Tumour suppressor merlin deficiency leads to the development of schwannomas, meningiomas, ependymomas and occur in a variety of cancers incl. glioblastomas and breast cancer. Using our in vitro model for schwannoma as a model for merlin-deficient tumours we have successfully detected and inhibited various receptors (PDGFR, IGF-IR, ErbB2/3) and signalling pathways (ERK, AKT, JNK) involved in schwannoma development using AZD6244, sorafenib, nilotinib, lapatinib and BEZ-235. Since, merlin-deficient tumour’s pathobiology involves multiple receptors and signalling pathways, all markers involved in tumour development must be revealed before best treatment could be established. Our previous experiments using phospho-RTK profiling and phospho-MAPK arrays showed that TAM family receptors (Tyro3, Axl and Mer) are overexpressed in schwannoma tissues. Axl is negatively regulated by merlin and positively regulated by E3 ubiquitin ligase CRL4DCAF1, a key regulator in schwannoma growth. We hypothesized that Axl is a good target to study in merlin-deficient tumours. Here, we demonstrated strong overexpression and activation of Axl receptor as well as its ligand Gas6 in human schwannoma primary cells compared to normal Schwann cells. We show that Gas6 is mitogenic and increases schwannoma cell-matrix adhesion and survival acting via Axl in schwannoma cells. Stimulation of the Gas6/Axl signalling pathway recruits Src, focal adhesion kinase (FAK) and NFkB. We showed that NFkB mediates Gas6/Axl-mediated overexpression of survivin, cyclin D1 and FAK, leading to enhanced survival, cell-matrix adhesion and proliferation of schwannoma. We conclude that Axl/FAK/Src/NFkB pathway is relevant in merlin-deficient tumours and is a potential therapeutic target for schwannoma and other merlin-deficient tumours.