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Author Correction: Arylsulfatases and neuraminidases modulate engagement of CCR5 by chemokines by removing key electrostatic interactions

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The original version of this Article contained an error in Figure 1, panel B, where the label “Hek/1/85a (sulfation-insensitive)” was incorrectly given as “Hek/1/85a (sulfation-sensitive)”. The original Figure 1 and accompanying legend appear below.

The original Article has been corrected.

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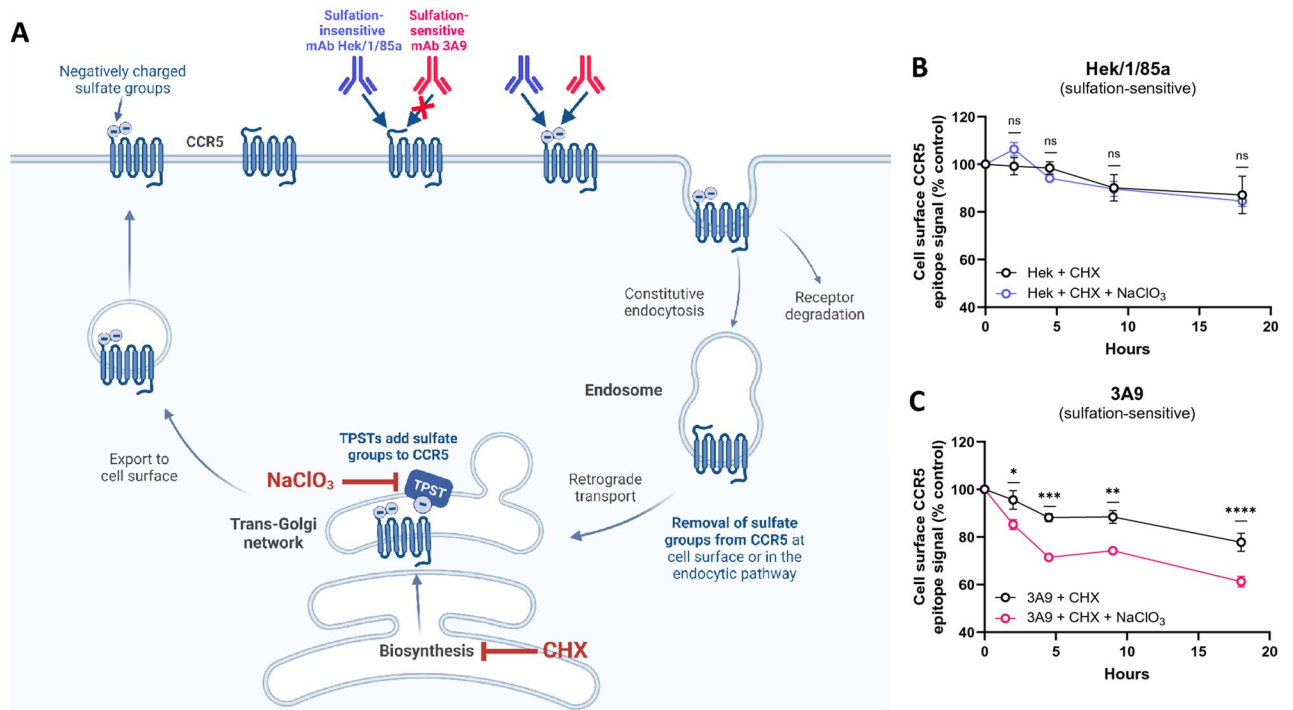


Figure 1. Rapid turnover of tyrosine degradation as CCR5 cycles through the cell. **(A)** CCR5 cycles spontaneously through the cell in a process of endocytosis followed by recycling to the cell surface via the TGN. HEK-CCR5 cells were treated with cycloheximide (CHX) to prevent newly biosynthesized CCR5 from entering the cellular CCR5 pool, and receptor turnover was measured using sulfation-sensitive (3A9) and sulfation-insensitive (Hek/1/85a) anti-CCR5 mAbs in the presence or absence of TPST blockade using sodium chlorate. Created with Biorender. **(B)** Time-course determination of cell surface CCR5 on HEK-CCR5 cells using flow cytometry with anti-CCR5 sulfation-insensitive mAb Hek/1/85a and **(C)** sulfation-sensitive anti-CCR5 mAb 3A9. Binding signals are expressed as % control (CTRL) $[(\text{median fluorescence intensity (MFI)}_{\text{CHX or CHX+NaClO}_3}) / \text{MFI}_{\text{CTRL}} \times 100]$, where CTRL corresponds to sulfate-free medium without inhibitors. Data show mean binding signals \pm SEM from 3 independent experiments. 2-way ANOVA analysis was performed on measurements made at each timepoint using log-transformed values.



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