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Corrigendum

The role of CD95 and CD95 ligand in cancer

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Since the publication of this article the authors have noted that they have inadvertently failed to list an important paper in the field in Table 2. This work (Kleber *et al.*) has now been added to the revised Table 2 shown below. The authors wish to apologize to these authors for the omission.

Table 2 Tumor promoting activities of CD95 and CD95L *in vitro* and *in mouse* models

Cancer type	Observation	Reference
Multiple cancers	Stimulation of 22 breast, ovarian, lung, colon, renal, melanoma, or glioblastoma cancer cell lines through CD95 causes them to increase in motility and invasiveness, and by activating NF-kB and MAP kinase pathways and upregulation of uPA	Barnhart *et al.*[64]
	Knockdown of either CD95 or CD95L resulted in reduced growth of ovarian, liver, colon, breast cancer cell lines *in vitro* and of ovarian cancer cell lines in xenografted mice	Chen *et al.*[44]
	In lung cancer, GBM, and hepatocellular carcinoma cell lines CD95L increased motility and cell growth through binding to c-Met	Lin *et al.*[177]
	Knockdown of either CD95 or CD95L resulted in induction of cell death in 12 cancer cell lines representing ovarian, liver, breast, cervical, colon, renal cancer, neuroblastoma, or glioblastoma	Hadji *et al.* [119]
	Stimulation of CD95 on breast, ovarian, renal, colon cancer, and glioblastoma cell lines increases cancer stemness	Ceppi *et al.* [115]
Breast cancer	Stimulation of CD95 on triple negative breast cancer cells by soluble CD95L resulted in Yes/Orai1/EGFR/PI3K mediated migration	Malleter *et al.* [82]
	Blockade of CD95 signaling in 4T1 cancer cells markedly reduced tumor growth, inhibited tumor metastasis *in vivo*, and prolonged survival of tumor-bearing mice	Liu *et al.*[178]
Colon cancer	Expression of CD95L on colon cancer cells greatly increased their local growth and ability to metastasize to the liver	Li *et al.*[150]
	CD95 driven liver metastasis of CD95 stimulated colon cancer cells is dependent on oncogenic Kras	Hoogwater *et al.*[70]
	Radiofrequency ablation of colorectal liver metastases induces hypoxia that causes autocrine activation of CD95 promoting local invasion and accelerated metastasis outgrowth	Nijkamp *et al.*[75]
	CD95 triggering resulted in an increased metastatic ability and activation of EMT in cells resistant to oxaliplatin	Ametller *et al.*[125]
	CD95 stimulation induced phosphorylation of phospholipase C-g1 through the platelet-derived growth factor receptor-b, resulting in phosphatidylinositol (4,5)-bisphosphate (PIP2) hydrolysis, liberating cofilin from the plasma membrane to initiate cortical actin remodeling in turn increasing tumor cell invasion	Steller *et al.*[81]
Gastrointestinal cancer	CD95 stimulation induced ERK1/2 driven EMT and motility	Zheng *et al.*[112]
Glioblastoma	Stimulation of CD95 by CD95L induced invasiveness through recruitment of the Yes src kinase and the p85 subunit of phosphatidylinositol 3-kinase to CD95, resulting in activation of GSK3β and subsequent expression of matrix metalloproteinases	Kleber *et al.*[74]
Hepatocellular carcinoma	Neutralizing CD95L in a transgenic model of hepatocellular carcinogenesis reduced both inflammation and tumor formation	Nakamoto *et al.*[179]
	Mice with a point mutation in the CD95 DD expressed only on nonhematopoietic cells developed spontaneous liver cancer independent of the lack of apoptosis induction through CD95	Park *et al.*[180]
	Mice with tissue specific deletion of CD95 in hepatocytes showed a 50% reduced occurrence of DEN induced liver cancer	Chen *et al.*[44]
Histiocytic sarcoma	Cancer formed in the liver of mice engineered to express only soluble and lacking expression of membrane bound CD95L	La *et al.*[78]
Lung cancer	CD95 overexpressing Lewis lung carcinoma (3LL) cells grew faster *in vivo* in syngeneic mice when compared with control transfected cells	Lee *et al.*[68]
	CD95 ligation-induced 3LL cells to produce the proinflammatory factor PGE2 by activating p38 contributing to CD95 ligation-induced chemoattraction of myeloid-derived suppressor cells	Zhang *et al.*[69]
	CD95 mediated activation of NF-kB was found to contribute to the resistance of lung cancer to a EGFR tyrosine kinase inhibitor	Bivona *et al.*[79]
Melanoma	Stimulation of B16 cells by exosome-derived CD95L *in vitro* activates NF-kB and ERK, and *in vivo* increases migration to the lung	Cai *et al.*[181]
Ovarian cancer	Mice lacking expression of CD95 in the surface epithelial cells of the ovaries barely developed cancer in a mouse model of endometrioid ovarian cancer driven by oncogenic *Kras* and deletion of *pten*	Chen *et al.*[44]
	Tissue specific deletion of CD95 in the ovaries resulted in an increase in inflammation in the ovaries and reduced tumor development in a model of low grade ovarian cancer driven by oncogenic *Kras* and deletion of *pten*. All outgrowing cancer cells still expressed at least one allele of wt *CD95*	Hadji *et al.*[119]
Pancreatic cancer	Stimulation of TRAF2 overexpressing cells resulted in increased invasiveness by activating NF-kB and AP-1 resulting in upregulated uPA	Trauzold *et al.*[73]
	Stimulation of CD95 on FADD knockdown cell lines mediates cell survival by recruiting calmodulin and Src resulting in activation of ERK	Yuan *et al.*[72]
	CD95 was identified as upregulated on cancer stem cells driving cell cycle progression by using Sck. Invasiveness and tumor growth could be inhibited *in vivo* by blocking CD95L	Teodorczyk *et al.*[83]
Thyroid cancer	Stimulation of CD95 induced cell growth through ERK, NF-kB, and AP-1	Mitsiades *et al.*[182]