

pEntYN10 a plasmid of *Escherichia coli* O169:H41 associated with adherence and toxin production

Armando Navarro*

Departamento de Salud Pública; Facultad de Medicina; Universidad Nacional Autónoma de México, México City, México

Keywords: E coli O169:H41, ETEC, pEntYN10 plasmid

In this issue of *Virulence*, Ban E *et al.* described the complete sequence of the pEntYN10 plasmid of *Escherichia coli* O169:H41. The plasmid has 182 coding sequences (CDs); among the identified CDs, some were identified as colonization factors (CFs), one was an enterotoxin and others were insertion sequences (IS). In this editorial, the main findings, as well as the epidemiological significance and spontaneous loss of pEntYN10 plasmid of *E. coli* O169:H41 strains are discussed.

Ban *et al.*¹ described the complete sequence results of the pEntYN10 plasmid of *Escherichia coli* O169:H41, which was isolated from an epidemic outbreak of diarrhea in Osaka, Japan in 1991. Since then, other outbreaks have been reported in different countries including the Republic of Korea,² Japan³ and the USA.⁴ From 1996 to 2003, 16 epidemic outbreaks related to the transmission of enterotoxigenic *E. coli* (ETEC) in food were reported in the USA, and 10 of these were associated with ETEC O169:H41.⁴ These observations suggest that ETEC-related diarrhea is no longer just a problem of travelers to developing countries, but it is now an endemic problem in the region as a whole.⁵ In Japan, where the association of *E. coli* O169:H41 with food borne intestinal infection was first identified in 1991,^{6,7} a series of new epidemic outbreaks of *E. coli* O169:H41-related diarrhea were reported.^{3,8} Recently in the Republic of Korea (2012) in the area of Incheon City, an outbreak of intestinal infection related to *E. coli* O169² was reported in 7 schools that affected 230 students after eating Kimchi distributed by a food manufacturer. Infections produced by ETEC strains are a worldwide problem and this pathotype is considered to be the second most important etiological

agent responsible for infantile diarrhea in the world, after Rotavirus and *Shigella*.⁹ Ban *et al.*¹ describe the complete sequence of the pEntYN10 plasmid identified in the *E. coli* O169:H41 strain. Its analysis revealed that the plasmid has 145,082 base pairs with 182 coding sequences (CDs), and contains 43.15% guanine and cytosine. Among the coding sequences, some were identified as colonization factors (CFs), one was an enterotoxin and others were insertion sequences (IS). Furthermore, the plasmid presented *psiAB* and *relE* genes both related to stability but different from those (*stbAB*, *psiAB*, *relE*, *mok/hok/soc*) found in ETEC H10407 (O78:H11). With respect to the colonization factors, the authors found the presence of CS6, CS8 (CFAIII)-like, K88 (F4)-like encoded redundantly in the plasmid. In the case of CS8 (CofA) that shows a high structural homology with TcpA from *Vibrio cholerae* and had a similar amino acid sequence to that of the pSH1134 plasmid from ETEC 206-1 strain. In addition, 2 paralogous copies of the *faeG* gene related to the main component of the fimbrial structure of K88 were identified; the structure was similar to *Salmonella enterica* serovar Infantis. Despite the presence of *psiAB* and *relE* genes, which are related to the stability of the

pEntYN10 plasmid, stability is lost fairly easily after passing the *E. coli* YN19 strain 7 times through trypticase soy broth (TSB). Analysis of the clones that lost the plasmid showed that ST toxin expression, as well as the adherence capacity of the aggregative pattern in HEP-2 cells, disappeared. Based on this last observation, the authors proposed that the genes present in pEntYN10 contribute to bacterial adherence to HEP-2 cells, probably related to the expression of CS6, CS8 (CFAIII)-like and K88 (F4)-like. Previous reports relating to *E. coli* O169:H41 strains isolated from epidemic outbreaks in Japan³ in which the spontaneous loss of a 100 MDa plasmid was observed, concluded that this loss was associated with the inability to produce the ST toxin and adherence of the aggregative pattern to HEP-2 cells. Although these observations are in keeping with those reported by Ban *et al.*¹ more studies are required to define the participation of the plasmids in the expression properties of *E. coli* O169:H41 wild strains as they relate to the bacteria's virulence.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

