

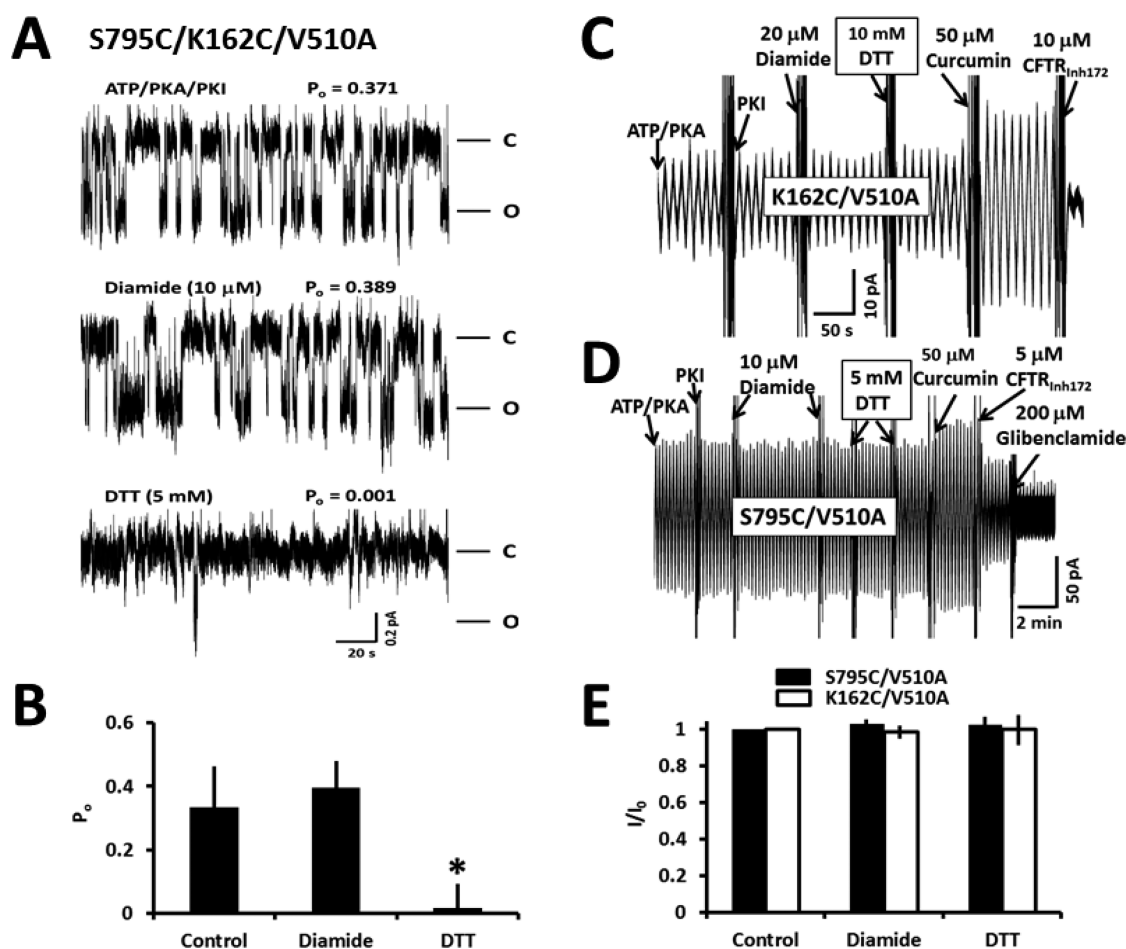
# Correction to Molecular Basis for Fe(III)-Independent Curcumin Potentiation of Cystic Fibrosis Transmembrane Conductance Regulator Activity

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The correct version of Figure 8 appears here.

Line 6 of page 2835, “from 0.164 to 0.180” is corrected as “from 0.333 to 0.396”. Line 9 of page 2835, “from 0.180 to 0.001” is corrected as “from 0.396 to 0.016”.



**Figure 8.** Effects of spontaneous disulfide cross-linking of K162C to S795C on single-channel opening. (A) Representative inside-out single-channel currents of S795C/K162C/V510A across an excised HEK-293T micropatch. The holding potential was  $-60$  mV. The channel was activated with 1.5 mM MgATP and 24 units/mL PKA, followed by PKI. Diamide was used to induce formation of a disulfide bond, while DTT was employed to disrupt the disulfide bond. (B) Changes in the open probability of S795C/K162C/V510A ( $n = 3$ ;  $*P < 0.001$ , from an unpaired Student's  $t$  test). Inside-out macroscopic currents of (C) K162C/V510A and (D) S795C/V510A across an excised HEK-293T patch. The channel was activated with 1.5 mM MgATP and 24 units/mL PKA, followed by PKI (1.4  $\mu$ g/mL). CFTR<sub>inh172</sub> (5–10  $\mu$ M) or glibenclamide (200  $\mu$ M) was used to block the total CFTR-mediated current. (E) Relative changes in currents in response to diamide (10–20  $\mu$ M) and DTT (5–10 mM) ( $n = 3$ ;  $*P < 0.001$ , from an unpaired Student's  $t$  test).

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