Zhang et al., found that TRIM8 activates STAT3 by suppressing the expression of PIAS3, an inhibitor of STAT3, most likely through E3-mediatiated ubiquitination and proteasomal degradation. Interestingly, they also found that STAT3 activation upregulates TRIM8, providing a mechanism for normalized TRIM8 expression in the setting of hemizygous gene deletion. These data demonstrate that bidirectional TRIM8-STAT3 signaling regulates stemness in GSC [1].

1)
Zhang C, Mukherjee S, Tucker-Burden C, Ross JL, Chau MJ, Kong J, Brat DJ. TRIM8 regulates stemness in glioblastoma through PIAS3-STAT3. Mol Oncol. 2017 Jan 18. doi: 10.1002/1878-0261.12034. [Epub ahead of print] PubMed PMID: 28100038.