Correction to: Cadmium exposure alters steroid receptors and proinflammatory cytokine levels in endothelial cells in vitro: a potential mechanism of endocrine disruptor atherogenic effect

S. Fittipaldi¹ · V. M. Bimonte^{2,3} · A. Soricelli^{1,4} · A. Aversa³ · A. Lenzi⁵ · E. A. Greco⁵ · S. Migliaccio²

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Unfortunately, the figure captions 4 and 6 were incorrectly published in the original publication. The complete correct captions are given below.

Figure 4 Cadmium modulation of AR-mediated signaling pathway. Representative blot which depicts AR and its phosphorylation at serine 308 (PhAR^{ser308}), GSK-3 β and its phosphorylation at serine 9 (PhGSK-3 β ^{ser9}) after 1 h of Cd exposure (a). Histograms represent the mean values of ratio of PhAR^{ser308} normalized over AR total protein level and the PhGSK-3 β ^{ser9} level normalized to GSK-3 β total protein content (b). Representative blot which depicts AR phosphorylation at serine 308 and AKT phosphorylation at serine 473 (PhAKT^{ser473}) in HUVECs after 1 h CdCl₂ (10 μ M) treatment, in the presence of GSK-3 β inhibitor, AR-A014418 (10 μ M) (c). Histograms represent the mean values of

ratio of PhAR^{ser308} normalized over AR total protein and PhAKT^{ser473} over total AKT (d). Effect of CdCl₂ with or without DHT on HUVECs proliferation (e). Cells were incubated with or without CdCl₂ (10 μ M), DHT (0.01 μ M) or CdCl₂ in the presence of DHT for 24 and 48 h. Number of viable cells was quantified using the MTS assay. Results are presented as mean $\pm$ SD of three separate experiments. ** $p \leq 0.01$ and * $p \leq 0.05$ vs vehicle-treated cells; § $p \leq 0.05$ vs 10 μ M CdCl₂-treated cells.

Figure 6 Effect of ER β blockage on proinflammatory cytokines expression. RT-qPCR showing TNF α (a), IL-6 (b), and IL-8 (c) mRNA levels of HUVECs cells exposed to CdCl₂ (10 μ M) or E2 (0.01 μ M) in the presence or absence of PHTPP (5 μ M), a selective inhibitor of ER β . * $p \leq 0.05$ and ** $p \leq 0.01$ vs vehicle treated cells; §§ $p \leq 0.01$ and § $p \leq 0.05$ vs 10 μ M CdCl₂-treated cells.

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✉ S. Migliaccio
silvia.migliaccio@uniroma4.it

¹ IRCCS SDN, Naples, Italy

² Department of Movement, Human and Health Sciences, Section of Health Sciences, “Foro Italico” University of Rome, Largo Lauro De Bosis 6, 00195 Rome, Italy

³ Department of Experimental and Clinical Medicine, Magna Graecia University, Catanzaro, Italy

⁴ Department of Motor Sciences and Healthiness, University of Naples Parthenope, Naples, Italy

⁵ Department of Experimental Medicine, Section of Medical Pathophysiology, Endocrinology and Nutrition, “Sapienza” University of Rome, Rome, Italy