

Helicobacter pylori Avoids the Critical Activation of NLRP3 Inflammasome-Mediated Production of Oncogenic Mature IL-1 β in Human Immune Cells

Suneesh Kumar Pachathundikandi, Nicole Blaser, Heiko Bruns and Steffen Backert

Table 1. *H. pylori* wild-type strains and mutants used in this study.

Name	Gene	Function	Resistance	Reference
P1	wt	<i>cagPAI</i> ⁺ type-I strain	None	[1]
P12	wt	<i>cagPAI</i> ⁺ type-I strain	None	[2]
N6	wt	<i>cagPAI</i> ⁺ type-I strain	None	[3]
UK123	wt	<i>cagPAI</i> ⁻ type-II strain	None	M. Achtman & S. Suerbaum (unpublished)
UK097	wt	<i>cagPAI</i> ⁻ type-II strain	None	M. Achtman & S. Suerbaum (unpublished)
Ka148	wt	<i>cagPAI</i> ⁻ type-II strain	None	[4]
P1 Δ <i>napA</i>	<i>napA</i>	Neutrophil activating protein A	Chl	[5]
P1 Δ <i>ureA</i>	<i>ureA</i>	Urease subunit A	Chl	This study
P1 Δ <i>vacA</i>	<i>vacA</i>	Vacuolating cytotoxin A	Kan	[6]
P12 Δ <i>cagA</i>	<i>cagA</i>	T4SS effector protein	Chl	[7]
P12 Δ <i>cagL</i>	<i>cagL</i>	T4SS tip protein, integrin binding	Chl	[8]
P12 Δ <i>cagPAI</i>	<i>cagPAI</i>	Entire T4SS apparatus and effector protein CagA	Kan	[9]
P12 Δ <i>vacA</i>	<i>vacA</i>	Vacuolating cytotoxin A	Chl	[6]
P12 Δ <i>flaA</i>	<i>flaA</i>	Flagella monomer	Chl	[7]
P12 Δ <i>ggT</i>	<i>ggT</i>	γ -glutamyl transferase	Chl	This study
P12 Δ <i>hbp</i>	<i>hbp</i>	Phosphoheptose isomerase	Chl	This study
P12 Δ <i>hopQ</i>	<i>hopQ</i>	Outer membrane protein and adhesin	Chl	[10]
P12 Δ 1191	<i>Hp1191</i>	Glycosyl-transferase	Kan	[11]
P12 Δ <i>cgt</i>	<i>cgt</i>	Cholesterol-glycosyl transferase	Kan	This study
P12 Δ <i>slt</i>	<i>slt</i>	Lytic murein transglycosylase	Chl	This study
N6 Δ <i>htrA</i>	<i>htrA</i>	Secreted serine protease	Kan	[12]

Abbreviations: Chl: Chloramphenicol, Kan: Kanamycin, wt: Wild-type

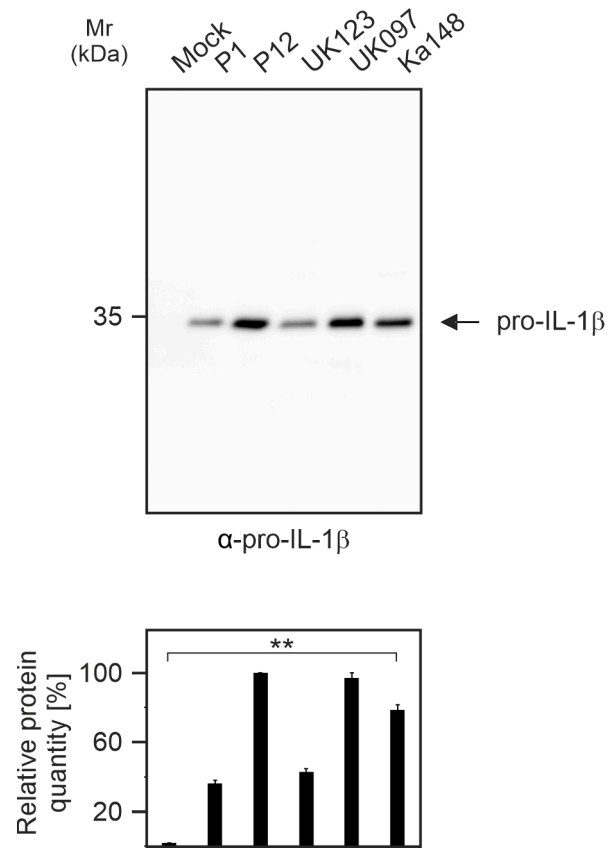


Figure S1: Full-size blot of Figure 1A and quantification of pro-IL-1 β protein expression in *H. pylori*-infected THP1 monocytes. Shown is the blot of Figure 1A detecting pro-IL-1 β using a specific antibody against pro-IL-1 β . The band intensities were quantified using ImageLab Software version 5.0 and shown as relative intensities. The strongest band was set as 100 %. ** $p \leq 0.01$

