frontiers in Neurology

CORRECTION
published: 09 November 2021
doi: 10.3389/fneur.2021.792230

Corrigendum: A Novel Variation in the Mitochondrial Complex I Assembly Factor NDUFAF5 Causes Isolated Bilateral Striatal Necrosis in Childhood

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Keywords: bilateral striatal necrosis, NDUFAF5, mitochondrial complex I deficiency, whole-exome sequencing, novel variation

A Corrigendum on

A Novel Variation in the Mitochondrial Complex I Assembly Factor NDUFAF5 Causes Isolated Bilateral Striatal Necrosis in Childhood

by Bi, H., Guo, H., Wang, Q., Zhang, X., Zhao, Y., Li, J., Zhao, W., Tuo, H., and Zhang, Y. (2021). Front. Neurol. 12:922. doi: 10.3389/fneur.2021.675616

OPEN ACCESS

Approved by:
Frontiers Editorial Office, Frontiers Media SA, Switzerland

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Specialty section:
This article was submitted to Neurogenetics, a section of the journal Frontiers in Neurology

Received: *10 October 2021*
Accepted: *11 October 2021*
Published: *09 November 2021*

Citation:
Bi H, Guo H, Wang Q, Zhang X, Zhao Y, Li J, Zhao W, Tuo H and Zhang Y (2021) Corrigendum: A Novel Variation in the Mitochondrial Complex I Assembly Factor NDUFAF5 Causes Isolated Bilateral Striatal Necrosis in Childhood. Front. Neurol. 12:792230. doi: 10.3389/fneur.2021.792230

Error in Table

On a recent occasion, we realized that in the original article, there was a mistake in **Table 1** as published. **The citation numbers in the *Table 1* referring to the *NDUFAF5* mutations in various ethnic groups did not match the given reference order list in the published article. In Table 1, (1) reference 26 should be reference (1); (2) reference 27 should be reference 30; (3) reference 28 should be reference (2); (4) reference 12 should be reference 31; (5) reference 29 should be reference 32; and (6) reference 13 should be reference (3)**. The corrected ***Table 1*** appears below.

Missing Citation

In the original article **References 30, 31, and 32** were not cited/included in the published article. The citation has now been inserted in **Table 1**, under the section ***Discussion***.

New References to be Added in the continuing order:

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32. Gerards M, Sluiter W, van den Bosch BJ, de Wit LE, Calis CM, Frentzen M, et al. Defective complex I assembly due to C20orf7 mutations as a new cause of Leigh syndrome. *J Med Genet.* (2010) 47:507–12. doi: 10.1136/jmg.2009.067553

The authors apologize for this error and confirm that it does not change the scientific conclusions of the article in any way. The original article has been updated.

TABLE 1 | Clinical features of patients with NDUFAF5 variations reported in literature.

References	Ethnicity	Sex*	Mutation	Onset age	Clinical features	MRI findings	Outcome
Saada et al. (1)	Ashkenazi Jewish	M	c.749G>T, c.749G>T	12 m	Motor development retardation, ataxia, bilateral ptosis, optic atrophy, diffuse hypotonia	Symmetrical lesions of bilateral basal ganglia, striatum and cortical areas	Death at ∼2.5 y
	Ashkenazi Jewish	M	c.749G>T, c.749G>T	12 m			Death at ∼6 y
	Ashkenazi Jewish	F	c.749G>T, c.749G>T	12 m			Death at ∼4.5 y
	Ashkenazi Jewish	F	c.749G>T, c.749G>T	12 m			Death at ∼6 y
	Ashkenazi Jewish	F	c.749G>T, c.749G>T	12 m			Death at ∼7 y
Fang et al. (30)	Chinese		c.212C>T, c.698G>T		Developmental delay and regression, seizures	Bilateral lesions of brainstem and basal ganglia	
Sugiana et al. (2)	Egyptian	M	c.719T>C, c.719T>C	Birth	Intrauterine growth retardation, facial dysmorphism, corpus callosum agenesis, ventricular septation, left diaphragmatic hernia, adrenal insufficiency	–	Death at ∼7 d
Tong et al. (31)	Chinese	F	c.145C>G, c.836T>G	8 m	Neurodevelopmental delay, swallowing dysfunction, dyspnea	Bilateral medulla oblongata lesions	Death at 21 m
Gerards et al. (32)	Moroccan	M	c.477A>C, c.477A>C	3 y	Dysarthria, dystonic posture, spastic quadriplegia, mental retardation	Caudate, putamen, substantia nigra and peri-aqueductal grey area lesions, bifrontal atrophy	Alive at 23 y
	Moroccan	M	c.477A>C, c.477A>C	3 y			Alive at 29 y
Simon et al. (3)	Taiwanese	F	c.155A>C, c.836T>G	6 m	Developmental delay, global hypotonia, difficulty swallowing	Symmetrical thalamic and midbrain lesions, corpus callosum dysgenesis	Death at 27 m
	Taiwanese	F	c.836T>G, c.836T>G	27 m	Vision loss, strabismus, nystagmus, muscle weakness, inability to walk	Hyperintense lesions in posterior fossa, caudate and cervical spinal cord	Death at 19 y
	Caucasian	M	c.327G>C, c.223–907A>C	3 m	Seizures, hypotonia, loss of vision, feeding difficulty	T2 hyperintensity in thalamus, midbrain, upper spinal cord	Death at 8 m
	Ashkenazi Jewish	M	c.327G>C, c.749G>T	5 m	Torticollis, nystagmus, swallowing and feeding difficulty	Bilateral lesions in thalamus, putamen and frontal lobes	Death at 17 m
This pedigree	Chinese	F	c.425A > C, c.836T > G	6y	Generalized dystonia, spastic quadriplegia, dysphagia and dysarthria		Alive at 23 y
	Chinese	F	c.425A > C, c.836T > G	6y	Generalized dystonia, optic atrophy, dysphagia and dysarthria	Abnormal symmetric signals in the posterior region of the bilateral putamen	Alive at 20 y
	Chinese	F	c.425A > C, c.836T > G	6y	Generalized dystonia, febrile convulsions (1-3 y), dysphagia and dysarthria	Abnormal symmetric signals in the posterior region of the bilateral putamen	Alive at 18 y

M, Male; F, Female.

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