Surveillance of *Bacillus cereus* Isolates in Korea from 2012 to 2014

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**Objectives:** To investigate the prevalence and toxin production characteristics of non-emetic and emetic *Bacillus cereus* strains isolated via the laboratory surveillance system in Korea.

**Methods:** A total of 667 *B. cereus* strains were collected by the Korea National Research Institute of Health laboratory surveillance system from 2012 to 2014. The collected strains were analyzed by geographical region, season, patient age, and patient sex. Additionally, the prevalence rates of enterotoxin and emetic toxin genes were evaluated.

**Results:** The isolation rate of *B. cereus* strains increased during the summer, but the isolation rate was evenly distributed among patient age groups. Emetic toxin was produced by 20.2% of the isolated strains. The prevalence rates of five enterotoxin genes (*entFM*, *nheA*, *cytK2*, *hblC*, and *bceT*) were 85.0, 78.6, 44.5, 36.6, and 29.7%, respectively, among non-emetic strains and 77.8, 59.3, 17.8, 11.9 and 12.6%, respectively, among emetic strains. Thus, the prevalence rates of all five enterotoxin genes were lower in emetic *B. cereus*.

**Conclusion:** The prevalence of enterotoxin genes differed between non-emetic and emetic *B. cereus* strains. Among emetic *B. cereus* strains, the prevalence rates of two enterotoxin genes (*cytK2* and *hblC*) were lower than those among the non-emetic strains. In both the emetic and non-emetic strains isolated in Korea, *nheA* and *entFM* were the most prevalent enterotoxin genes.

**Key Words:** *Bacillus cereus*, epidemiology, enterotoxins

**INTRODUCTION**

Foodborne diseases represent a serious threat to public health. Diarrheal disease is significantly increasing in prevalence worldwide year on year [1]. The bacterium *Bacillus cereus* is widely present in nature and can survive in harsh environments. *B. cereus* causes two types of gastrointestinal diseases: emesis and diarrhea. It produces one emetic toxin (cereulide) and several enterotoxins (*hblC*, *hblD*, *hblA*, *nheA*, *nheB*, *nheC*, *cytK2*, *entFM*, and *bceT*). Both the emesis and diarrhea caused by *B. cereus* are generally mild and self-limiting, although more serious and even lethal cases have occurred [2–4]. The diarrheal type is attributed to single or multiple enterotoxins. Particularly, a group of proteins including two heat-labile toxins and a three-component hemolysin (HBL; consisting of three proteins: B, L1, and L2) with enterotoxin activity have been purified and characterized [5]. Additionally, non-hemolytic enterotoxin (NHE, encoded by *nheA*, *nheB*, and *nheC*) is a key component contributing to *B. cereus*-mediated diarrhea [6,7]. Furthermore, single-component toxins, such as enterotoxin T (*bceT*) [8], enterotoxin FM (*entFM*) [9], and cytotoxin K (*cytK*) [10] are thought to be involved in *B. cereus* food poisoning. The pore-forming toxin, *cytK*, has two different forms, *cytK1* and *cytK2*, which have 89% amino acid...
sequence homology [11,12]. Emesis is caused by a single heat-stable toxin, cereulide, which is produced in food [8,13,14]. This toxin is enzymatically synthesized by non-ribosomal peptide synthesis, and its genetic determinants are located within a 23-kb gene cluster (ces) on a large plasmid [2,15–17].

Several countries have reported B. cereus outbreaks [18–20]. Vomiting-type food poisoning is 10 times more prevalent than diarrheal-type food poisoning in Japan. However, in North America and Europe, diarrheal-type B. cereus infection is most frequent [21]. In Korea, 27 food poisoning outbreaks associated with B. cereus were reported from 2001 to 2008, but few cases of vomiting-type food poisoning caused by B. cereus were reported [22].

