



Corrigendum

The impairment of HCCS leads to MLS syndrome by activating a non-canonical cell death pathway in the brain and eyes

Alessia Indrieri, Ivan Conte, Giancarlo Chesi, Alessia Romano, Jade Quartararo, Rosarita Tatè, Daniele Ghezzi, Massimo Zeviani, Paola Goffrini, Ileana Ferrero, Paola Bovolenta & Brunella Franco

DOI 10.15252/emmm.201470060

Correction to: EMBO Mol Med (2013) 5: 280-293. DOI 10.1002/emmm.201201739

In the above article, holo-cytochrome c-type synthase was used instead of holocytochrome c-type synthase, the official gene name. This error occurs in three places in the text and the correct sentences should read:

In the abstract:

Now we provide the evidence that non-canonical mitochondrial-dependent apoptosis explains the phenotype of microphthalmia with linear skin lesions (MLS), an X-linked developmental disorder caused by mutations in the holocytochrome c-type synthase (HCCS) gene.

In the introduction:

HCCS is a highly conserved gene from fungi to metazoans and encodes a mitochondrial holocytochrome c (Cytc)-type synthase, also known as 'heme lyase', located on the outer surface of the inner

mitochondrial membrane (Schaefer *et al*, 1996; Schwarz & Cox, 2002).

In 'The paper explained' section:

We demonstrate that inactivation of holocytochrome c-type synthase (HCCS), a transcript important for the mitochondrial respiratory chain (MRC), is associated with unconventional activation of caspase-9 in the mitochondria triggered by MRC impairment and overproduction of reactive oxygen species.



License: This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited