



Differentiation of the virulence potential of three *Campylobacter jejuni* strains by use of gene expression analyses and a Caco-2 assay

Poli, Vanessa Fadanelli Schoenardie; Thorsen, Line; Olesen, Inger; Jespersen, Lene

Published in:

22nd International ICFMH Food Micro 2010

Publication date:

2010

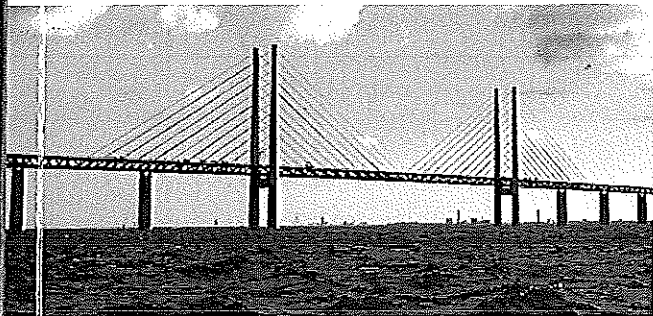
Document version

Early version, also known as pre-print

Citation for published version (APA):

Poli, V. F. S., Thorsen, L., Olesen, I., & Jespersen, L. (2010). Differentiation of the virulence potential of three *Campylobacter jejuni* strains by use of gene expression analyses and a Caco-2 assay. In *22nd International ICFMH Food Micro 2010* Copenhagen.

