Corrigendum

Corrigendum to “Pioglitazone Attenuates Drug-Eluting Stent-Induced Proinflammatory State in Patients by Blocking Ubiquitination of PPAR”

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In the article titled “Pioglitazone Attenuates Drug-Eluting Stent-Induced Proinflammatory State in Patients by Blocking Ubiquitination of PPAR” [1], there are additional methods that should be added to the article which are mainly about the methods for PPAR gamma binding and ubiquitination. The additional methods are shown below:

2.8. Immunoblotting. Protein levels of p65, p50, PPAR-γ, and Gapdh of MNC were detected by western blotting with Santa Cruz antibodies against the p65 subunit (sc-372), p50 subunit (sc-114), PPAR-γ (sc-7273), and Gapdh (SC-48166) as described previously [24, 25]. Mononuclear cells were lysed in RIPA buffer (Cybrdi, Gaithersburg, MD, USA) that contained protease inhibitor (Roche Diagnostics, Indianapolis, IN, USA). Equal amounts of protein were loaded onto 4–12% Bis-Tris precast gels for electrophoresis and were electrotransferred onto a PVDF membrane (Roche Diagnostics). After blocking for 1h at room temperature, membranes were sequentially incubated with primary Abs at 4°C overnight and secondary Ab at room temperature for 1h. The protein signal was detected by chemiluminescence and all values were corrected by loading with Gapdh.

2.9. Immunoprecipitation. Immunoprecipitation assays were performed as described previously [25, 26]. Cell extracts were prepared by using modified RIPA buffer (Cell Signaling Technology). Cell lysates (300 μg) were incubated with 1 μg of PPAR Ab (sc-7273) at 4°C overnight and precipitated with protein G agarose beads (Santa Cruz) at 4°C for 2h. Immunoprecipitated proteins were separated by SDS-PAGE and transferred onto a PVDF membrane. Membranes were then sequentially incubated with primary ubiquitination Ab (SC-8017) and secondary Abs. Bands were visualized by using an ECL system.

In addition, the below part should be added to the Discussion section after the third paragraph:

“The peroxisome proliferator-activated receptor-γ (PPAR-γ) is a member of the nuclear receptor superfamily of ligand-dependent transcription factors which regulate adipocyte differentiation, glucose homeostasis, and macrophage activation [27]. PPAR-γ ubiquitination and degradation have been found in adipocyte, and proteasome inhibitors inhibited the downregulation of PPAR-γ [28]. Proinflammation cytokine was also found to induce PPAR-γ degradation [29]. Herein, we found a novel mechanism such that PIO enhances PPAR-γ binding activity though inhibiting its ubiquitination and degradation, which may be important for TZDs clinical use.”

Moreover, the references below should be included in the References list:


**References**