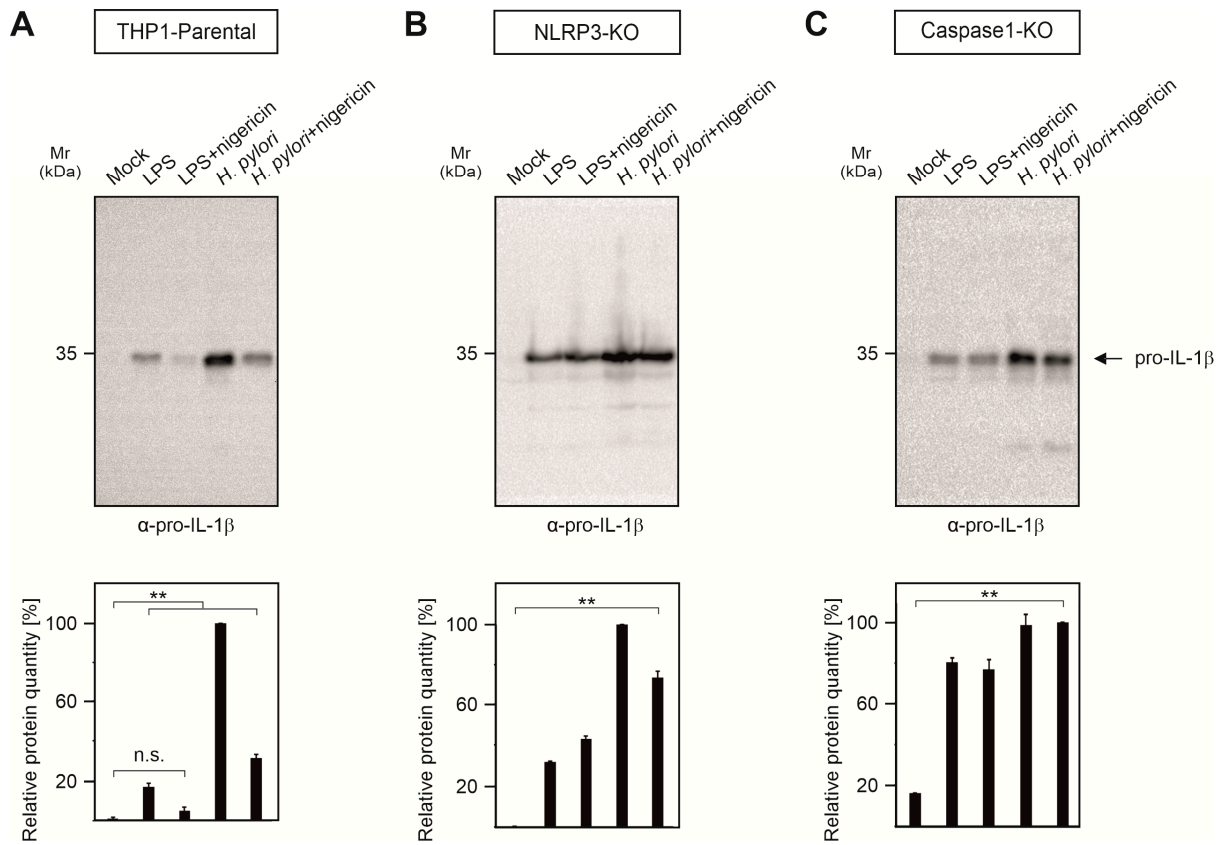
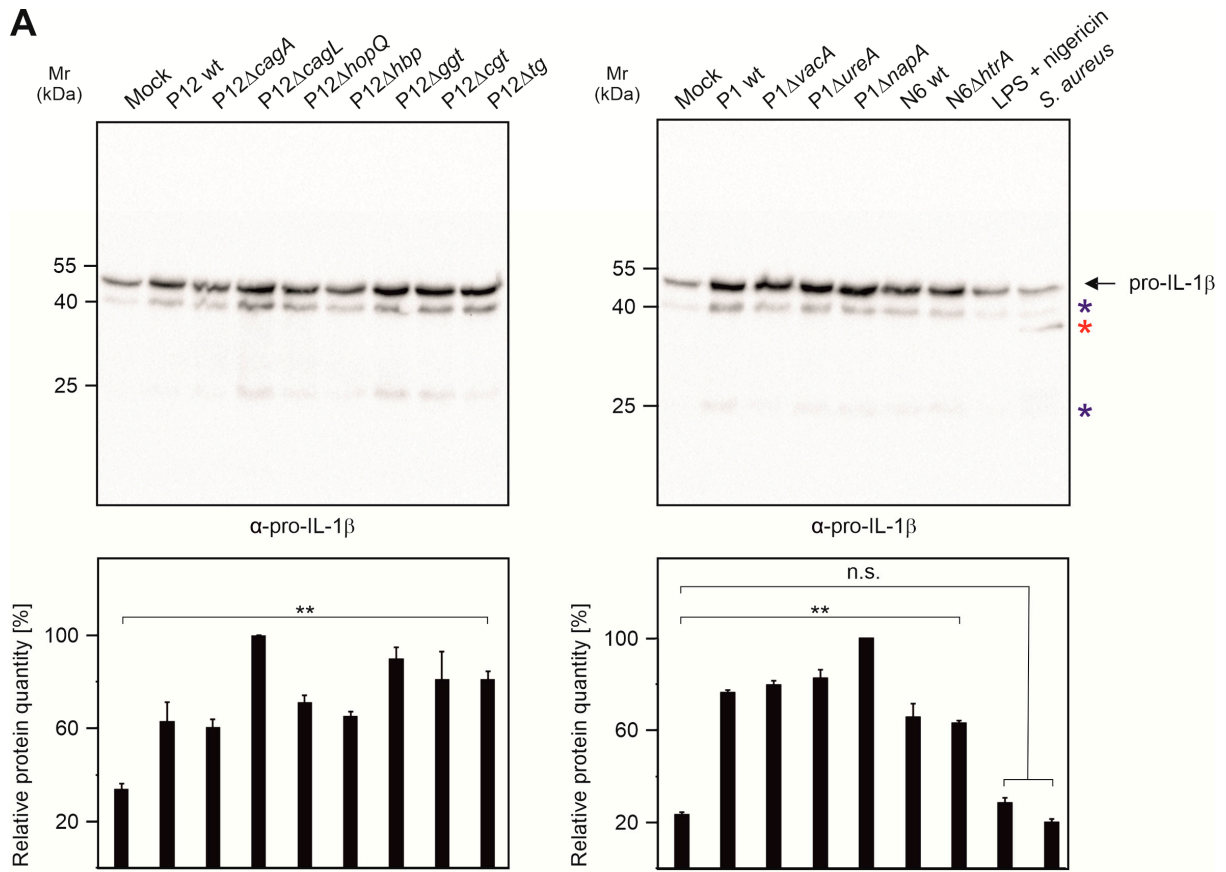
