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3 Figure S1: CHO-K1 cells presented functionally active ErbB receptors at their cell

4 surface after transfection.

(A) Cells were transfected with the empty control vector (pcDNA) or plasmids, that either
encoded for EGFR, ErbB2 or ErbB4. WCL extracts were analyzed by Western blotting
using appropriate ErbB antibodies and demonstrated heterologous expression of EGFR,
ErbB2 and ErbB4 in CHO-K1. In parallel, ErbB receptor expression on the cell surface
was estimated by FACS analysis. The data show mean values of the geometric means
of 3 independent experiments.

(B) pcDNA or EGFR transfected CHO-K1 cells were incubated with 100 ng/ml EGF for
 20 minutes. Lysates were blotted on nitrocellulose and probed with an α-EGFR antibody.

Subsequently, the nitrocellulose was stripped and analyzed using α-p-Tyr-100,
demonstrating phosphorylation of EGFR in transfected cells.







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Figure S2: Adhesion and invasion of CHO-K1 cells by encapsulated and unencapsulated meningococcal strain expressing or lacking Opc

CHO-K1 cells were infected with Opc+ and Opc- derivates of the encapsulated strain MC58 and the unencapsulated strain MC58 *siaD* for 4 hours and adhesion and invasion was estimated by gentamicin protection assay. Percentages of adhesion and invasion of Opc- derivates were compared to the Opc+ derivates. Values and S.D. are calculated from the results of three independent experiments done in duplicate. * P <0.05, ** P<0.01.

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31 Figure S3: CRM197 inhibited shedding of HB-EGF after PMA treatment

HBMEC were pretreated with CRM197 or were left untreated and stimulated with
250ng/ml Phorbol 12-myristate 13-acetate (PMA) for 1 hour and membrane-bound HBEGF was dissolved by washing with 1 .5 M NaCl/PBS/1% BSA. HB-EGF concentration
was quantified using a commercial HB-EGF-ELISA.