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stathmin is an f-actin destabilizing factor that is rapidly and transiently overexpressed in response to cellular stress and may act as a trigger of cell cycle arrest or apoptosis by reducing the growth inhibitory phosphorylation of prb (retinoblastoma) by cyclin-dependent kinases. it is also involved in microtubule assembly and functions as a molecular switch for the reorganization of the cytoskeleton during mitosis and cytokinesis. however, high levels of stathmin have also been associated with metastatic progression of cancer cells, suggesting that stathmin promotes tumor progression and metastasis by triggering cellular programs that are important in cancer cell migration and adhesion. stathmin has also been associated with aggressive breast cancer, in which it is a strong predictor of disease recurrence and represents an independent marker for unfavorable prognosis it has been a challenge to establish cell death in hdr and ddr conditions. so far, only a few cell death markers have been found to be upregulated, such as p19arf, and the marker for caspase-3 cleavage, casp3 cleaved, seen in supplementary fig. 1. an in depth analysis, including the use of additional quantitative methods and monitoring of apoptosis by annexin-v and 7aad staining, as well as immunoblotting for p19arf, casp3 cleaved, cytokeratin 18 fragments (ck18-f) and the necrosis marker lactate dehydrogenase (ldh) is ongoing. we are also investigating if non-cleaved casp3, also known as pro-caspase 3, that also occurs in ddr cells, is a more relevant marker. when read-out, we will therefore have to consider the significance of the cleaved and the non-cleaved forms.

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