

Role of *dprA* in transformation of *Campylobacter jejuni*

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Abstract

The role of a *dprA* ortholog (Cj0634) in *Campylobacter jejuni* transformation was phenotypically assessed using two strains. *C. jejuni* strain 11168 was naturally competent for transformation by chromosomal DNA, while efficiency decreased 100-fold in a Cj0634::*aphA* mutant, whereas *C. jejuni* strain 480 was not naturally competent. *C. jejuni* strain 480 but not 11168 could be electro-transformed by shuttle plasmid pRY111, an effect completely abolished by Cj0634 interruption. Complementation of the Cj0634 mutation in *C. jejuni* strain 480 *in trans* with vectors containing the *dprA* homologs from *C. jejuni*, *Helicobacter pylori*, or *Haemophilus influenzae*, completely (for Cj0634) or partially (*H. pylori* > *H. influenzae*) restored electro-transformation. Thus, *C. jejuni* expresses a DprA ortholog that functionally most closely resembles that of *H. pylori* and is involved in DNA transformation.

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1. Introduction

Campylobacter jejuni is a gram negative microaerophilic bacterium that colonizes the gastrointestinal tract in avian and mammalian species, and is a common cause of diarrheal diseases in humans [1]. The genetically heterogeneous *C. jejuni* are naturally competent to take up and integrate DNA [2,3]; this heterogeneity may result, at least in part, from intraspecies horizontal gene transfer. The ability for horizontal DNA transfer may be an

important characteristic enabling survival of *C. jejuni* in various natural hosts. Characterization of the factors involved in *C. jejuni* DNA transfer may lead to insights that are generally applicable to other species.

Prior work by our group had drawn our attention to DprA as one of the factors involved in bacterial uptake and integration of DNA. Analysis of the complete genome sequence of *C. jejuni* 11168 [4] revealed a 774-bp open reading frame (Cj0634) predicted to encode a protein with homology to DprA (encoded by *dprA*) of *Haemophilus influenzae* and *Helicobacter pylori*. In *H. influenzae*, DprA is essential for transformation by chromosomal DNA [5], whereas in *H. pylori*, DprA facilitates but is not essential for transformation by chromosomal DNA, and is necessary for plasmid uptake [6,7].

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