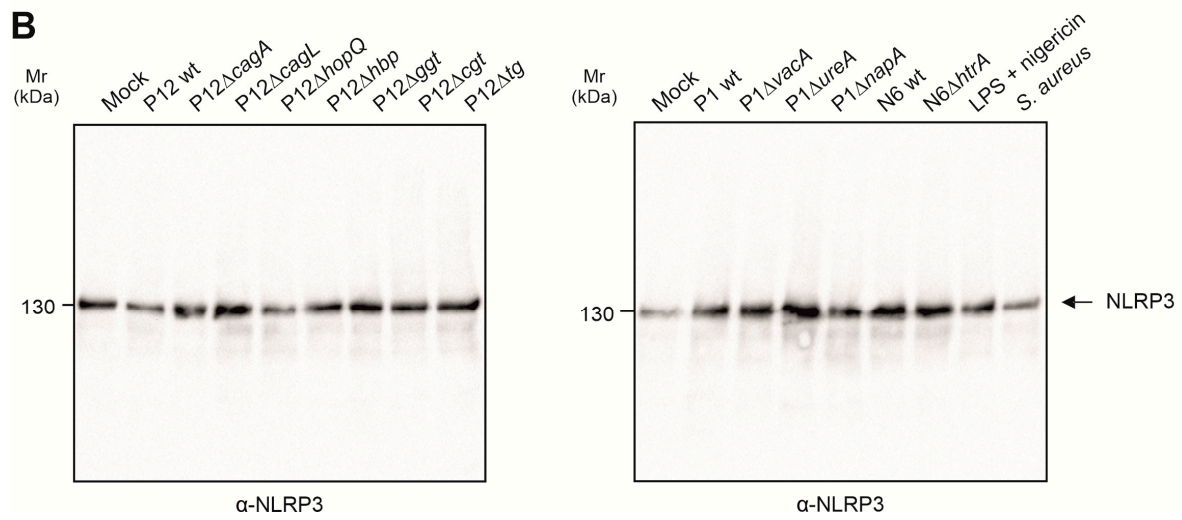