Emetic-type food poisoning caused by B. cereus occasionally includes symptoms of vomiting and diarrhea [7]. Therefore, the characterization of the enterotoxins produced by emetic B. cereus is necessary to obtain a better understanding of the food poisoning caused by this organism and to prevent misdiagnosis between diarrheal and emetic food poisoning.

EnterNet-Korea, an acute diarrheal laboratory surveillance system, was established in 2007 to improve laboratory activities and enhance reporting proficiency. This surveillance system is coordinated by the Korea National Research Institute of Health (NIH) and comprises 17 local public health institutes and 105 participating hospitals. The target pathogens include 10 genera of bacteria and 5 types of viruses.

In this study, we analyzed the isolation trends and toxin gene profiles of B. cereus strains obtained via Enter-Net Korea from 2012 to 2014.

### MATERIALS AND METHODS

#### 1. Isolation of B. cereus from clinical samples

A total of 57,050 stool samples were collected by Enter-Net Korea from 2012 to 2014. The Enter-Net system is coordinated by the Korean NIH and comprises 17 local public health institutes and 105 participating hospitals. Stool samples were collected from patients who had diarrheal symptoms, and a total of 667 B. cereus strains were isolated from these samples.

We determined the isolation rate of B. cereus from the stool specimens during each 12-month period. Next, we divided patients into eight categories by age (< 10, 10–19, 20–29, 30–39, 40–49, 50–59, 60–69, and > 70 years) and surveyed the age and gender distributions of B. cereus isolation rates.

Mannitol-egg yolk-polymyxin B agar (Oxoid, Basingstoke, UK) was used as a selective medium for B. cereus isolation. For primary identification, the isolates were characterized by standard physiological and biochemical tests using the API® 50CHB and API 20E® bacterial identification systems (bioMérieux, Marcy-l’Étoile, France).

#### 2. Polymerase chain reaction (PCR) amplification of enterotoxin and emetic toxin genes

DNA from each isolate was extracted using the Maxwell® 16 System Purification Kit (Promega, Madison, WI, USA) in accordance with the manufacturer’s instructions. All PCRs were performed using the Expanded High Fidelity Polymerase System (Roche, Basel, Switzerland) or Taq polymerase (Takara Bio, Otsu, Japan) according to the manufacturer’s instructions. The PCR primer sequences used in this study are shown in Table 1.

| Target gene | Sequence (5’ to 3’) | Size (bp) | Reference |
|-------------|---------------------|-----------|-----------|
| entFM       | CAAAAGACTTCTGTAACAAAAAGGTGTT | 290 | Yang et al, 2005 [7] |
|             | TGTTTACTCCGCCCTTTTCAAACCTT | | |
| nheA        | ATTACAGGGTTATGGTTACAGCAGT | 475 | Yang et al, 2005 [7] |
|             | AATCTTGCTCCATACTCTCTGTGGATGCT | | |
| cytK2       | CAATCCCTGGGCTGCTAGTCA | 585 | Guinebretirere et al, 2006 [12] |
|             | GTGTAGCCGTGACGAAAGTTGG | | |
| hblC        | CCTATCAATCTCTCGCAACACCAAAT | 386 | Yang et al, 2005 [7] |
|             | TTTTCTTGTATTGCAGCAATTCTTCT | | |
| bceT        | AGCTTGGACGGAGCGACAGACTATGT | 701 | Yang et al, 2005 [7] |
|             | GTATTTCTTCTGCTGCTTCTTCTT | | |
| cer         | ATCATAAGGTGCGAAACAGA | 188 | Kim et al, 2010 [22] |
|             | AAGATCAACCGAATGCAAATCG | | |
hblC, nheA, entFM, and bceT were amplified according to the methodology described by Yang et al [7]; cytK2 was amplified as described by Guinebretiere et al [12]; and cereulide peptide synthetase (cer) was amplified as described by Kim et al [23].

