Retraction and Correction

RETRACTION

MEDICAL SCIENCES

Retraction for "Dominant suppression of inflammation by glycan-hydrolyzed IgG," by Kutty Selva Nandakumar, Mattias Collin, Kaisa E. Happonen, Allyson M. Croxford, Susanna L. Lundström, Roman A. Zubarev, Merrill J. Rowley, Anna M. Blom, and Rikard Holmdahl, which appeared in issue 25, June 18, 2013, of *Proc Natl Acad Sci USA* (110:10252–10257; first published May 13, 2013; 10.1073/pnas.1301480110).

The authors wish to note the following: "Using studies of IgG hydrolyzed by the streptococcal glycan hydrolyzing enzyme EndoS, we found that treatment of mice with hydrolyzed IgG blocked antibody mediated arthritis. As an explanation for this observation, we suggested that EndoS-hydrolyzed IgG per se dominantly blocks local immune complex formation.

"With new data from our own follow up experiments, we have now found that this conclusion was incorrect.

"Our new data shows that injection of EndoS is much more potent in vivo than we could logically anticipate, as i.v. injection of doses containing less than 0.1 μg EndoS mixed with IgG suppressed arthritis using the same model as the one reported in the initial paper (collagen antibody-induced arthritis). We previously excluded the possibility that contaminating EndoS could play a role, as this contaminating amount was not detected using standard methods in the hydrolyzed IgG fraction we used in the experiments. Furthermore, much higher doses of EndoS injected in the same mouse strain as a control experiment did not affect collagen induced arthritis in earlier experiments. The correct interpretation of our collective data is that EndoS operates very potently in vivo on an immune complex-mediated disease, possibly by accumulating within immune complexes. Because this interpretation is different from our major conclusion of the published paper, the authors have unanimously decided to retract this paper to be able to publish the data connected with a correct interpretation. We sincerely apologize to readers of this paper, who might have been misled by our earlier interpretation."

Kutty Selva Nandakumar
Mattias Collin
Kaisa E. Happonen
Allyson M. Croxford
Susanna L. Lundström
Roman A. Zubarev
Merrill J. Rowley
Anna M. Blom
Rikard Holmdahl

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CORRECTION

MEDICAL SCIENCES

Correction for "Increased muscle PGC-1α expression protects from sarcopenia and metabolic disease during aging," by Tina Wenz, Susana G. Rossi, Richard L. Rotundo, Bruce M. Spiegelman, and Carlos T. Moraes, which appeared in issue 48, December 1, 2009, of *Proc Natl Acad Sci USA* (106:20405–20410; first published November 16, 2009; 10.1073/pnas.0911570106).

The authors note that the α-tubulin loading control blot in Fig. 4*D* appeared incorrectly. The corrected figure and its legend appear below.

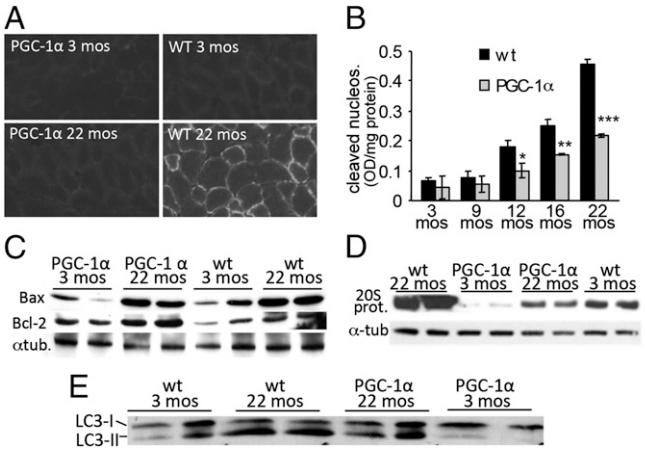

Fig. 4. Increased PGC-1α levels in aging muscle prevent degradative processes. (*A*) Immunohistochemistry of biceps femoris using anti-active caspase 3 antibody to detect apoptosis. (*B*) Apoptotic index in skeletal muscle homogenates of wild-type and MCK-PGC-1α of different age-groups based on nucleosome fragmentation ($n = 6$ for each group). *, $P < 0.05$, **, $P < 0.01$, ***, $P < 0.001$. (*C*) Western blot of Bax and Bcl-2 in skeletal muscle homogenates. (*D*) Western blot of the 20S subunit of the proteasome and tubulin in skeletal muscle homogenates. (*E*) Western blot of LC3-I and LC3-II in skeletal muscle homogenates.

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