Figure S2: Full-size blots of Figure 3A and quantification of pro-IL-1 β protein expression in THP1 parental (A), THP1 NLRP3-KO (B) and THP1 Caspase-1-KO (C) monocytes. After treatment of the cells with *E. coli* LPS, *H. pylori* infection and NLRP3 inflammasome activation by nigericin, pro-IL-1 β protein expression was detected by Western blotting using a specific antibody. Using ImageLab Software version 5.0 the band intensities of pro-IL-1 β were quantified and relative amounts are displayed. The strongest band in each blot was set as 100 %. ** $p \leq 0.01$

A**B****Figure S3:** To be continued.

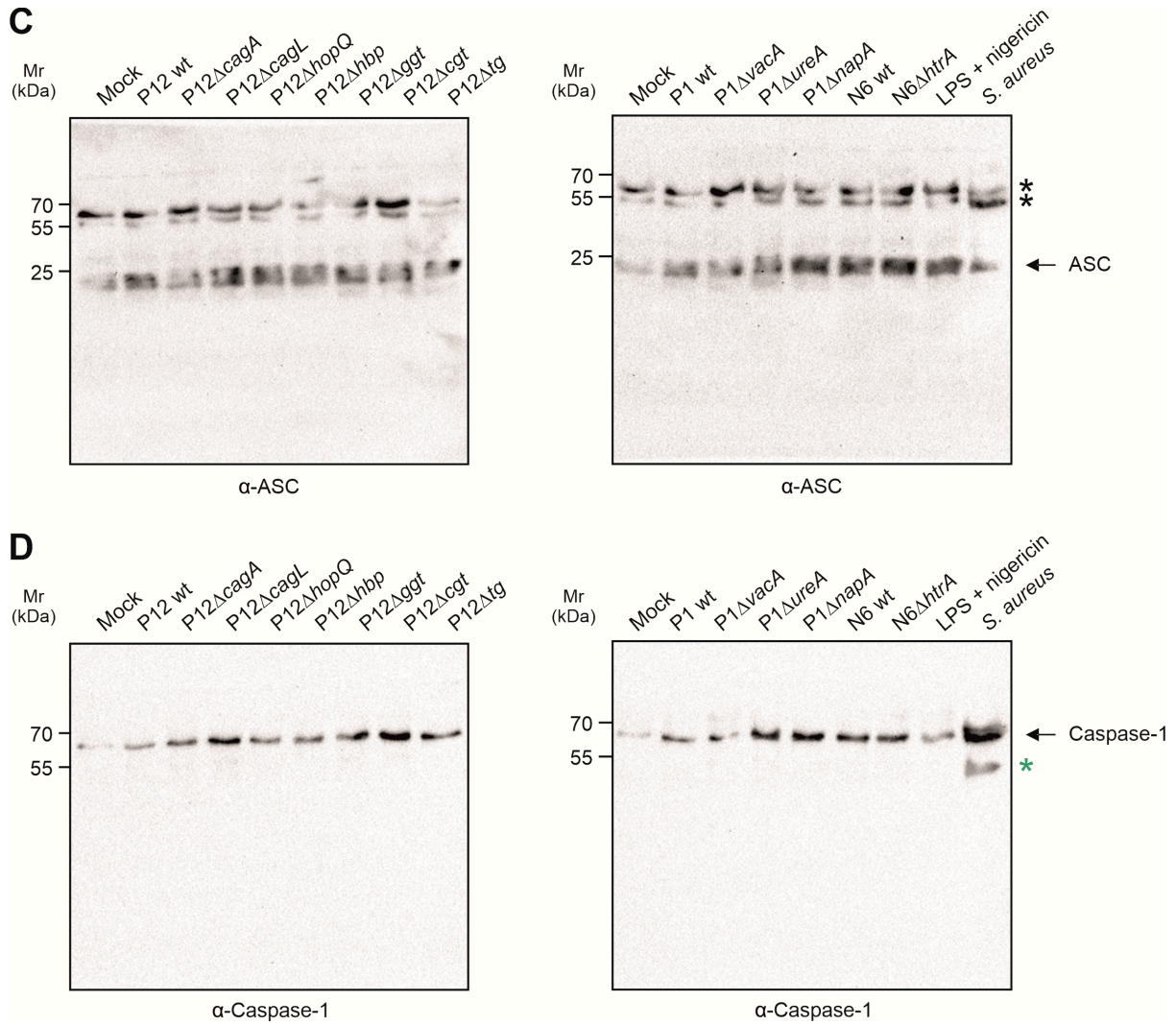


Figure S3: Full-size blots of Figure 7 and quantification of pro-IL-1 β protein expression in *H. pylori*-infected HEK293-NLRP3-INSOME cells. These cells were infected with *H. pylori* deletion variants or the corresponding wt strains. In addition, these cells were treated with *E. coli* LPS and nigericin or infected with *S. aureus*. Using Western blotting the pro-IL-1 β protein expression was detected by a specific antibody (A). A *S. aureus*-specific cleavage product of IL-1 β was detected (red asterisk). Additionally, cross-reaction of the antibody led to bands of unknown origin with a molecular weight of 40 kDa and 25 kDa (blue asterisk). The band intensities of pro-IL-1 β were quantified using ImageLab Software version 5.0 and shown as relative amounts. The strongest band was set as 100 %. As control, the protein expression of NLRP3 (B), ASC (C) and Caspase-1 (D) were investigated using specific antibodies. The antibody against ASC showed some cross-reactivity leading to a double band of unknown origin having a molecular weight between 55 kDa and 70 kDa (black asterisks). Detection of caspase-1 revealed a *S. aureus*-specific band with a molecular weight of 55 kDa (green asterisk). ** $p \leq 0.01$

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