22nd International ICFMH Symposium Food Micro 2010



Copenhagen 30th August - 3rd September



Final Programme Et Abstract Book



www.foodmicro.dk

D2.15	Sondergaard T	PEB1.30	Sutherland JP	PEA2.18	Thorsen Line	PEA1.70
C1.44	Song EA	PEB2.10		PEB2.39	*Thorsen Line	PEB1.32
D2.41	Song KW	PEC1.42		PSA1.01	Thorup Cohn M	PEB2.21
A1.07	Sood R	PEB1.17	Sutyak, KE	PSA2.06	Thrane U	PEA2.44
A2.05	Soumaya Messaoudi	PEA1.33	Suzzi G	PEA1.56	Thuault D	PEC1.81
C1.65	Spaziani M	PEB2.17	Suzzi G	PEC1.78		PEC1.82
A1.59	Speybroeck N	PEC1.30	Svendsen C	PED2.50		PEC1.103
A2.41	Stabler R	PEB2.32	Svensson B	PEB2.06	Timan ADJ	PEB1.06
A2.46	Stabler R	PEB2.38	Svensson L	PEC1.92	Timke M	PEA2.29
D2.61	Stals A	PEC2.15	Sweeney T	PED1.01	Todorov S	PEA2.14
B1.31	Stals A	PEC2.48	Szlavik, Julie	PSD2.03	Todorov SD	PEA2.08
A2.03	Stamatiou A	PEC1.72	Söderholm Henna	PEB2.60	Todorov SD	PED2.48
B2.23	Stampelou I	PEC2.55	Söderholm, H	PSB2.01	Todorov Svetoslav	PEA2.23
A1.47	Stastkova Zora	PEC1.10	Sørensen G	PEC2.01	Tofalo Rosanna	PEA1.56
D2.24	Staufenbiel Anja	PEA2.06	Sørensen KI	PEA1.40		PEC1.78
D2.11	Stecchini M	PEB2.17	Sørensen LM	PEA1.10	Tomasevic I	PED2.49
B2.30	Stefanelli E	PEB2.37	Sørensen S	PEB1.21	Tomic N	PED2.49
B2.47	Stephan R	PED1.15	Sørensen SJ	PEE2.14	Tononi P	PEB2.37
B2.50	Stephan, R	PSB1.02,	Sørensen, SJ	PSE1.03	Torabi P	PEB1.27
C2.46	Stessl B	PEC1.95	Tabanelli G	PEA1.30	Torriani S	PEA1.29
B1.06		PEC1.98		PEE2.08		PEA1.30
D2.21		PEC1.99	Tahar A	PED2.01		PEB2.37
C1.63	Stevens G	PEE2.02	Taivosalo A	PEA1.15	Torrieri E	PED2.31
A1.17	Stevens, M	PSE1.01,	Tajbakhsh M	PEB1.27	Toyofuku Hajime	PEC2.18
C2.47	Steyn Cató	PED2.02	Talon R	PEA1.04	Tran-Dinh N	PEB2.56
D1.35		PED2.03	Talon R	PEB2.03	Traversa A	PEA2.42
D2.16	Stjepanovic Aleksandra	PEA1.17	Taminato F	PEB2.23	Trivedi Krina	PEA1.09
A1.48	Stonsaovapak S	PED2.11	Tanfani F	PEB2.17	Trivedi Krina	PEB1.05
D2.55	Stonsaovapak Siriporn	PEA1.08	Tango N	PEA1.68	Troianiello GD	PEA1.75
A2.44	Storm C	PED1.23	Tanner R	PEB1.03	Tromp, S-O	PSC2.02
C2.37	Storm Ida Marie		Tanner, S	PSE1.01,	Truchado P	PED2.42
D2.49	Lindhardt Drejer	PEA2.44	Tano-Debrah K	PEA1.36	Truelstrup Hansen L	PED2.07
D1.27	Strachan Norval	PEC2.29	Tano-Debrah K	PEA1.37	Tsai SM	PEA1.07
C2.06	Strachan, Norval	PSC2.06	Taoukis P	PEC1.87	Tsakalidou E	PEA1.11
B2.04	Strand Å	PEA1.57	Taoukis P	PEC1.96	Tsevdou Maria	PEC1.87
B1.11		PED1.31	Tarczynska AS	PEC1.27	Tsevdou Maria	PEC1.96
C1.84	Straver J	PEC2.37	Tarczynska AS	PEC1.28	Tsironi Theofania	PEC1.87
D2.52	Strini A	PED1.16	Tasara, Taurai	PSB1.02	Tudela, JA	PSD1.04
C1.51	Strydom Amy	PEC1.23	Tassoni A	PEA1.68	Tungtrakul P	PEA1.08
D1.27	Studeníková A	PEC1.14	Tassou C	PEC1.53	Turhan Ö	PEA2.16
C1.31	Stulova I	PEC1.24	Team RELU	PEC2.29	Turpin Williams	PEA1.44
C1.33	Stulova Hana	PEA1.15	Teixeira JA	PEA1.26	Uhrig S	PEE2.10
C1.81	Stüber E	PED1.11	Teixeira P	PEA2.41	Urbán Carrillo G	PEC2.38
C1.82	Stüber Elisabeth	PEB1.14	Teixeira P	PEB1.31	Uyttendaele M	PEC1.30
C1.03	Suba S	PEB1.17	Tekin E	PEA1.47		PEC1.46
C2.48	Subires Alicia	PEB2.28	Tempelaars M	PEB2.29		PEB2.07
C2.44	Subires, Alicia	PSB2.03,	Tenehaus F	PEC2.37		PEC2.07
C2.62	Sudharshana MR	PED1.10	Ter Beek A	PEB2.04		PEC2.15
C1.06	Sugita-Konishi Y	PEB1.13	Tersteeg-Zijderveld MHG	PEB1.06		PEC2.18
C2.21	Suhajda Á	PED1.19	Theron MM	PEC1.51		PEC2.35
C2.24	Suhajda Á	PED1.20	Thevenot-Sergentet D	PEC1.22		PEC2.36
C2.11	Susitha K	PED2.24	Thiel S	PEC2.23		PEC2.48
C2.13	Sutherland Jane P	PEC2.28	Thierry A	PEE1.01		PEC2.56
C2.14			Thierry, A	PSA1.06		PED2.49
C2.01,			Thorsen L	PED2.50		

ative *Clostridium*

ctor for the devel-
75 -toxin. The aim
etB toxin in a well
nce with observa-
ealthy birds, with
e coding sequence
itro NetB produc-
whereas 12 out of
ther confirms nor
posed that gene-
ure investigations
d potentially also

d industry

le Católica Por-

the hands of 162
ilase, DNase and
1.6% in the nose
linase, hemolysis
es were sensitive
5, 4.6, 12.1, 13.6,
icillin and nitro-
since they were
most prevalent.
d to prevent the

- X PEB1.32 Differentiation of the virulence potential of three *Campylobacter jejuni* strains by use of gene expression analyses and a Caco-2 assay
V Fadanelli Schoenardie (1), Line Thorsen (1), I Olesen (1), L Jespersen (1)
(1) Faculty of Life Sciences, Copenhagen University, Denmark

