Losses of the **tumor suppressor (2/3) BIN1 (2/3)** in **breast (1) carcinoma (5)** are frequent and reflect deficits in **programmed cell death (6)** capacity. Oncogenic activation of **MYC (2/3)** occurs often in breast carcinoma and is associated with poor prognosis. Loss or inactivation of mechanisms that restrain MYC may therefore be involved in **tumor progression (6)**. In this study, we show that the MYC-interacting adaptor protein BIN1 is frequently missing in **malignant breast cells (1)** and that this loss is functionally significant. BIN1 was expressed in **normal and benign cells and tissues (1)** but was undetectable in 6/6 **estrogen receptor-positive or estrogen receptor-negative carcinoma cell lines (1)** examined. Similarly, complete or partial losses of BIN1 were documented in 30/50 (60%) cases of **malignant breast tissue (1)** analyzed by **immuno-histochemistry (10)** or **RT-PCR (10)**. Abnormalities in the organization of the BIN1 gene were apparent in only a minority of these cases, suggesting that most losses were due to epigenetic causes. Nevertheless, they were functionally significant because ectopic BIN1 induced programmed cell death in malignant cells lacking endogenous BIN1 but had no effect on the viability of benign cells. We propose that loss of BIN1 may contribute to **breast cancer progression (6)** by eliminating a mechanism that restrains the ability of activated MYC to drive **cell division (6)** inappropriately.