3. Statistical analysis

The collected data were analyzed using IBM SPSS Statistics ver. 21.0 (IBM Co., Armonk, NY, USA). The distribution of toxin genes from non-emetic and emetic isolated strains was analyzed using the chi-square or chi-square trend test. For statistical analysis, differences at \( p < 0.05 \) were considered to be significant.

RESULTS

1. B. cereus isolation rates

A total of 667 (6.9%) B. cereus strains were isolated from 57,050 stool samples by Enter-Net. The isolation rate of B. cereus slightly increased in the summers of 2012 and 2013, with strong seasonality in 2014 (Figure 1A). The age distribution of isolation rates showed no specific trend from 2012 to 2014 in any of the eight age groups (Figure 1B). Lastly, we divided the isolation rates by gender and found that females showed a slightly higher isolation rate than males, albeit with no statistically significant difference (Figure 1C).

2. Detection of toxin genes by PCR

In this study, five enterotoxin genes (hblC, bceT, cytK2, entFM, and nheA) and one emetic toxin gene (cer) were detected using PCR (Table 1). Among all 667 strains, the prevalence rates of entFM, nheA, cytK2, hblC, bceT, and cer were 85.0, 78.6, 44.5, 36.6, 29.7, and 20.2%, respectively (Table 2). The emetic toxin-producing B. cereus strains comprised 20.2% of total strains isolated (n = 135). The prevalence rates of the assessed enterotoxin

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**Figure 1.** Isolation rates of Bacillus cereus by (A) year of isolation, (B) patient age, and (C) patient gender in Korea, 2012–2014.

[Insert Figure 1 here]
Values are presented as number (%).

rs of five enterotoxin genes (entFM, nheA, cytK2, hblC, and bceT) among the emetic toxin-producing B. cereus strains were 77.8, 59.3, 17.8, 11.9, and 12.6%, respectively, lastly. Lastly, the prevalence rates of the five aforementioned enterotoxin genes among the non-emetic B. cereus strains were 86.8, 83.5, 51.3, 42.9, and 34.0%, respectively (Table 2).

3. Profiling of toxin genes

According to the presence or absence of enterotoxin genes, B. cereus strains harboring emetic and enterotoxin genes in our study could be divided into 29 and 20 groups, respectively (Table 3). In the B. cereus strains harboring the enterotoxin gene, the most common toxin profile was group F (entFM+, nheA+, hblC-, cytK2+, and bceT+). Group F was detected in 41.5% of these strains. The next most common gene profile was group G (entFM+), which was detected in 16.3% of enterotoxin-producing strains. Approximately 2% to 4% of B. cereus strains harboring the enterotoxin gene contained two to six toxin genes in total (Table 3). B. cereus strains harboring the enterotoxin gene, which lacked cer genes, were more diverse than B. cereus harboring the enterotoxin gene. Among non-emetic B. cereus strains, the group H toxin profile, which lacked cytK2, hblC, and bceT, was the most common (detec-
tion rate, 23.5%). The second most common toxin profile group was group A (14.5%, containing all five enterotoxin genes). Approximately 0.2% to 13% of non-emetic strains contained one to five toxin genes.

DISCUSSION

In our study, 667 (6.9%) B. cereus strains were isolated from 57,050 stool samples. The isolation rate of B. cereus increased with rising temperatures and peaked from June to September, with an especially pronounced summer peak in 2014. Additionally, the isolation rate of B. cereus by age was evenly distributed.

