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Corrigendum: Fc engineered ACE2-Fc is a potent multifunctional agent targeting SARS-CoV2

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A corrigendum on

Fc engineered ACE2-Fc is a potent multifunctional agent targeting SARS-CoV2.

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In the published article, there was an error in the legend for Figure 3 as published. The statistics style output rather than the significance levels was reported. The corrected legend appears below.

“Characterization of engineered human fACE2-Fc and trACE2-Fc fusion proteins. (A) Size-exclusion chromatography (SEC) of IEX fractions containing fACE2-Fc-WT using a Superose 6 column, with oligomeric, monomeric forms and low mw impurities (†) indicated; and (B) SEC of IEX fractions containing fACE2-Fc H429Y, showing the high proportion of oligomeric species. (C–F) Biolayer interferometry (BLI) analysis of ACE2-Fc proteins which were immobilized on anti-human Fc (BLI) sensors and reacted with the indicated concentrations of RBD. The dissociation constants, K_D (nM), are derived from global fitting of the association and dissociation curves to a Langmuir binding model. The ACE2-Fc proteins were, (C) trACE2-Fc WT (D) fACE2-Fc WT, (E) fACE2-Fc H429F and (F) the RBD binding-enhanced triple mutant of ACE2 fused to Fc; EflACE2-Fc WT (representative of $n = 2$ independent experiments). (G) Native Gel-shift analysis of ACE2-Fc proteins (1 μ g, ~ 5 pmol) alone or combined with SARS-CoV-2 spike RBD-Ig (0.5 μ g, ~ 5 pmol) and analyzed by native PAGE. The resulting shift in size of the proteins in the mixtures demonstrated the formation of ACE2-Fc: Cov2-RBD complexes. (H) Binding of different formats of ACE2-Fc-WT, and their Fc variants to immobilized RBD-Ig was determined by ELISA. EC_{50} (nM) values are from agonist versus response curve fits, mean $\pm$ SD, n is indicated by individual symbols for each independent experiment. One-way ANOVA with Dunnett’s multiple comparisons test, $p > 0.05$ (ns), ≤ 0.05 (*), ≤ 0.01 (**), ≤ 0.0001 (****).”

In the published article, there was an error in the legend for Figure 4 as published. The statistics style output rather than the significance levels was reported. The corrected legend appears below.

“(A–C) SARS-CoV-2 neutralization potency of ACE2-Fc fusion proteins is increased by both the ACE2 scaffold and the H429Y Fc mutation. Neutralization potencies of the ACE2 enzymatic ectodomain polypeptide (trACE2) and the three formats of ACE2-Fc-WT fusion and variant proteins were determined by titration of the cytopathic effect to endpoint in a micro-neutralization assay. The fusion proteins were (A) trACE2-Fc-WT, (B) fACE2-Fc-WT and (C) EflACE2-Fc-WT, incorporating triple mutation of ACE2 engineered (43) for enhanced affinity to RBD and their Fc variants (Eu numbering), E430G, G; H429F, F; H429Y oligomers on SEC, Yog; and H429Y monomers on SEC, Ymn. A further variant trACE2-Fc fusion protein is the glycan-modified trACE2-Fc-kif produced in the presence of kifunensine. Mean $\pm$ SEM, one-way ANOVA with Dunnett’s multiple comparisons test, $p > 0.05$ (ns), ≤ 0.05 (*), ≤ 0.01 (**), independent experiments (n) are indicated as individual symbols.”

In the published article, there was an error in the legend for Figure 5 as published. The statistics style output rather than the significance levels was reported. The corrected legend appears below.

“Fc γ R and complement dependent effector functions of the ACE2-Fc decoy proteins. (A) Activation of Fc γ RIIIa by ACE2-Fc proteins. ACE2-Fc proteins activated Fc γ RIIIa, except for the Fc H429Y mutants which failed to stimulate in any ACE2 format either as oligomeric or monomeric forms. Ramos-S target cells were opsonized with trACE2-Fc, fACE2-Fc and EflACE2-Fc, WT and Fc variants, including H429F, F; H429Y, Y; E430G, G or trACE2-Fc-kif, produced from trACE2-Fc WT in 293Expi cells in the presence of the mannosidase inhibitor kifunensine. In some experiments Ramos-S target cells were separately opsonized with Rituximab, RIT. These opsonized targets were incubated with Fc γ RIIIa/NF- κ B-RE nanoluciferase reporter cells and Fc γ RIIIa activation measured by the induction of nanoluciferase (RLU). Activation data (Supplementary Figures S1B, C) were fitted to agonist response curves to estimate EC_{50} (nM); nd, not determined as there was insufficient activity for the data to be fitted. EC_{50} values from the curve fits are shown. Mean $\pm$ SEM, n is indicated by individual symbols for each independent experiment, one-way ANOVA with Dunnett’s multiple comparisons test, comparing to trACE2-Fc WT. $p > 0.05$ (ns), ≤ 0.05 (*), ≤ 0.01 (**), ≤ 0.001 (***), ≤ 0.0001 (****). (B) H429F, and E430G Fc mutant ACE2-Fc proteins are potent mediators of complement lysis of SARS-CoV-2 S expressing cells. Flow cytometric analysis of complement-dependent cytotoxicity (CDC) of opsonized Ramos-S cells was determined in the presence of a 1/3 dilution of a pool of normal human serum (from >5 individuals) as a source of complement. Plots are mean $\pm$ SEM, $n = 3$ independent experiments. Two-way ANOVA with Dunnett’s multiple comparisons test comparing to trACE2-Fc-WT for main column effect, $p > 0.05$ (ns), ≤ 0.01 (**), ≤ 0.0001 (****). EC_{50} (nM) values are mean $\pm$ SEM each from 3 curve fits.”

The authors apologize for these errors and state that they do not change the scientific conclusions of the article in any way. The original article has been updated.

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