Host cell kinases, $\alpha_5$ and $\beta_1$ integrins, and Rac1 signalling on the microtubule cytoskeleton are important for non-typable Haemophilus influenzae invasion of respiratory epithelial cells

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In Figs 1–5 and 7 of the published manuscript, the multiplier was given incorrectly on the $y$-axis of each graph. In each case, the number had a negative exponent instead of a positive exponent. The correct figures, with their legends, are shown below.

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**Fig. 1.** Involvement of PI3K downstream molecules in NTHi invasion of A549 cells. (a, b) NTHi invasion (a) and adhesion (b) were monitored in the absence (CON) or presence of AktVIII. (b) NTHi375 adhesion was unaltered by AktVIII. (c, d) Invasion (c) and adhesion (d) were monitored in the absence (CON) or presence of U-73122, Gö6983, calphostin C and GF109203X. (c) PLC, but not PKC, prevents NTHi A549 cell invasion. *P<0.05 compared with cells infected in the absence of treatment. Error bars, SEM.
Fig. 2. Involvement of integrins in NTHi invasion. (a) A549 cells were treated with Mn²⁺ and NTHi375 invasion was determined. (b) Cells were left untreated or were preincubated with control anti-β1 integrin Alex1/4, activating anti-β1 integrin TS2/16 or blocking anti-β1 integrin Lia1/2 antibodies. NTHi375 invasion was determined in each case. (c) Cells were treated with a pool of three siRNAs to β1 integrin, and infected with NTHi375 to determine invasion. Inset, immunoblot analysis of integrin β1, shown in control (AS-CON) and siRNA-treated cells; tubulin was a loading control. (d) Integrin β1 is recruited to the site of bacterial adhesion. A549 cells were infected with NTHi375. NTHi (DNA) was stained with Hoescht 33342 (blue). Integrin β1 was stained with goat anti-β1 integrin and Cy2-conjugated donkey anti-goat antibodies (green). Image was taken at 120 min post-infection. α5 Integrin is involved in NTHi375 epithelial infection. (e, f) Cells were preincubated with increasing concentrations of blocking anti-α5 P1D6 antibody and bacterial invasion (e) and adhesion (f) were determined. (g, h) Cells were preincubated with increasing concentrations of RGD peptide. NTHi invasion (g) and adhesion (h) were determined. (i) α5 Integrin is involved in epithelial infection by a set of NTHi clinical isolates. Cells were left untreated (U) or were preincubated with 20 μg anti-α5 P1D6 antibody ml⁻¹ and 5 μM RGD peptide. Bacterial adhesion was determined for NTHi398, 1566, 1606 and 1619. *P<0.05 compared with cells infected in the absence of treatment. Error bars, SEM. CON, Untreated cells.
PTKs are required for NTHi invasion. (a) NTHi375 invasion was monitored in the absence or presence of genistein, PF-573228, PP2 and PP3. FAK and Src are involved in NTHi invasion. (b) Activation of Src PTK upon infection was detected using anti-Src pY418 antibody. Extracts were prepared from cells not infected (−) or infected for 20–120 min with NTHi375. Tubulin was a loading control. FAK is required for NTHi invasion, but not for NTHi adhesion. (c, d) A549 cells were treated with two siRNAs to FAK, and infected with NTHi375 to determine invasion (c) and adhesion (d). (e) Upper: immunoblot analysis of FAK in control (AS-CON) and siRNA–treated cells. Lower: Src immunodetection in control cells and cells transfected with a construct encoding the kinase-inactive form SrcK297M. In both cases, tubulin was a loading control. (f, g) Src is required for NTHi invasion, but not for NTHi adhesion. A549 cells were transiently transfected with pSrcK297M and infected with NTHi375; invasion (f) and adhesion (g) were determined. (h) Src is recruited to the site of bacterial adhesion. A549 cells were transiently transfected with a construct encoding Src–GFP and infected with NTHi375. NTHi was stained with rabbit anti-NTHi and donkey anti-rabbit-Rhodamine antibodies (red, upper) and with Hoescht 33342 (blue, lower). Src–GFP is shown in green. The image was taken at 90 min post-infection. *P<0.05 compared with cells infected without treatment. Error bars, SEM. CON, Untreated cells.
Fig. 4. Rho GTPases are coordinately required for NTHi invasion. (a) Invasion was monitored in the absence or presence of the Rho GTPase inhibitors *C. botulinum* toxin B, NSC23766 and CT04. (b) Rac1 and Pak1 are implicated in NTHi invasion. A549 cells were treated with siRNAs to Rac1 and Pak1, and infected with NTHi375 to determine invasion. (c) Interference of Rac1 (upper) and Pak1 (lower) was evaluated by Western blotting of cell lysates from control (AS-CON) and siRNA-treated cells. Tubulin was a loading control. (d) Rac1 activation, detected by the amount bound to the GST-Pak Rac1 interaction binding site, was determined in A549 cells uninfected (–) or infected for 60 or 120 min with NTHi375. Tubulin was a loading control before the pull-down. (e) Vav2 GEF interference reduces NTHi invasion. A549 cells were knocked down with siRNA to Vav2 and infected with NTHi375 to determine invasion. (f) Vav2 interference was evaluated in cell lysates from control (AS-CON) and siRNA-treated cells. Tubulin was a loading control. (g) Op18/stathmin interference increases NTHi invasion. A549 cells were treated with two siRNAs to Op18/stathmin and infected with NTHi375 to determine invasion. (h) siRNA-interference of Op18/stathmin was evaluated in cell lysates from control (AS-CON) and siRNA-treated cells; tubulin was a loading control. *P<0.05 compared with cells infected in the absence of treatment. Error bars, SEM. CON, Untreated cells.
Fig. 5. The increase of cytosolic cAMP reduces NTHi invasion. (a) Invasion was monitored in the absence or presence of H-89, exogenous PGE₂ or forskolin. (b) A549 cell pretreatment with H-89, PGE₂ or forskolin did not alter NTHi adhesion. *P<0.05 compared with cells infected without treatment. Error bars, SEM. CON, Untreated cells.

Fig. 7. RhoA and ROCK diminish the recovery of intracellular NTHi. (a) A549 cells were treated with siRNA to RhoA and infected to determine invasion. (b) Cells were treated with siRNA to RhoA and infected to determine adhesion. (c) RhoA interference was evaluated by Western blotting of cell lysates from control and siRNA-treated cells; tubulin was a loading control. (d) ROCK inhibition by cell treatment with Y-27632 increases bacterial invasion; invasion was restored to control levels in cells pretreated with Y-27632 before colchicine addition. (e) NTHi adhesion was not altered, independently of the absence or presence of colchicine and Y-27632. *P<0.05 compared with cells infected without treatment. Error bars, SEM. AS-CON or CON, Untreated cells.