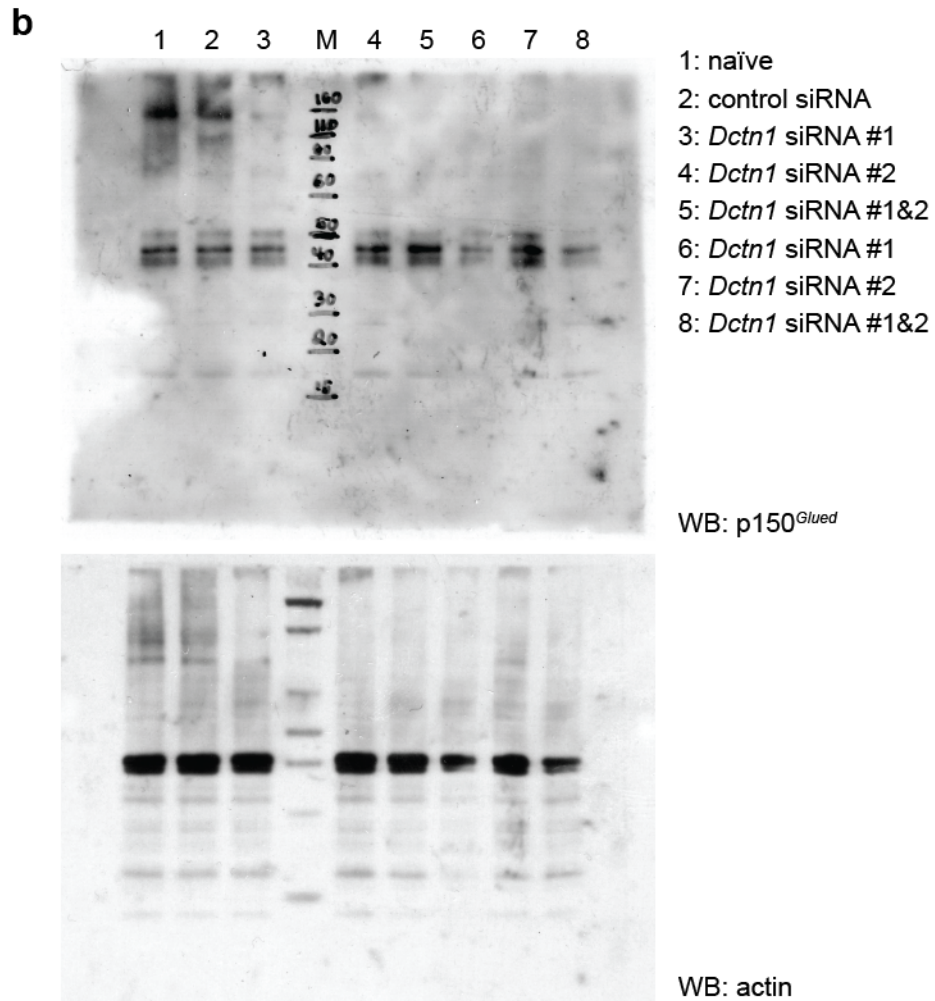
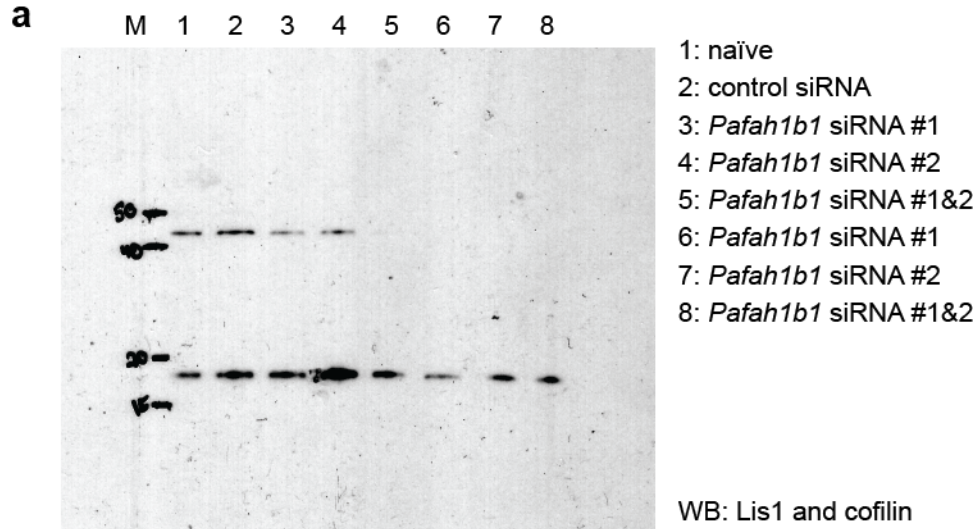
**Supplementary Figure 2. FISH for NudE and NudEL transcripts in axons**

DRG neurons were cultured in microfluidic chambers for 3 DIV, at which time the NGF concentration in the axonal chamber was changed to 5 ng ml<sup>-1</sup>. 24 h later, the NGF concentration in the axonal chamber was adjusted to 0, 5, or 100 ng ml<sup>-1</sup> NGF for 12 h, and axonal *Nde1* or *Ndel1* mRNA levels were determined by FISH. Means ± SEM of 45-60 optical fields per condition. \*\*, p≤0.01; \*\*\*, p≤0.001. One-way ANOVA. Scale bar, 10 μm.



**Supplementary Figure 3. Model for the NGF-dependent regulation of retrograde axonal transport through locally produced dynein cofactors**

NGF stimulation leads to the axonal translation of *Dctn1* and APC-bound *Pafah1b1*. Locally synthesized Lis1 required for the retrograde transport of vesicular cargoes greater than 1 μm in diameter, and both axonally-derived Lis1 and p150<sup>Glued</sup> are necessary for the retrograde transport of NGF-signaling endosomes. NGF withdrawal has no effect on p150<sup>Glued</sup> synthesis but causes the production of Lis1 off not APC-bound *Pafah1b1* transcripts. Lis1 produced in response to NGF withdrawal is required for the retrograde transport of a death signal.



**Supplementary Figure 4. Full size images of Western Blots**  
Full size scans of immunoblots presented in supplementary Fig. 1.