Corrections

BIOCHEMISTRY. For the article "Crystal structure of a DEAD box protein from the hyperthermophile *Methanococcus jannaschii*" by Randall M. Story, Hong Li, and John N. Abelson, which appeared in number 4, February 13, 2001, of *Proc. Natl. Acad. Sci. USA* (98, 1465–1470), the authors note the following. The data deposition code for this article was incorrectly set as 1HVs instead of 1HV8. The online version of this article has been corrected.

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PHYSIOLOGY. For the article "Assessment of mitochondrial energy coupling in vivo by ¹³C/³¹P NMR" by Beat M. Jucker, Sylvie Dufour, Jianming Ren, Xueying Cao, Stephen F. Previs, Brian Underhill, Kevin S. Cadman, and Gerald I. Shulman, which appeared in number 12, June 6, 2000, of Proc. Natl. Acad. Sci. USA (97, 6880-6884; First Published May 23, 2000; 10.1073/ pnas.120131997), there was an error in the algorithm used to generate the 2-¹³C and 4-¹³C glutamate turnover curves. Consequently, we recalculated the tricarboxylic acid (TCA) cycle flux by using CWAVE software (Graeme F. Mason, Yale University, New Haven, CT). This mathematical modeling was based on nonlinear least squares fitting of the calculated parameters (4- and 2-¹³C citrate, α -ketoglutarate, glutamate) from the set of isotopic mass balance equations describing the label flow through the TCA cycle to the acquired NMR data using a Runge-Kutta algorithm with an adaptive step size. After recalculation, the absolute TCA cycle flux in all groups [control, triido-L-thyronine (T₃), and 2,4-dinitrophenol (DNP)] was found to be higher than originally reported. Consistent with our initial estimates, the revised calculations indicate that the TCA cycle flux (Fig. 3B) did significantly increase in the T₃ and DNP groups vs. the control group (P < 0.05 and P < 0.01, respectively). Additionally, the mitochondrial energy coupling (Fig. 3C) was reduced in the T₃ and DNP groups vs. the control group (P <0.01 and P < 0.001, respectively) with no significant difference between the T₃ and DNP groups. Therefore, although the absolute TCA cycle fluxes have increased as a result of the new calculations, our main conclusion regarding a reduction in mitochondrial energy coupling following T₃ and DNP treatments remains unchanged. Fig. 3 shows the results of our revised calculations.

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Fig. 3. Mitochondrial energy coupling measurements. (*A*–*C*) Data obtained from ¹³C and ³¹P NMR experiments in chronic T₃-treated and acute DNP-treated (known uncoupling agent) rats. (*A* and *B*) The calculated unidirectional ATP synthesis (*A*) and TCA cycle flux (*B*). There was a significant increase in TCA cycle flux in the T₃-treated vs. the control group and a much greater increase in the DNP-treated rats. These data suggest that, although tissue viability was not compromised by the T₃ and DNP treatment as reflected by similar ATP synthesis flux in all three groups (*A*), there was a significantly increased rate of substrate oxidation required to generate the ATP (*B*). Therefore, when comparing the degree of coupling (normalized ratio of ATP synthesis flux to TCA cycle flux) between these two measurements (*C*), it is evident that there was significantly greater mitochondrial uncoupling occurring in the T₃ and DNP vs. control group. Data are presented as means ± SEM. N.S., not significant.