SUPPLEMENTARY ONLINE DATA

S-palmitoylation regulates biogenesis of core glycosylated wild-type and F508del CFTR in a post-ER compartment

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FIGURE LEGENDS

Figure S1 2-BP blunts vectoral chloride (Cl^-) transport

Transepithelial Cl^- transport was measured by Ussing chamber analysis for CFBE (**A**,**B**) and Calu-3 (**C**,**D**) cells grown on permeable supports following treatment with 2-BP (4 h prior to study). Summary bar graphs (B,D) depict the overall effect of forskolin (20 μM) and genistein (50 μM) to activate CFTR. Inh172 (10 μM) is a blocker of CFTR function. Results are shown as mean ± SEM and normalized to control cells. *$P < 0.05$; $n = 4$.

Figure S2 Immunofluorescence of HeLa wild-type cells in the presence or absence of DHHC-7

Wild-type CFTR was labeled in the absence (column I) and presence (columns II-IV) of overexpressed DHHC-7. CFTR and HA-tagged DHHC-7 were labeled with anti-NBD1 and anti-HA antibodies, respectively. Golgi (top panel IV) or ER (lower panel IV) compartments were detected with CellLight GFP (Molecular Probes). Immunofluorescence identified strong co-localization of wild-type CFTR with DHHC-7 and Golgi markers.

