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CORRECTION | NOVEMBER 15 2008

Expression of a soluble TGF- β receptor by tumor cells enhances dendritic cell/tumor fusion vaccine efficacy. **FREE**

M. Zhang; ... et. al

J Immunol (2008) 181 (10): 7428.

<https://doi.org/10.4049/jimmunol.181.10.7428-b>

Related Content

Expression of a Soluble TGF- β Receptor by Tumor Cells Enhances Dendritic Cell/Tumor Fusion Vaccine Efficacy

J Immunol (September,2008)

Tumor-Derived TGF- β Reduces the Efficacy of Dendritic Cell/Tumor Fusion Vaccine

J Immunol (April,2003)

A Novel Bifunctional Fusion Protein Comprising of TGF- β RII trap and IL15/IL-15R α as an Immunotherapeutic against Cancer

J Immunol (May,2020)

Corrections

Palazzo, M., S. Gariboldi, L. Zanobbio, S. Selleri, G. F. Dusio, V. Mauro, A. Rossini, A. Balsari, and Cr. Rumio. 2008. Sodium-dependent glucose transporter-1 as a novel immunological player in the intestinal mucosa. *J. Immunol.* 181: 3126–3137.

In **Results**, under the subhead titled *SGLT-1 activation blocks NF- κ B nuclear translocation induced by LPS and CpG-ODN*, the corrected text in the fourth, fifth, and six sentences of the paragraph should read as follows: “In HT-29, LCC-18, and STC-1 cells stimulated with LPS or CpG-ODN, we observed activation of NF- κ B, i.e., translocation to the nucleus, whereas this activation was not detected in cells pretreated with D-glucose (Fig. 6A) or 3-OMG (data not shown). NF- κ B in the cytoplasm is complexed to members of the I κ B family of inhibitory proteins. Western blot analysis revealed degradation of I κ B when NF- κ B translocates to the nucleus upon LPS or CpG-ODN stimulation; degradation of I κ B in stimulated cells was inhibited by D-glucose (Fig. 6B) or 3-OMG (data not shown) pretreatment.”

In addition, changes have been made in Fig. 6 concerning the Western blots in B for LCC-18 and STC-1 and minor changes have also been made to the figure legend. Both the revised Fig. 6 and the revised legend are shown below.

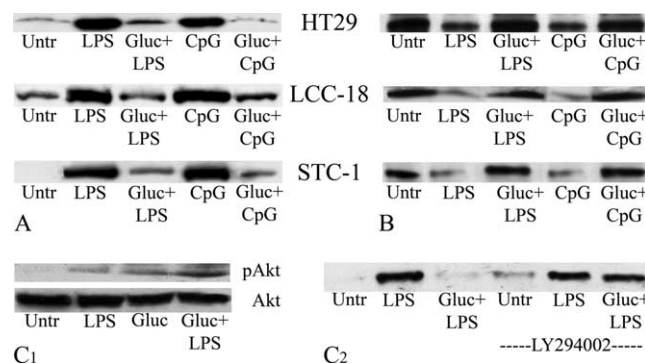


FIGURE 6. A, Western blot analysis for NF- κ B in IEC nuclear extracts. HT29, LCC-18, and STC-1 cells were stimulated with LPS or CpG-ODN with or without D-glucose pretreatment. Nuclear extracts were analyzed by Western blotting with anti-NF- κ B p65 Ab. B, Western blot analysis for I κ B in IEC extracts. HT29, LCC-18, and STC-1 cells were stimulated with LPS or CpG-ODN with or without D-glucose pretreatment. Total protein extracts were analyzed by Western blotting with anti-I κ B Ab. C, Involvement of Akt signaling pathway. HT29 protein extracts, following LPS and/or glucose treatment, were analyzed by Western blotting with anti-phospho-Akt and anti-Akt Abs (C₁). In addition, cells were stimulated with LPS, with or without D-glucose pretreatment, in the presence or absence of Akt inhibitor LY294002. Nuclear extracts were analyzed by Western blotting with anti-NF- κ B p65 Ab (C₂). Untr, Untreated.

Brincks, E. L., A. Katewa, T. A. Kucaba, T. S. Griffith, and K. L. Legge. CD8 T cells utilize TRAIL to control influenza virus infection. 2008. *J. Immunol.* 181: 4918–4925.

Reference 14 should read as follows: “Ishikawa, E., M. Nakazawa, M. Yoshinari, and M. Minami. 2005. Role of TNF-related apoptosis-inducing ligand in immune response to influenza virus infection in mice. *J. Virol.* 79: 7658–7663.”

Zhang, M., B. E. Berndt, J.-J. Chen, and J. Y. Kao. 2008. Expression of a soluble TGF- β receptor by tumor cells enhances dendritic cell/tumor fusion vaccine efficacy. 2008. *J. Immunol.* 181: 3690–3697.

In **References**, Ref. 33 should be added as follows: “Wang, F. L., W. J. Qin, W. H. Wen, F. Tian, B. Song, Q. Zhang, C. Lee, W. D. Zhong, Y. L. Guo, and H. Wang. 2007. TGF- β insensitive dendritic cells: an efficient vaccine for murine prostate cancer. *Cancer Immunol. Immunother.* 56: 1785–1793.”

Ref. 33 was not cited in **Discussion**. The text should read as follows: “In a study similar to the present study, Wang and colleagues showed that DCs genetically engineered to secrete dominant-negative TGF- β -R when pulsed with tumor lysate were more efficacious in generating an antitumor response compared with nonsecreting tumors in a mouse prostate adenocarcinoma model (33).”