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**Nokia Flashing Cable Driver 8.47.0.**

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nokia flashing cable driver 8.47.0.Nokia-Flashing-Cable-Driver-v8.47.0.2014. cell phosphorylation ([@B57]; [@B16]). Moreover, the MEK pathway has been shown to be required for induction of neuronal plasticity ([@B32]; [@B4]). In addition, the presence of a cross-talk between the Hippo pathway and the MEK/ERK signaling pathway has been shown to be essential for proper neuronal morphogenesis. Indeed, although *Drosophila* neurons lacking Hpo can develop, their axonal and dendritic domains are small, which is also the case for those generated upon ERK1/2 overexpression ([@B24]). Although *hpo* mutants display similar brain overgrowth as our ctA-expressing *hpo*<sup>RNAi</sup> cells, *Drosophila* neurons developed with less prominent endomitotic cell divisions and frequently extended their axonal extensions throughout the brain ([@B26]). In conclusion, our data provide evidence

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that oncogenic \*CTK-DD\* drives the Notch pathway in the brain of \*Drosophila\* via Hpo pathway activation. Moreover, neuronal ectopic expression of \*ctk\* could promote the recruitment of an EMT program in these cells that in turn induces cell invasion. The absence of transcriptional and/or post-transcriptional regulation of \*ctk\* in the developing brains suggests that it could promote tumorigenesis by increasing the availability of the Notch ligand. The accumulation of activated Notch could then induce endomitotic divisions in close proximity to the tumor and promote cell proliferation and invasion. Therefore, the tumor suppressor \*ctk\* could provide a feedback mechanism controlling Notch activity in a niche where oncogenic proliferation and invasion occur. Indeed, the expression of \*hpo\* has been reported to be altered in human breast cancer and to be able to suppress tumorigenesis by inhibiting tumor

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invasion ([@B57]). Our results suggest that this pathway could be implicated in the suppression of tumor invasion. Therefore, it would be interesting to determine whether tumor cells driven by oncogenic *ctk* expression display alterations in *hpo* expression. Author Contributions

===== AM, AP, AL, and LP planned the experiments, carried out the research, analyzed the data, and wrote the manuscript. AB



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