Figure S1

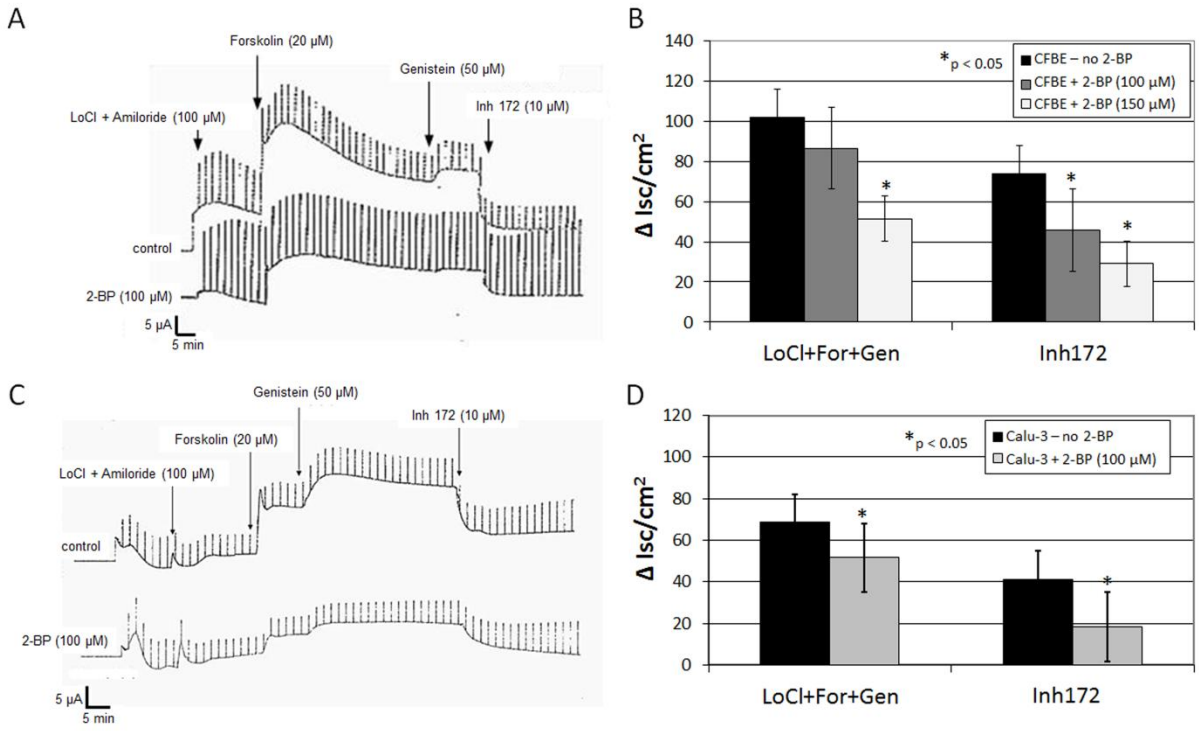
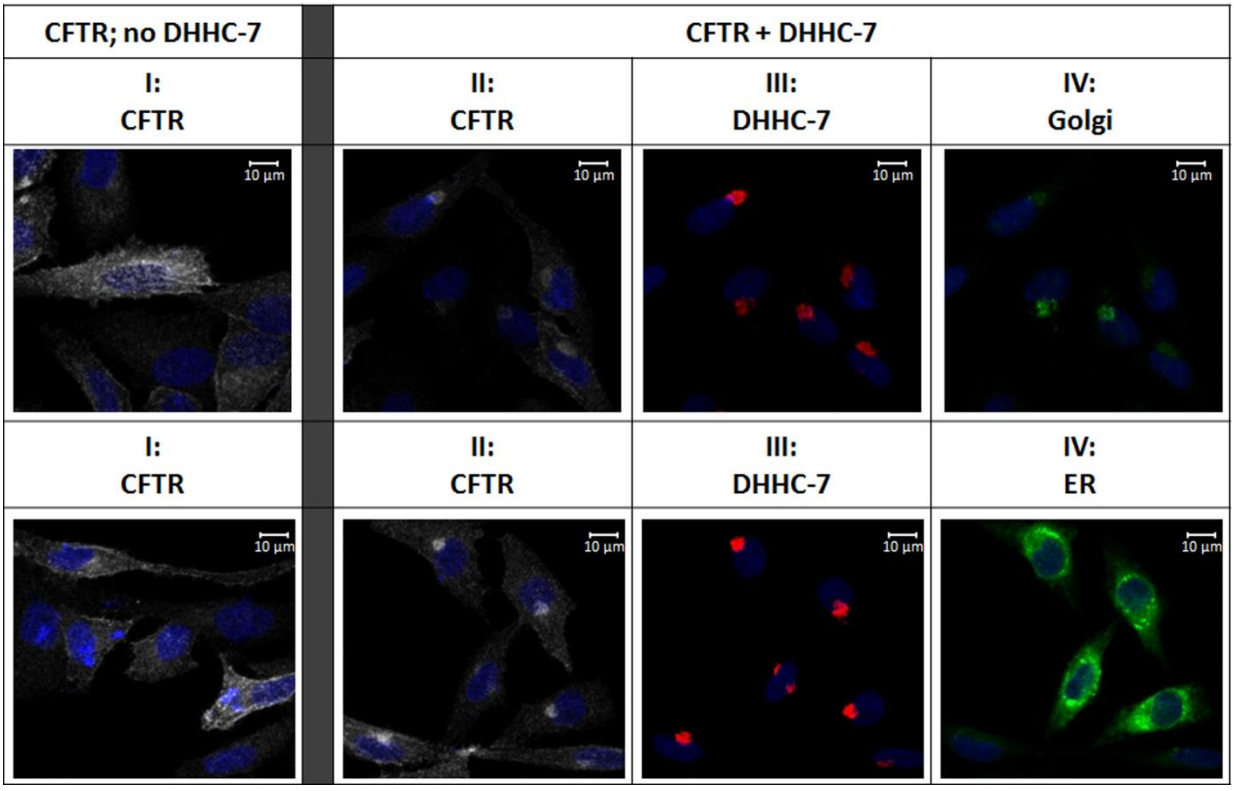

A
Forskolin (20 µM)
Genistein (50 µM)
Inh 172 (10 µM)
LoCl + Amiloride (100 µM)
control
2-BP (100 µM)
5 µA
5 min
B
*p < 0.05
CFBE – no 2-BP
CFBE + 2-BP (100 µM)
CFBE + 2-BP (150 µM)
Δ Isc/cm²
LoCl+For+Gen
Inh172
C
Genistein (50 µM)
Inh 172 (10 µM)
Forskolin (20 µM)
LoCl + Amiloride (100 µM)
control
2-BP (100 µM)
5 µA
5 min
D
*p < 0.05
Calu-3 – no 2-BP
Calu-3 + 2-BP (100 µM)
Δ Isc/cm²
LoCl+For+Gen
Inh172

Figure S2


CFTR; no DHHC-7
CFTR + DHHC-7
I:
CFTR
II:
CFTR
III:
DHHC-7
IV:
Golgi
I:
CFTR
II:
CFTR
III:
DHHC-7
IV:
ER
10 µm