Human Molecular Genetics, 2019, Vol. 28, No. 7 1225

doi: 10.1093/hmg/ddy408
Corrigendum

CORRIGENDUM

SLP-2 interacts with Parkin in mitochondria and prevents mitochondrial dysfunction in Parkin-deficient human iPSC-derived neurons and *Drosophila*

Alessandra Zanon[1,†], Sreehari Kalvakuri[2,†], Aleksandar Rakovic[3,†], Luisa Foco[1], Marianna Guida[1], Christine Schwienbacher[1], Alice Serafin[1], Franziska Rudolph[3], Michaela Trilck[3], Anne Grünewald[3,4], Nancy Stanslowsky[5], Florian Wegner[5], Valentina Giorgio[6], Alexandros A. Lavdas[1], Rolf Bodmer[2], Peter P. Pramstaller[1,7,8], Christine Klein[3,*], Andrew A. Hicks[1,*], Irene Pichler[1,†] and Seibler Philip[3,†]

[1]Institute for Biomedicine, Eurac Research, Affiliated Institute of the University of Lübeck, 39100 Bolzano, Italy, [2]Development, Aging and Regeneration Program, Sanford Burnham Prebys Medical Discovery Institute, La Jolla, CA 92037, USA, [3]Institute of Neurogenetics, University of Lübeck, 23562 Lübeck, Germany, [4]Molecular and Functional Neurobiology Group, Luxembourg Centre for Systems Biomedicine, University of Luxembourg, L-4367 Belvaux, Luxembourg, [5]Department of Neurology, Hannover Medical School, 30625 Hannover, Germany, [6]University of Padova, 35122 Padova, Italy, [7]Department of Neurology, General Central Hospital, 39100 Bolzano, Italy and [8]Department of Neurology, University of Lübeck, 23562 Lübeck, Germany

*To whom correspondence should be addressed at: Institute for Biomedicine, Eurac Research, Via Luigi Galvani 31, 39100 Bolzano, Italy. Tel: +39 0471055521; Fax: +39 0471055599; Email: andrew.hicks@eurac.edu (A.A.H.); Institute of Neurogenetics, University of Lübeck, Maria-Goeppert-Straße 1, 23562 Lübeck, Germany. Tel: +49 451 31018200; Fax: +49 451 31018204; Email: christine.klein@neuro.uni-luebeck.de (C.K.)

Human Molecular Genetics, 2017, 26(13), 2412–2425
doi: 10.1093/hmg/ddx132.

The authors of the article "SLP-2 interacts with Parkin in mitochondria and prevents mitochondrial dysfunction in Parkin-deficient human iPSC-derived neurons and Drosophila" would like to report the incorrect description of an iPSC line in the paper.

Authentication analysis at Coriell Institute for Medical Research (Camden, New Jersey) revealed that the cell line named iPS-HFF in the article was not derived from newborn foreskin fibroblasts (as reported in Table S2) but from cells of a female patient with osteogenesis imperfecta (GM17602/GM17604), carrier of a heterozygous missense mutation (p.G700C; NM_000089) in the COL1A2 gene. Repeated karyotype analysis performed for the line named iPS-HFF showed a normal female karyotype, which means that the male karyotype displayed in Figure S4B is mislabeled and was not derived from iPS-HFF.

Differentiated dopaminergic neurons from the line named iPS-HFF were examined for mitochondrial complex I activity (Figure 4A) and mitochondrial morphology (Figure 4B) as one of in total three analyzed control iPSC lines compared to Parkin mutant lines.

Importantly, both experiments display robust phenotypes and are replicative analysis of results shown in Parkin knockdown SH-SY5Y cells: Loss-of-Parkin leads to decreased complex I activity (Figure 2B) and an increased amount of fragmented mitochondria (Figure 2D), while overexpression of SLP-2 rescues both phenotypes. This confirms that the analyzed data in Figure 4 are not influenced by the COL1A2 mutation. Furthermore, mutations in the COL1A2 gene have not been associated with neurodegenerative diseases and the symptoms of neurological movement disorders have not been described for osteogenesis imperfecta.

Therefore, the fact that this particular cell line is from a diseased individual with osteogenesis imperfecta rather than a healthy control does not alter the interpretation of our results as pertaining to rescue of mitochondrial phenotypes as described in the manuscript. The authors would like to apologize for this mistake.