# Retraction Note: APP binds DR6 to trigger axon pruning and neuron death via distinct caspases

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The authors have retracted this article[1]. Our subsequent work confirmed aspects of the article, notably that DR6 and APP interact and function in a genetic pathway involving caspases to control axon pruning and neuron death[2,3]. However, our later research also showed that certain conclusions reached in the article were incorrect, notably the role of caspase-3, the necessity for beta-secretase enzyme activity for APP-DR6 binding, and the model for the APP-DR6 interaction[2,3].

More recently, the following anomalies were identified:

- Figure 1d: the NGF-deprived +IgG panel appears to be identical to the NGF-deprived, 24h + Control IgG panel of Figure 5e.
- Supplementary information Figure 9c: the NGF-deprived + Bax inhibitor control panel appears to be identical to the + anti-NGF control panel of Supplementary information Figure 17c.
- Supplementary information Figure 6d: the fourth beta-Actin blot for Casp-3 siRNA appears to be identical to the first beta-Actin blot for Casp-6 siRNA.
- Certain biostatistical calculations underlying some figures contained errors.

We believe that these additional anomalies do not affect the conclusions presented in the affected figures. However, given the lack of original data for several of these figures due to the age of the paper, and since our subsequent research showed that certain specific claims in the original article were not correct and we reported a correction for those claims elsewhere[2,3], we consider that the appropriate course of action is to retract the article. All the authors agree with this retraction.

1. Nikolaev, A. et al. APP binds DR6 to trigger axon pruning and neuron death via distinct caspases. *Nature* **457**, 981–989 (2009).
2. Simon, D. J. et al. A caspase cascade regulating developmental axon degeneration. *J. Neurosci.* **32**, 17540–17553 (2012).
3. Olson, O. et al. Genetic analysis reveals that Amyloid Precursor Protein and Death Receptor 6 function in the same pathway to control axonal pruning independent of β-secretase. *J. Neurosci.* **34**, 6438–6447 (2014).