

CORRECTION



Correction: CEACAM1 impedes thyroid cancer growth but promotes invasiveness: a putative mechanism for early metastases

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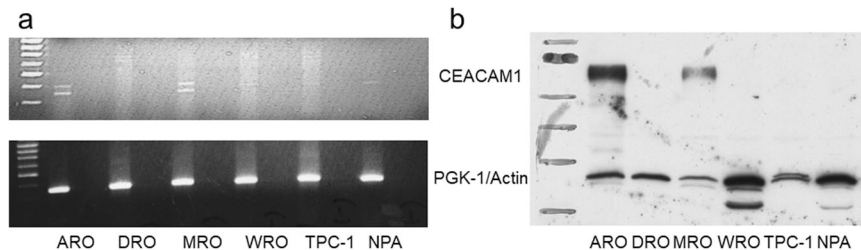
Oncogene (2023) 42:3159–3160; <https://doi.org/10.1038/s41388-023-02833-0>

Correction to: *Oncogene* <https://doi.org/10.1038/sj.onc.1210077>, published online 23 October 2006

The authors are issuing a correction for the above article. In Figure 1a, b, the original experiments included the DRO cell line which was thought to be a thyroid cancer cell line. At the time of publication in 2006, the authors had just been informed that DRO cells were not thyroid cancer. This information was subsequently published (Schweppe RE et al., *J. Clin. Endocrinol. Metab.* 2008 93:4331–4341 originally published online Aug 19, 2008; <https://doi.org/10.1210/jc.2008-1102>). On receiving this information immediately prior to submission of the manuscript, the authors removed the data on the DRO cells. Figure 1a, b are being replaced by the original data with the recognition that

DRO cells are not thyroid cancer cells. The overall results and conclusions are not affected by the change.

Figure 1a, b. CEACAM1 is expressed in human thyroid carcinoma-derived cell lines. (a) RNA extracted from human thyroid carcinoma-derived cell lines as well as the DRO cell line was PCR-amplified using primers for CEACAM1. Products consistent with CEACAM1-L (408 bp) and the shorter CEACAM1-S (355 bp) form were identified in ARO anaplastic and MRO follicular carcinoma cells. The lower panel represents the PGK-1 control. Each lane is followed by the same reaction with omission of the reverse transcriptase. (b) Western immunoblotting of corresponding cell lysates demonstrates CEACAM1 expression in the ARO and MRO cell lines. The actin control is shown below.



During investigation of this paper in 2016, the original blots for actin in Figure 2a could not be found. However, the blots for CEACAM1 include the residual loading control for actin in the lower bands as shown here. These clearly show the appropriate loading of protein, confirming the accuracy of the data.

Figure 2a. CEACAM1 impacts different parameters of thyroid cancer cell growth. Forced expression of CEACAM1 in deficient WRO cells (left) and downregulation using stable siRNA in CEACAM1-expressing MRO cells (right) was established in multiple stable clones.

