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Corrigendum

ING1 induces apoptosis through direct effects at the mitochondria

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The ING family of tumor suppressors acts as readers and writers of the histone epigenetic code, affecting DNA repair, chromatin remodeling, cellular senescence, cell cycle regulation and apoptosis. The best characterized member of the ING family, ING1, interacts with the proliferating cell nuclear antigen (PCNA) in a UV-inducible manner. ING1 also interacts with members of the 14-3-3 family leading to its cytoplasmic relocalization. Overexpression of ING1 enhances expression of the *Bax* gene and was reported to alter mitochondrial membrane potential in a p53-dependent manner. Here we show that ING1 translocates to the mitochondria of primary fibroblasts and established epithelial cell lines in response to apoptosis inducing stimuli, independent of the cellular p53 status. The ability of ING1 to induce apoptosis in various breast cancer cell lines correlates well with its degree of translocation to the mitochondria after UV treatment. Endogenous ING1 protein specifically interacts with the pro-apoptotic BCL2 family member BAX, and colocalizes with BAX in a UV-inducible manner. Ectopic expression of a mitochondria-targeted ING1 construct is more proficient in inducing apoptosis than the wild type ING1 protein. Bioinformatic analysis of the yeast interactome indicates that yeast ING proteins interact with 64 mitochondrial proteins. Also, sequence analysis of ING1 reveals the presence of a BH3-like domain. These data suggest a model in which stress-induced cytoplasmic relocalization of ING1 by 14-3-3 induces ING1-BAX interaction to promote mitochondrial membrane permeability and represent a paradigm shift in our understanding of ING1 function in the cytoplasm and its contribution to apoptosis.

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Since the publication of this article, the authors have noted errors in the abstract section. In the sentence beginning 'ING1 also interacts' the text in brackets '(members of the Tetratrico Peptide Repeat (TPR) superfamily)' has been removed. The word 'we' has been removed before 'established epithelial cell lines in response to apoptosis-inducing stimuli, independent of the cellular p53 status'.

The correct abstract is shown above. The article with the corrected abstract appears online together with this corrigendum.

The authors would like to apologize for any inconvenience.