© Armando Navarro

*Correspondence to: Armando Navarro; Email: arnava@unam.mx

Submitted: 10/02/2015; Accepted: 10/02/2015

<http://dx.doi.org/10.1080/21505594.2015.1104450>

Comment on: Ban E, Yoshida Y, Wakushima M, Wajima T, Hamabata T, Ichikawa N, Abe H, Horiguchi Y, Hara-Kudo Y, Kage-Nakadai E, Yamamoto T, Wada T, and Nishikawa Y. Characterization of unstable pEntYN10 from enterotoxigenic *Escherichia coli* (ETEC) O169:H41; <http://dx.doi.org/10.1080/21505594.2015.1094606>

This is an Open Access article distributed under the terms of the Creative Commons Attribution-Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited. The moral rights of the named author(s) have been asserted.

References

1. Ban E, Yoshida Y, Wakushima M, Wajima T, Hamabata T, Ichikawa N, Abe H, Horiguchi Y, Hara-Kudo Y, Kage-Nakadai E, et al. Characterization of unstable pEntYN10 from enterotoxigenic *Escherichia coli* (ETEC) O169:H41. *Virulence* 2015.
2. Cho SH, Kim J, Oh KH, Hu JK, Seo J, Oh SS, Hur MJ, Choi YH, Youn SK, Chung GT, et al. Outbreak of enterotoxigenic *Escherichia coli* O169 enteritis in schoolchildren associated with consumption of kimchi, Republic of Korea, 2012. *Epidemiol Infect* 2014; 142:616–23; PMID:23800632; <http://dx.doi.org/10.1017/S0950268813001477>
3. Nishikawa Y, Helander A, Ogasawara J, Moyer NP, Hanaoka M, Hase A, Yasukawa A. Epidemiology and properties of heat-stable enterotoxin-producing *Escherichia coli* serotype O169:H41. *Epidemiol Infect* 1998; 121:31–42; PMID:9747753; <http://dx.doi.org/10.1017/S0950268898001046>
4. Beatty ME, Bopp C A, Wells JG, Greene K D, Puhf ND, Mintz ED. Enterotoxin-producing *Escherichia coli* O169: H41, United States. *Emerg Infect Dis* 2004; 10:518–21; PMID:15109427; <http://dx.doi.org/10.3201/eid1003.030268>
5. Devasia RA, Jones TF, Ward J, Stafford L, Hardin H, Bopp C, Beatty M, Mintz E, Schaffner W. Endemically acquired foodborne outbreak of enterotoxin-producing *Escherichia coli* serotype O169: H41. *Am J Med* 2006; 119:168-e7-10; PMID:16443428; <http://dx.doi.org/10.1016/j.amjmed.2005.07.063>
6. Ando K, Itaya T, Aoki A, Saito A, Masaki H, Tokumura Y. An outbreak of food poisoning caused by enterotoxigenic *Escherichia coli* O169:H41. *Jpn J Food Microbiol* 1993; 10:77–81.
7. Nishikawa Y, Hanaoka M, Ogasawara J, Moyer NP, Kimura T. Heat-stable enterotoxin-producing *Escherichia coli* O169: H41 in Japan. *Emerg Infect Dis* 1995; 1:61; PMID:8903162; <http://dx.doi.org/10.3201/eid0102.950206>
8. Harada T, Itoh K, Yamaguchi Y, Hirai Y, Kanki M, Kawatsu K, Kumeda Y. A foodborne outbreak of gastrointestinal illness caused by enterotoxigenic *Escherichia coli* serotype O169:H41 in Osaka, Japan. *Jpn J Infect Dis* 2013; 66:530–3; PMID:24270144; <http://dx.doi.org/10.7883/yoken.66.530>
9. Huilan S, Zhen LG, Mathan MM, Mathew MM, Olarte J, Espejo R, Khin Maung U, Ghafoor MA, Khan MA, Sami Z. Etiology of acute diarrhoea among children in developing countries: a multicentre study in five countries. *Bull World Health Organ* 1991; 69:549–55; PMID:1659953