Supplementary Figure 1. Mer inhibition impedes glioblastoma chemokinesis. Control (shControl) and Mer knockdown (shMer1B) A172 cells were plated in complete media at equal densities in the upper chamber of a transwell. Complete media was also used in the lower chamber of the transwell. Cells that successfully migrated through a polycarbonate membrane with 8 µm pores were stained and optic density measured. Bar graph represents migration relative to shControl cells. Mean values and standard error were derived from three independent experiments. Means were statistically compared with a Student's paired t-test. P=0.0073.

Supplementary Figure 2. Viability of glioblastoma cells is not affected by Mer or Axl knockdown over the short experimental timepoint. **(a)**Control (shControl), Mer knockdown (shMer1A, shMer1B), and Axl knockdown (shAxl9, shAxl8) A172 cells were serum deprived for three hours, plated at equal density in serum replete media, and then evaluated for viability at 21 hours mimicking the conditions and timecourse of the transwell migration assays. Mean values and standard error were derived from three independent experiments. Means were statistically compared with a Student's paired t-test and there were no statistically significant differences amongst any of the groups.

Supplementary Figure 3. Axl RTK inhibition does not alter FAK activation. **(a)**Immunoblot of phospho-FAK activation in theA172 glioblastoma cell line following introduction of a doxycycline inducible shRNA against a non-targeting vector (NTV) or Axl (i-shAxl). i-shAxl(-) represents the clone was not treated with doxycycline (Axl

expression intact) while i-shAxl(+) was doxycycline treated (Axl expression inhibited). Cells were serum starved for 2 hours then treated with complete media replacement (Serum+), left untreated (Serum-, Gas6-), or treated with 200nM Gas6 for 10 minutes (Serum -, Gas6 +). Tubulin shown as a protein loading control.