



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

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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* (Cyt_c)-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.



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