ERRATUM

Erratum to: Brain-derived neurotrophic factor is produced by skeletal muscle cells in response to contraction and enhances fat oxidation via activation of AMP-activated protein kinase

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It has been brought to our attention following an investigation into the work of Bente Klarlund Pedersen by

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the Danish Committees for Scientific Dishonesty, that the erratum published in 2012 was insufficient to correct this article. Although the data published in the *Diabetologia* paper were previously unpublished, the data from the biological material collected from the additional eight healthy men presented in Fig. 1b and c originated from a previous study that was not referenced [1]. In addition, while these eight healthy subjects performed the same type of exercise at the same intensity, the duration was different. The following description of the methodology and Fig. 1 legend correct these oversights.

The authors would like to reiterate that these methodological oversights in no way affect either the data presented in the paper or the conclusions reached. The authors also apologise to both the journal and its readership for these oversights.

Methods

Human in vivo experiment

Twenty healthy, physically active but untrained men (age 25.6 ± 3.5 years, weight 78.9 ± 9 kg, height 185 ± 6.5 cm, BMI 21.3 ± 2.11 kg/m² [mean \pm SD]) were randomised to either an exercise (n=10) or control (n=10) group. There was no difference between the two groups with regard to age, weight, height or $\dot{V}O_{2\text{max}}$. Subjects either performed 120 min of bicycle exercise at 60% $\dot{V}O_{2\text{max}}$ followed by a 6 h recovery period (exercise) or rested in bed for 8 h (control). Subjects also reported to the laboratory after an overnight fast at 24, 48 and 72 h after the commencement of the experimental trial. Blood was obtained at time points 0, 2, 3, 5, 8, 24, 48 and 72 h. Muscle biopsy samples were obtained from vastus lateralis at

time points 0, 2, 3, 5, 8, 24, 48 and 72 h using a percutaneous needle biopsy technique with suction. Samples were snapfrozen before being analysed. Serum levels were measured by ELISA (R&D Systems, Wiesbaden-Nordenstadt, Germany). Platelet counts were determined by standard laboratory procedures. Data from this study are included in Figs 1a,d and 2a,b. Because of a lack of sufficient muscle biopsy material for BDNF protein analyses by western blot, we included another eight healthy men from a previous cohort [1] of 15 subjects: eight exercising and seven resting controls. We included muscle biopsies from the eight exercising men only (age 28.4±3 years, weight 82.3±8 kg, height 182.4 ± 5.9 cm, BMI 24.8 ± 2.1 kg/m² [mean \pm SD]). These subjects underwent the same exercise protocol as the first cohort (bicycle exercise at 60% $\dot{V}O_{2max}$) but for 180 min. A full set of muscle biopsies were available for western blot analysis pre- and post-exercise and 3 and 24 h post-exercise. The data from the latter study are used in Fig.1b,c.

Figure 1 Legend

Fig. 1 BDNF is increased in contracting skeletal muscle in vivo. *BDNF* mRNA levels (a) and protein expression in muscle tissue measured by western blot (b,c) and immunohistochemistry (d) at time points ranging from 0 to 72 h after 2 h (a,d) or 0 to 24 h after 3 h (b,c) of ergometer bicycle exercise at 60% of $\dot{V}O_{2\text{max}}$. *p<0.05, difference from preexercise (Pre-Ex); n=10 (a,d) and n=8 (b,c) per time-point. Material from two separate cohorts of subjects was used for (a,d) and (b,c). For *BDNF* mRNA studies (a), black columns indicate the exercised cohort and white columns indicate the resting cohort. Only exercise data are shown for protein expression studies (b,c). Values are means \pm SEM

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