

## Retraction

# Retracted: Treadmill Training Increases SIRT-1 and PGC-1 $\alpha$ Protein Levels and AMPK Phosphorylation in Quadriceps of Middle-Aged Rats in an Intensity-Dependent Manner

### Mediators of Inflammation

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Mediators of Inflammation has retracted the article titled “Treadmill Training Increases SIRT-1 and PGC-1 $\alpha$  Protein Levels and AMPK Phosphorylation in Quadriceps of Middle-Aged Rats in an Intensity-Dependent Manner” [1] due to unintentional reuse of a previously published image. After a concern was raised to them by a reader, the authors contacted the journal to replace the anti-ACC blot in Figure 2(b), providing the original figure. The journal found that the published blot in Figure 2(b) was similar in other articles [2–9]. Due to this, the journal reassessed the other figures and found that, for the  $\beta$ -actin bands in Figure 1(c), lanes 3 and 4 are similar to lanes 5 and 6, respectively. Although the authors provided the journal with the underlying blots for all the figures, the journal and authors decided to retract the manuscript. The replacement blot for Figure 2(b) and the underlying blots for all figures are available in the Supplementary Materials. The authors stated that the representative loading control bands in Figures 1(c) and 2(b) were improperly assembled, leading to repetitions of bands, and that these errors do not affect the results, once several papers have demonstrated that exercise does not change both, ACC [10, 11] and actin [12–14] protein content, in the skeletal muscle of rodents. In light of the figure preparation issues, the authors sincerely apologize to the scientific community for any misunderstanding that these errors may have caused.

### References

- [1] N. R. Oliveira, S. O. Marques, T. F. Luciano et al., “Treadmill training increases SIRT-1 and PGC-1 $\alpha$  protein levels and AMPK phosphorylation in quadriceps of middle-aged rats in an intensity-dependent manner,” *Mediators of Inflammation*, vol. 2014, Article ID 987017, 11 pages, 2014.
- [2] L. S. S. Pauli, E. C. C. Ropelle, C. T. de Souza et al., “Exercise training decreases mitogen-activated protein kinase phosphatase-3 expression and suppresses hepatic gluconeogenesis in obese mice,” *Journal of Physiology*, vol. 592, no. 6, pp. 1325–1340, 2014.
- [3] E. C. Chiarreotto-Ropelle, L. S. S. Pauli, C. K. Katashima et al., “Acute exercise suppresses hypothalamic PTP1B protein level and improves insulin and leptin signaling in obese rats,” *American Journal of Physiology - Endocrinology and Metabolism*, vol. 305, no. 5, pp. E649–E659, 2013.
- [4] E. R. Ropelle, M. B. Flores, D. E. Cintra, G. Z. Rocha, Pauli, J. r et al., “IL-6 and IL-10 Anti-Inflammatory Activity Links Exercise to Hypothalamic Insulin and Leptin Sensitivity through IKK $\beta$  and ER Stress Inhibition,” *PLOS Biology*, vol. 8, no. 8, Article ID e1000465, 2010.
- [5] Flores, B. S. Marcelo et al., “Obesity-Induced Increase in Tumor Necrosis Factor- $\alpha$  Leads to Development of Colon Cancer in Mice,” *Gastroenterology*, vol. 143, no. 3, pp. 741–753.e4, 2012, <http://dx.doi.org/10.1053/j.gastro.2012.05.045>.
- [6] P. O. Prada, P. G. F. Quresma, A. M. Caricilli et al., “Tub has a key role in insulin and leptin signaling and action in vivo in hypothalamic nuclei,” *Diabetes*, vol. 62, no. 1, pp. 137–148, 2013.
- [7] V. A. Barbosa, T. F. Luciano, S. O. Marques et al., “Acute exercise induce endothelial nitric oxide synthase phosphorylation via Akt and AMP-activated protein kinase in aorta of rats: Role of reactive oxygen species,” *International Journal of Cardiology*, vol. 167, no. 6, pp. 2983–2988, 2013.
- [8] Statement of Retraction, “Tub Has a Key Role in Insulin and Leptin Signaling and Action In Vivo in Hypothalamic Nuclei,” *Diabetes*, vol. 62, pp. 137–148, 2013.
- [9] J. Expression of concern. *Physiol*, “Expression of concern,” *The Journal of Physiology*, vol. 594, pp. 5027–5028, 2016.
- [10] H. M. O’Neill, J. S. Lally, S. Galic et al., “Skeletal muscle ACC2 S212 phosphorylation is not required for the control of fatty acid

- oxidation during exercise,” *Physiological Reports*, vol. 3, no. 7, Article ID e12444, 2015.
- [11] R. A. Gulli, J. M. Tishinsky, T. MacDonald, L. E. Robinson, D. C. Wright, and D. J. Dyck, “Exercise restores insulin, but not adiponectin, response in skeletal muscle of high-fat fed rodents,” *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, vol. 303, no. 10, pp. R1062–R1070, 2012.
- [12] C. He, M. C. Bassik, V. Moresi et al., “Exercise-induced BCL2-regulated autophagy is required for muscle glucose homeostasis,” *Nature*, vol. 481, no. 7382, pp. 511–515, 2012.
- [13] J. H. Woo, K. O. Shin, Y. H. Lee, K. S. Jang, J. Y. Bae, and H. T. Roh, “Effects of treadmill exercise on skeletal muscle mTOR signaling pathway in high-fat diet-induced obese mice,” *Journal of Physical Therapy Science*, vol. 28, no. 4, pp. 1260–1265, 2016.
- [14] G. Li, J. Wang, J. Ye, Y. Zhang, and Y. Zhang, “PPAR $\alpha$  protein expression was increased by four weeks of intermittent hypoxic training via AMPK $\alpha$ 2-dependent manner in mouse skeletal muscle,” *PLoS ONE*, vol. 10, no. 4, Article ID e0122593, 2015.