We surveyed the distribution of toxin genes among non-emetic strains (n = 532) and emetic strains (n = 135). The prevalence rates of five enterotoxin genes (entFM, nheA, cytK2, hblC, and bceT) were quite different between groups at 86.8, 83.5, 51.3, 42.9, and 34.0%, respectively, amongst non-emetic strains and 77.8, 59.3, 17.8, 11.9, and 12.6%, respectively, among emetic strains. The most prevalent toxin genes in the non-emetic strains were entFM and nheA (23.5%, group H, 125 strains), and those in the emetic strains were entFM and nheA (41.5%, group F, 56 strains). Non-emetic strains showed highly diverse toxin gene profiles (29 patterns) while emetic strains showed less diversity (20 patterns). Based on our results, we deduced that B. cereus has a high level of genetic diversity in Korea. Toxin gene profiling studies of strains from the environment and food have produced some different results from those of our survey of clinical strains. In particular, cytK and emetic toxin were not found in environmental isolates from silo tanks, and isolates from food dominantly harbored the nhe toxin gene only [24]. In another report, the most prevalent toxin genes in isolates from food were nhe and entFM [17]. In addition, a severe foodborne outbreak of diarrheal disease caused by B. cereus strains harboring cytK was reported [25,26].

As our next steps, we plan to survey the molecular profiles of all isolates using multiple locus sequence typing and/or pulsed-field gel electrophoresis. Our results are in accordance with those of previous studies conducted in other countries, which highlighted that nheABC and entFM were carried by emetic toxin-producing strains [16,17,22,23], as well as those of previous Korean studies [23,27,28]. The cytK gene is frequently detected in isolates from patients with diarrheal-type food poisoning caused by B. cereus [26]. However, the occurrence of cytK was lower than that of other enterotoxin genes in our study.

Given the public health importance of acute diarrheal disease, surveillance is performed in many countries. Examples include the Foodborne Diseases Active Surveillance Network (FoodNet, www.cdc.gov/foodnet) in the United States [29]; OzFoodNet (www.ozfoodnet.gov.au) in Australia [30]; and FoodNet-Canada (www.phac-aspc.gc.ca/foodnetcanada) in Canada. These surve-

Table 2. Presence of enterotoxin genes in emetic and non-emetic Bacillus cereus

| Target gene | Total strains (n = 667) | Non-emetic strains (n = 532) | Emetic strains (n = 135) |
|-------------|------------------------|-----------------------------|-------------------------|
| entFM*      | 567 (85.0)             | 462 (86.8)                  | 105 (77.8)              |
| nheA*       | 524 (78.6)             | 444 (83.5)                  | 80 (59.3)               |
| cytK2*      | 297 (44.5)             | 273 (51.3)                  | 24 (17.8)               |
| hblC*       | 244 (36.6)             | 228 (42.9)                  | 16 (11.9)               |
| bceT*       | 198 (29.7)             | 181 (34.0)                  | 17 (12.6)               |
| cer*        | 135 (20.2)             | 135 (100)                   |                         |

Values are presented as number (%).
*p < 0.05 by chi-square test.
### Table 3. Enterotoxin gene profiles of non-emetic and emetic toxin strains

