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CORRECTION

Correction to: Dying to communicate: apoptotic functions of Eph/Ephrin proteins

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Correction to: Apoptosis (2018) 23:265–289
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The original version of this article contained a mistake in reference. The references in Table 1 are incorrect. The corrected table with proper citation is given below.

The field codes ADDIN REFMGR.CITE inadvertently appeared along the article. This was overlooked during the process.

The original article can be found online at https://doi.org/10.1007/s10495-018-1458-7.

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Table 1 In vivo genetic models with reported specific functions of Ephs and Ephrins in cell death

Gene(s)	Genetic model	Phenotype	References
EphrinB3	EphrinB3 knockout mice	Increase in cell proliferation along the lateral ventricle and greater numbers of cell death in the SVZ	(50)
EphrinA5/EphrinA2	EphrinA5/EphrinA2 Double knockout mice	Neural epithelial cell death reduced in embryos. Expression EphrinA5-Fc or full-length EphrinA5 strongly induced apoptosis in neural epithelial cells and a severe brain malformation during embryonic development	(24)
EphrinA5/Caspase3	EphrinA5 transgenic/Caspase3 knockout mice	In vivo deficiency of Caspase3 attenuated apoptosis in animals overexpressing EphrinA5	(24)
EphrinB3/EphA4	EphrinB3 and EphA4 knockout mice	EphrinB3 deletion increases cell death. Absence of EphA4 results in excessive numbers of neuroblasts	(27)
EphrinA5/EphA7	EphrinA5 transgenic mice	Gain of EphrinA5 in early cortical progenitors expressing EphA7 depleted the cortex of neural progenitor cells that underwent apoptosis	(22)
EphrinA5/EphA7	EphA7 knockout mice	EphA7 gene disruption resulted in a reduction in apoptosis in forebrain neural progenitors, and in an increase in the cortical size	(22)
EphrinA5/EphA8	EphA8-Fc transgenic mice	Ectopic expression of EphA8-Fc induced apoptosis of EphrinA5-expressing neural epithelial cells, with a dramatic decrease in brain size	(49)
EphrinA2	EphrinA2 knockout mice	EphrinA2 deficiency promotes the proliferation of progenitor cells and an increased neurogenesis in the adult mouse olfactory bulb	(23)
EphrinB3/EphB3	EphrinB3 and EphB3 knockout mice	In either EphrinB3(−/−) or EphB3(−/−) mice, proliferation is increased in the SVZ. However, cell death is reduced in EphB3(−/−) but increased in EphrinB3(−/−) mice	(29)
EphrinB3	EphrinB3 knockout mice	EphrinB3 genetic ablation resulted in enhanced cell proliferation and neuronal differentiation around the lesion site upon induction of stroke	(62)
EphrinB3/EphB3	EphB3 knockout mice	EphB3/EphrinB3 interactions modulate oligodendrocyte (OL) cell death in injured spinal cord. Either EphB3 ablation or EphrinB3 administration after spinal cord injury (SCI) promotes OL survival	(65)
EphA2	EphA2 knockout mice	EphA2 has a protective role in blood–brain barrier damage and neuronal death following ischemic stroke-induced brain inflammation. EphA2(−/−) brains had significantly lower apoptosis as compared to WT group	(66)
EphrinA5	EphrinA5 transgenic mice	Massive apoptosis in both the nasal and temporal retinas	(92)
EphB2/EphB3	$EphB2^{-/-}$ and $EphB3^{-/-}$ knockout mice. Transgenic mice for EphB2 truncated in the C-terminus	Both EphB2 and EphB3 null mutant mice exhibited more severe axonal degeneration than the wild type in a model of laser-induced ocular hypertension (LIOH) for experimental glaucoma	(100)
EphrinB2	Knock-in mice with a targeted mutation of five tyrosine residues ($EphrinB2^{5Y/5Y}$) in the cytoplasmic tail	Tyrosine phosphorylation of EphrinB2 is critical for the regulation of endothelial cell death and vessel pruning in the eye vasculature	(111)
EphB2/EphB3	EphB2 and/or EphB3 simple and double knockout mice	Thymic hypocellularity correlated with increased apoptotic cells	(129)
EphB2/EphB3	EphB2 and/or EphB3 simple and double knockout mice. Mice transgenic for a truncated EphB2, devoid of the cytoplasmic domain	EphB2 contributes more significantly than EphB3 in the control of DP thymocytes' progression. In animals with EphB2-, EphB3- or EphB2/B3-deficient progenitors, apoptosis was observed mainly in the DP and SP–CD4 cells. This role is EphrinB-dependent	(130)

Table 1 (continued)

Gene(s)	Genetic model	Phenotype	References
EphA4	EphA4 knockout mice	EphA4-deficiency resulted in the alteration of thymocyte maturation, leading to defective T cell development. The thymi have lower double-positive (CD4+CD8+) cells, block of T cell precursor differentiation, decreased cell proliferation and increased cell apoptosis	(125)
EphB4	EphB4 transgenic mice	Development of the mammary epithelium at puberty and during pregnancy was delayed (e.g. less lobules during pregnancy). Kinetic of epithelial DNA synthesis and apoptotic cell death during pregnancy was perturbed at early post-lactational involution	(139)
EphrinB2	EphrinB2 knockout mice	Severe architecture defects. Lactating glands in EphrinB2-deficient mice resembled that of involuting controls, with overall decreased epithelial cell number, and collapsed lobulo-alveolar structures, because of massive epithelial cell death overpowering a concomitant significant cell proliferation	(140)
EphrinB2	EphrinB2 knockout mice	EphrinB2 deficiency results in early osteoblast and osteocyte apoptosis which appears to be responsible for the observed reduced bone strength and impaired anabolic response to PTH, and their response to bone anabolic effect of the parathyroid hormone (PTH)	(166)
EphB2/EphB3	EphB2 and EphB3 knockout mice. Mice transgenic for a truncated EphB2, devoid of the cytoplasmic domain	Reduced Proliferation in Colon Crypts in Mice Lacking EphB2 and EphB3. Apoptosis not significantly affected	(185)
EphA2	EphA2 knockout mice	EphA2 deficiency inhibits tumor growth and induces apoptosis in a model of Kras-mutant non-small cell lung cancer (NSCLC)	(176)
EphrinB2	EphrinB2 knockout mice	Inhibition of EphrinB2 function results in a disruption of mammary epithelial cell–cell contacts leading to concomitant apoptosis and proliferation	(140)