Campylobacter jejuni is the leading cause of bacterial diarrhoeal disease in humans and has become part of the most important concerns in food safety. Contaminated poultry and poultry products are recognized as the main vehicle of infection. Despite the significance of *C. jejuni* as a foodborne pathogen, little is known about its response to stressful conditions, and, especially, about how it modulates its virulence under such stresses. The aim of the present study was to assess the effect of temperature shift in a broth model system on virulence expression and cell survival of three *Campylobacter jejuni* strains: a clinical isolate (TB1048), a sequenced clinical strain (NCTC11168) and a chicken isolate (DFVF1099). Firstly, cells were transferred from 42 to 4°C to investigate the effect of low temperature storage for short (30 min) and long (24 h) periods of time. Then, the effect of a shift in temperature from 4 to 37°C for 30 min was observed. The virulence properties of *C. jejuni* were evaluated by quantitative Real Time-PCR (qRT-PCR) analysis of the expression of the virulence associated genes *cdtB*, *ciaB*, *cadF* and *clpP*, and by its ability to adhere to and invade Caco-2 cells. The results obtained in this study indicated cell survival and growth inhibition for all strains at 4°C, and no change in cell counts was observed after transfer to 37°C for 30 min. From the virulence perspective, interstrain variation was observed. The expression level of *cdtB* and *clpP* were significantly up-regulated in only one strain (NCTC11168) and invasion ability into Caco-2 cells was observed in the clinical strains only. After exposure to temperature stress, none of the three strains showed significant difference in adhesion and invasion properties as compared to unstressed cells. This was also displayed by the qRT-PCR analysis of the *cadF* and *ciaB* genes, which are known to be involved in the adhesion and invasion process of *C. jejuni*. As a conclusion, the qRT-PCR analyses and Caco-2 assay showed to be useful tools for differentiating the virulence potentials of the three investigated *C. jejuni* strains under growth conditions where the cell survival was similar. Generally a low storage temperature is not enough to control the survival and virulence of *C. jejuni*.

- PEB1.33 The functional importance of Bacterial lysozyme Inhibitors
Lien Callewaert (1), L Vanderkelen (1), JM Van Herreweghe (1), E Ons (1), BM Goddeeris (1), CM Michiels (1)
(1) Katholieke Universiteit Leuven, Belgium

Lysozymes are ancient and important components of the innate immune system of animals that exert an antibacterial activity by hydrolysing peptidoglycan, the major bacterial cell wall polymer. Three major lysozyme types have been identified in the animal kingdom, commonly designated as the c-type (chicken type), the g-type (goose type) and the i-type (invertebrate type) lysozyme. Although their phylogenetic distribution and expression patterns vary greatly among animals, the defensive role lysozymes against pathogenic bacteria is widely recognised and well documented in different host organisms. From this point of view, it is not surprising that bacteria have in turn evolved mechanisms to evade or subvert the action of lysozyme, f.ex. by producing specific lysozyme inhibitors. Using dedicated function-based screenings for inhibitors of each of the three main animal lysozyme types, we have identified three novel families of lysozyme inhibitors corresponding to each of the three lysozyme families. Although these families of inhibitors lack significant overall similarity at amino acid level, they appear to share a common conserved motif that may be involved in binding to active site residues of lysozyme. This strongly suggests a common ancestral origin for these inhibitors.

Regarding to their function, we demonstrated that knockout of inhibitor production renders bacteria more sensitive to the corresponding lysozyme. Furthermore, challenge experiments using c-type lysozyme inhibitor-defective mutants of APEC (avian pathogenic *Escherichia coli*) in the chicken indicated an important contribution of this lysozyme inhibitor in the virulence of this pathogen. Interestingly, we have strong indications that the g-type lysozyme inhibitor also inhibits a bacterial autolysin that is related to g-type lysozyme, suggesting a role for this inhibitor family in the regulation of autolysin activity. In conclusion, these newly identified lysozyme inhibitors occur in a wide range of gramnegative bacteria, probably originate from a common ancestor, and may have different functions including defence against animal host lysozyme and regulation of autolysin activity. The study of lysozyme inhibitors will provide new insights in bacterial physiology and ecology, and they may constitute an attractive novel target for antibacterial drug development.