| Strains       | Group | entFM | nheA | cytK2 | hblC | bceT | cer | Strains, n (%) |
|---------------|-------|-------|------|-------|------|------|-----|----------------|
| Non-emetic strains (n = 532) |       |       |      |       |      |      |     |                |
| A             | +     | +     | +    | +     | +    | +    | –   | 77 (14.5)      |
| B             | +     | +     | +    | +     | –    | –    | –   | 71 (13.3)      |
| C             | +     | +     | +    | –     | –    | –    | –   | 35 (6.6)       |
| D             | +     | +     | +    | –     | +    | –    | –   | 33 (6.2)       |
| E             | +     | +     | +    | –     | +    | +    | –   | 9 (1.7)        |
| F             | +     | +     | –    | –     | –    | +    | –   | 20 (3.8)       |
| G             | +     | +     | –    | +     | –    | –    | –   | 25 (4.7)       |
| H             | +     | +     | –    | –     | –    | –    | –   | 125 (23.5)     |
| I             | +     | –     | +    | +     | +    | –    | –   | 6 (1.1)        |
| J             | +     | –     | –    | +     | +    | –    | –   | 10 (1.9)       |
| K             | +     | –     | +    | +     | –    | –    | –   | 6 (1.1)        |
| L             | +     | –     | +    | –     | –    | –    | –   | 10 (1.9)       |
| M             | +     | –     | +    | –     | +    | +    | –   | 1 (0.2)        |
| N             | +     | –     | –    | –     | +    | –    | –   | 7 (1.3)        |
| O             | +     | –     | –    | +     | –    | –    | –   | 5 (0.9)        |
| P             | +     | –     | –    | –     | –    | –    | –   | 22 (4.1)       |
| Q             | –     | +     | +    | +     | +    | –    | –   | 3 (0.6)        |
| R             | –     | +     | +    | +     | –    | –    | –   | 6 (1.1)        |
| S             | –     | +     | +    | –     | –    | –    | –   | 9 (1.7)        |
| T             | –     | +     | +    | +     | –    | –    | –   | 2 (0.4)        |
| U             | –     | +     | –    | –     | +    | –    | –   | 1 (0.2)        |
| V             | –     | +     | –    | –     | +    | –    | –   | 3 (0.6)        |
| W             | –     | +     | –    | –     | –    | –    | –   | 25 (4.7)       |
| X             | –     | –     | +    | –     | +    | –    | –   | 2 (0.4)        |
| Y             | –     | –     | +    | +     | –    | –    | –   | 1 (0.2)        |
| a             | –     | –     | +    | –     | –    | –    | –   | 4 (0.8)        |
| b             | –     | –     | +    | –     | –    | +    | –   | 9 (1.7)        |
| c             | –     | –     | –    | +     | –    | –    | –   | 4 (0.8)        |
| d             | –     | –     | –    | –     | –    | –    | –   | 1 (0.2)        |
| Emetic strains (n = 135) |       |       |      |       |      |      |     |                |
| A             | +     | +     | +    | +     | +    | +    | +   | 3 (2.2)        |
| B             | +     | +     | +    | +     | –    | +    | –   | 4 (3.0)        |
| C             | +     | +     | +    | –     | +    | –    | +   | 4 (3.0)        |
| D             | +     | +     | +    | –     | –    | –    | +   | 1 (0.7)        |
| E             | +     | +     | +    | –     | –    | +    | –   | 1 (0.7)        |
| F             | +     | +     | +    | –     | –    | –    | +   | 56 (41.5)      |
| G             | +     | +     | +    | –     | +    | +    | –   | 4 (3.0)        |
| H             | +     | +     | +    | –     | +    | +    | –   | 4 (3.0)        |
| I             | +     | –     | +    | +     | +    | –    | +   | 2 (1.5)        |
| J             | +     | –     | +    | –     | –    | –    | +   | 1 (0.7)        |
| K             | +     | –     | +    | –     | +    | –    | +   | 1 (0.7)        |
| L             | +     | –     | –    | +     | +    | –    | +   | 1 (0.7)        |
| M             | +     | –     | –    | –     | +    | +    | –   | 1 (0.7)        |
| N             | +     | –     | –    | –     | +    | –    | +   | 22 (16.3)      |
| O             | –     | +     | +    | –     | –    | –    | +   | 1 (0.7)        |
| P             | –     | +     | –    | +     | –    | +    | –   | 1 (0.7)        |
| Q             | –     | +     | –    | –     | –    | +    | –   | 5 (3.7)        |
| R             | –     | –     | +    | –     | +    | +    | –   | 1 (0.7)        |
| S             | –     | –     | +    | –     | –    | +    | +   | 2 (1.5)        |
| T             | –     | –     | –    | –     | –    | –    | +   | 20 (14.8)      |
reus could be an important emerging public health threat. Thus, as a preventive measure, hygiene education on diarrheal diseases should be addressed.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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REFERENCES

