Cutaneous squamous cell carcinoma (cSCC), a form of non-melanoma skin cancer, is the second most common form of cancer in the United States, with over one million cases diagnosed annually. *Tpl2*, a gene in the mitogen-activated protein kinase (MAPK) family, can function as a tumor suppressor gene in skin cancer. Mice with a *Tpl2* deletion have both an increased incidence of skin carcinogenesis and increased propensity for tumor metastasis. This study investigates whether stromal-epithelial interactions and alterations in c-MET signaling are necessary to drive skin cancer development and progression in *Tpl2*−/− mice, thus contributing to our understanding of the mechanisms by which Tpl2 acts as a tumor suppressor gene in skin cancer.