



## Phage therapy reduces *Campylobacter jejuni* colonization in broilers

Jaap A. Wagenaar<sup>a,\*</sup>, Marcel A.P. Van Bergen<sup>a</sup>, Mark A. Mueller<sup>b</sup>,  
Trudy M. Wassenaar<sup>c</sup>, Richard M. Carlton<sup>b</sup>

<sup>a</sup> Division of Infectious Diseases, Animal Sciences Group, P.O. Box 65, 8200 AB Lelystad, The Netherlands

<sup>b</sup> Exponential Biotherapies Inc., Washington DC, USA

<sup>c</sup> Molecular Microbiology and Genomics Consultants, Zotzenheim, Germany

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### Abstract

The effect of phage therapy in the control of *Campylobacter jejuni* colonization in young broilers, either as a preventive or a therapeutic measure, was tested. A prevention group was infected with *C. jejuni* at day 4 of a 10-day phage treatment. A therapeutic group was phage treated for 6 days, starting 5 days after *C. jejuni* colonization of the broilers had been established. Treatment was monitored by enumerating *Campylobacter* colony forming units (CFU) and phage plaque forming units (PFU) from caecal content. Counts were compared with control birds not receiving phage therapy. A clear 3 log decline in *C. jejuni* counts was initially observed in the therapeutic group, however, after 5 days bacterial counts stabilized at a level 1 log lower than that of the control group. Colonization of *C. jejuni* in the prevention group was delayed by the treatment and after an initial 2 log reduction, colonization stabilized within a week at levels comparable to the therapeutic group. The CFU and PFU counts displayed opposing highs and lows over time, indicative of alternating shifts in amplification of bacteria and phages. There were no adverse health effects from the phage treatment. Two different phages were combined as therapeutic treatment of *Campylobacter* positive chickens challenged at the age approaching broiler harvest. This again resulted in a significant decrease in *Campylobacter* colonization. We conclude that phage treatment is a promising alternative for reducing *C. jejuni* colonization in broilers.

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### 1. Introduction

*Campylobacter jejuni* is an important human pathogen and is the most common cause of bacterial

gastroenteritis worldwide (Lindqvist et al., 2001; Adak et al., 2002; Samuel et al., 2004). Poultry flocks are frequently colonized with *C. jejuni* without apparent symptoms (Shane, 2000) and risk assessment analyses have identified handling and consumption of poultry meat as one of the most important sources of human campylobacteriosis (Evans et al., 2003; Potter et al., 2003; Friedman et al., 2004).

\* Corresponding author. Tel.: +31 320 238157;  
fax: +31 320 238961.

E-mail address: [Jaap.Wagenaar@wur.nl](mailto:Jaap.Wagenaar@wur.nl) (J.A. Wagenaar).