In a study, Sanz et al. identified a metalloproteinase-dependent mechanism necessary to promote growth in embryonic dorsal root ganglion cells (DRGs). Treatment of embryonic DRG neurons with pan-metalloproteinase inhibitors, tissue inhibitor of metalloproteinase-3, or an inhibitor of ADAM Metallopeptidase Domain 10 (ADAM10) reduces outgrowth from DRG neurons indicating that metalloproteinase activity is important for outgrowth. The IgLON family members Neurotrimin (NTM) and Limbic System-Associated Membrane Protein (LSAMP) were identified as ADAM10 substrates that are shed from the cell surface of DRG neurons. Overexpression of LSAMP and NTM suppresses outgrowth from DRG neurons. Furthermore, LSAMP loss of function decreases the outgrowth sensitivity to an ADAM10 inhibitor. Together our findings support a role for ADAM-dependent shedding of cell surface LSAMP in promoting outgrowth from DRG neurons [1].

1)
Sanz RL, Ferraro GB, Girouard MP, Fournier AE. Ectodomain shedding of Limbic System-Associated Membrane Protein (LSAMP) by ADAM Metallopeptidases promotes neurite outgrowth in DRG neurons. Sci Rep. 2017 Aug 11;7(1):7961. doi: 10.1038/s41598-017-08315-0. PubMed PMID: 28801670.