

Letters to the Editor:

**Reply to Jawaid *et al.*: Mitochondrial
Dysfunction and Decrease in Body Weight
of Transgenic Knock-in Mouse Model for
TDP-43: the Question of Glucose?**

Thomas Floss

J. Biol. Chem. 2014, 289:18594.

doi: 10.1074/jbc.L114.579193

Access the most updated version of this article at <http://www.jbc.org/content/289/26/18594>

Find articles, minireviews, Reflections and Classics on similar topics on the [JBC Affinity Sites](#).

Alerts:

- [When this article is cited](#)
- [When a correction for this article is posted](#)

[Click here](#) to choose from all of JBC's e-mail alerts

This article cites 2 references, 2 of which can be accessed free at
<http://www.jbc.org/content/289/26/18594.full.html#ref-list-1>

LETTER

Reply to Jawaid *et al.*: Mitochondrial Dysfunction and Decrease in Body Weight of Transgenic Knock-in Mouse Model for TDP-43: the Question of Glucose?

This is a response to a letter by Jawaid et al. (1).

In their letter Jawaid *et al.* (1) refer to our recent paper on a TDP-43 transgenic knock-in (ki) mouse. The authors claim that Fig. 7 does not show differences in glucose levels of the animals and that an elevation of glucose levels would not be expected after mitochondrial dysfunction. In our measurements, glucose levels of TDP-43 ki animals were slightly elevated in *ad libitum* fed mice as compared with wild-type controls (ANOVA genotype-related *p* value was 0.04). In contrast, there was no significant difference in animals that were starved overnight. In these animals the tendency was to rather slightly lower levels. In my opinion, Fig. 7 clearly illustrates both these findings.

Mitochondrial abnormalities have been found solely in the brain to date (*e.g.* not in spinal cords), and in a follow-up study using compound mutants of TDP-43 ki and another FTLD risk gene, we have just confirmed these findings. However, the reported observations concerning glucose and mitochondria are unrelated, as we underline in our paper (“Whether mitochondrial abnormality could explain the observed metabolic changes and altered body weight composition of hTDP-43^{A315T} animals will be subject of future studies.”) (2).

Thomas Floss¹

Helmholtz Zentrum München, Institut für Entwicklungsgenetik,
Ingolstaedter Landstrasse 1, 85764 Neuherberg, Germany

1. Jawaid, A., Gapp, K., and Schulz, P. E. (2014) Mitochondrial dysfunction and decrease in body weight of transgenic knock-in mouse model for TDP-43: the question of glucose? *J. Biol. Chem.* **289**, 18593
2. Stribl, C., Samara, A., Trümbach, D., Peis, R., Neumann, M., Fuchs, H., Gailus-Durner, V., Hrabě de Angelis, M., Rathkolb, B., Wolf, E., Beckers, J., Horsch, M., Neff, F., Kremmer, E., Koob, S., Reichert, A. S., Hans, W., Rozman, J., Klingenspor, M., Aichler, M., Walch, A. K., Becker, L., Klopstock, T., Glasl, L., Höltz, S. M., Wurst, W., and Floss, T. (2014) Mitochondrial dysfunction and decrease in body weight of a transgenic knock-in mouse model for TDP-43. *J. Biol. Chem.* **289**, 10769–10784

DOI 10.1074/jbc.L114.579193

¹E-mail: tfloss@helmholtz-muenchen.de