1. Nyachuba DG. Foodborne illness: is it on the rise? Nutr Rev 2010;68:257-69. https://doi.org/10.1111/j.1753-4887.2010.00286.x
2. Stenfors Arnesen LP, Fagerlund A, Granum PE. From soil to gut: Bacillus cereus and its food poisoning toxins. FEMS Microbiol Rev 2008;32:579-606. https://doi.org/10.1111/j.1574-6976.2008.00112.x
3. Ngamwongsatit P, Buasri W, Pianariyanon P, et al. Broad distribution of enterotoxin genes (hblCDA, nheABC, cytK, and entFM) among Bacillus thuringiensis and bacillus cereus as shown by novel primers. Int J Food Microbiol 2008;121:352-6. https://doi.org/10.1016/j.ijfoodmicro.2007.11.013
4. Schoenl JL, Wong AC. Bacillus cereus food poisoning and its toxins. J Food Prot 2005;68:636-48. https://dx.doi.org/10.3148/0362-028X-68.3.636
5. Beecher DJ, Schoeni JL, Wong AC. Enterotoxic activity of hemolysin BL from Bacillus cereus. Infect Immun 1995;63:4423-8.
6. Granum PE, O'Sullivan K, Lund T. The sequence of the non-haemolytic enterotoxin operon from Bacillus cereus. FEMS Microbiol Lett 1999;177:225-9. https://doi.org/10.1111/j.1574-6968.1999.tb13736.x
7. Yang IC, Shih DY, Huang TP, et al. Establishment of a novel multiplex PCR assay and detection of toxigenic strains of the species in the Bacillus cereus group. J Food Prot 2005;68:2123-30. https://doi.org/10.4315/0362-028X-68.10.2123
8. Agata N, Ohta M, Mori M, et al. A novel dodecadepsipeptide, cereulide, is an emetic toxin of Bacillus cereus. FEMS Microbiol Lett 1995;129:17-20. https://doi.org/10.1111/j.1574-6968.1995.tb07550.x
9. Asano SI, Nakumizu Y, Bando H, et al. Cloning of novel enterotoxin genes from Bacillus cereus and Bacillus thuringiensis. Appl Environ Microbiol 1997;63:1054-7.
10. Lund T, Granum PE. Characterisation of a non-haemolytic enterotoxin complex from Bacillus cereus isolated after a foodborne outbreak. FEMS Microbiol Lett 1996;141:151-6. https://doi.org/10.1111/j.1574-6968.1996.tb08377.x
11. Fagerlund A, Ween O, Lund T, et al. Genetic and functional analysis of the cytK family of genes in Bacillus cereus. Microbiology 2004;150:2689-97. https://doi.org/10.1099/mic.0.26975-0
12. Guinebretiere MH, Fagerlund A, Granum PE, et al. Rapid discrimination of cytK-1 and cytK-2 genes in Bacillus cereus strains by a novel duplex PCR system. FEMS Microbiol Lett 2006;259:74-80. https://doi.org/10.1111/j.1574-6968.2006.00247.x
13. Ehling-Schulz M, Fricker M, Scherer S. Bacillus cereus, the causative agent of an emetic type of food-borne illness. Mol Nutr Food Res 2004;48:479-87. https://doi.org/10.1002/mnfr.200400055
14. Rajkovic A, Uyttendaele M, Vermeulen A, et al. Heat resistance of Bacillus cereus emetic toxin, cereulide. Lett Appl Microbiol 2008;46:536-41. https://doi.org/10.1111/j.1472-765x.2008.02350.x
15. Hoton FM, Andrup L, Swiecicka I, et al. The cereulide genetic determinants of emetic Bacillus cereus are plasmid-borne. Microbiology 2005;151:2121-4. https://doi.org/10.1099/mic.0.28069-0
16. Ehling-Schulz M, Svensson B, Guinebretiere MH, et al. Emetic toxin formation of Bacillus cereus is restricted to a single evolutionary lineage of closely related strains. Microbiology 2005;151:183-97. https://doi.org/10.1099/mic.0.27607-0
17. Ehling-Schulz M, Guinebretiere MH, Monthan A, et al. Toxin gene profiling of enterotoxigenic and emetic Bacillus cereus. FEMS Microbiol Lett 2006;260:232-40. https://doi.org/10.1111/j.1574-6968.2006.00320.x
18. Al-Abri SS, Al-Jardani AK, Al-Hosni MS, et al. A hospital acquired outbreak of Bacillus cereus gastroenteritis, Oman. J Infect Public Health 2011;4:180-6. https://doi.org/10.1016/j.jiph.2011.05.003
19. Choi KB, Lim HS, Lee K, et al. Epidemiological investigation for outbreak of food poisoning caused by Bacillus cereus among the workers at a local company in 2010. J Prev Med Public Health 2011;44:65-73. https://doi.org/10.3961/jpmph.2011.44.2.65
20. Fricker M, Messelhauser U, Busch U, et al. Diagnostic real-time PCR assays for the detection of emetic Bacillus cereus strains in foods and recent food-borne outbreaks. Appl Environ Microbiol 2007;73:1892-8. https://doi.org/10.1128/AEM.02219-06
21. Granum PE, Lund T. Bacillus cereus and its food poisoning toxins. FEMS Microbiol Lett 1997;157:223-8. https://doi.org/10.1111/j.1574-6968.1997.tb12776.x
22. Kim JH, Lim EG, Jang HC, et al. A case of emetic toxin producing Bacillus cereus. J Korean Soc Appl Biol Chem 2008;51:263-6. https://dx.doi.org/10.4315/0362-028X-68.10.2123
23. Kim JB, Kim JM, Kim CH, et al. Emetic toxin producing Bacillus cereus Korean isolates contain genes encoding diarrheal-related enterotoxins. Int J Food Microbiol 2008;121:48-52. https://doi.org/10.1016/j.ijfoodmicro.2007.11.013
24. Seong SJ, Lim JS, Lee KG, et al. Toxin gene profiling of Bacillus cereus Korean isolates by PCR. J Korean Soc Appl Biol Chem 2008;51:263-6. https://doi.org/10.3839/jksabc.2008.046

