

Silencing of astrin induces the p53-dependent apoptosis by suppression of HPV18 E6 expression and sensitizes cells to paclitaxel treatment in HeLa cells

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Astrin is a microtubule-associated protein and localizes with mitotic spindles in the M-phase. We silenced the expression of astrin protein and tested the cell viability in response to paclitaxel treatment in paclitaxel-sensitive and paclitaxel-resistant cells. We found that the absence of astrin by siRNA resulted in the activation of a p53-dependent apoptosis, which elevated pro-apoptotic Bax expression and increased the activity of caspase-3 in astrin-depleted cells. The HPV18 E6 transcription was found to be inhibited along with the increase expression of p53. Intriguingly, the expression of astrin decreased in paclitaxel-sensitive HeLa cells but remained steady in paclitaxel-resistant cells in response to paclitaxel treatment. Furthermore, we identified that the depletion of astrin caused more cell death both in paclitaxel-sensitive and -resistant cells in combination with paclitaxel treatment. These findings suggest that the silencing of astrin induce a p53-dependent apoptosis and has an additive effect on paclitaxel treatment.