https://doi.org/10.24171/j.phrp.2017.8.1.10
25. Lund T, De Buyser ML, Granum PE. A new cytotoxicin from Bacillus cereus that may cause necrotic enteritis. Mol Microbiol 2000;38:254-61. https://doi.org/10.1046/j.1365-2958.2000.02147.x
26. Guinebretière MH, Broussolle V, Nguyen-The C. Enterotoxigenic profiles of food-poisoning and food-borne Bacillus cereus strains. J Clin Microbiol 2002;40:3053-6. https://doi.org/10.1128/JCM.40.8.3053-3056.2002
27. Chon JW, Kim JH, Lee SJ, et al. Toxin profile, antibiotic resistance, and phenotypic and molecular characterization of Bacillus cereus in Sunsik. Food Microbiol 2012;32:217-22. https://doi.org/10.1016/j.fm.2012.06.003
28. Chon JW, Kim JH, Lee SJ, et al. Prevalence, phenotypic traits and molecular characterization of emetic toxin producing Bacillus cereus strains isolated from human stools in Korea. J Appl Microbiol 2012;112:1042-9. https://doi.org/10.1111/j.1365-2672.2012.05277.x
29. Kendall ME, Crim S, Fullerton K, et al. Travel-associated enteric infections diagnosed after return to the United States. Foodborne Diseases Active Surveillance Network (FoodNet), 2004-2009. Clin Infect Dis 2012;54 Suppl 5:S480-7. https://doi.org/10.1093/cid/cis052
30. Kirk MD, McKay I, Hall GV, et al. Food safety: foodborne disease in Australia: the OzFoodNet experience. Clin Infect Dis 2008;47:392-400. https://doi.org/10.1086/589861