C. difficile is the main causative agent of nosocomial diarrhea. Besides hypervirulent PCR ribotypes (RTs), other epidemic RTs are currently a cause of concern (1, 2). In particular, RT018 has been detected in Italy and, more recently, in South Korea and Japan as a main cause of infections and outbreaks (3–5). RT018 isolates show resistance to several antibiotics and a transmission index 10-fold higher compared to that of RT078 strains (3, 6). Significant risk factors for complicated infections by RT018 are age ≥65 years, pulmonary comorbidity, and use of fluoroquinolones (2). For these reasons, the genome of the Italian strain IT1118, isolated during an outbreak, was analyzed to investigate features that may affect virulence.

Genomic DNA was sequenced using the HiSeq 2000 platform (GATC, Konstanz, Germany) in 50-bp single-read mode. A total of 18,123,358 reads were assembled into 256 contigs (>200 bp) using the Velvet assembler (7), with a total size of 4,238,925 bp and providing 218X coverage. The average contig length was 16,558 bp, with the largest contig being 178,925 bp. Gene prediction was performed using Glimmer version 3 (8), and contigs were mapped against the reference strain C. difficile 630 (RT012) (GenBank no. AM180355) using Geneious (9).

Mutations C245T in gyrA and G1514A in rpoB, found in the majority of European C. difficile isolates resistant to fluoroquinolones and rifampins (10–12), respectively, were detected in IT1118. Although IT1118 showed resistance to both erythromycin and clindamycin, neither resistance determinants nor mutations in the ribosomal proteins genes were observed.

The temporal activation of the sigK gene, involved in C. difficile sporulation, is regulated by the excision of a sigK intervening (skin) element (13). Results for the skin element of IT1118 (57 kb) were very different from those of 630 or M120 (RT078), but it had 99.9% sequence identity to that of strain BI-9 (GenBank no. FN688944), which belongs to the long-term problematic epidemic RT001. Diversity in the skin elements could differently affect strain sporulation and consequent transmissibility, although this hypothesis needs to be confirmed.

The surface layer (SL) of C. difficile is involved in pathogen-host interactions. Twelve different SL cassettes (containing the slpA, cwp66, and secA genes) have been identified (14). The 9.7-kb SL cassette of IT1118 showed 99.9% identity with the cassette 6 (14). Competitive assays in vivo suggest that strains with cassette 6 have an advantage in intestinal colonization compared to strains with other SL cassettes (15).

No significant differences in the pathogenicity locus, containing the genes encoding for the toxins A and B and their regulators, were observed between IT1118 and 630 (99.9% of identity). The sequence of the locus for the binary toxin, an additional toxin found in several RTs, was incomplete in both IT1118 and 630.

The genetic characteristics observed in IT1118 show this strain’s high capability to survive and propagate, giving a first explanation for the successful spreading of this C. difficile type.

**Nucleotide sequence accession numbers.** This genome sequence has been deposited in DDBJ/EMBL/GenBank under the accession number FAXM00000000. The version described in this paper is the first version, FAXM00100000.

**FUNDING INFORMATION**
This work, including the efforts of François Wasels, Fabrizio Barbanti, and Patrizia Spigaglia, was funded by EC | Seventh Framework Programme (FP7) (237942).

**REFERENCES**
1. Freeman J, Vernon J, Morris K, Nicholson S, Todhunter S, Longshaw C, Wilcox MH, Pan-European Longitudinal Surveillance of Antibiotic Resistance among Prevalent Clostridium difficile Ribotypes Study Group. 2015. Pan-European longitudinal surveillance of antibiotic resistance among prevalent Clostridium difficile ribotypes Study Group. 2015. Pan-European longitudinal surveillance of antibiotic resistance among prevalent Clostridium difficile ribotypes. Clin Microbiol Infect 21: e9.http://dx.doi.org/10.1016/j.cmi.2014.09.017.
2. Bauer MP, Notermans DW, van Benthem BHB, Brazier JS, Wilcox MH, Rupnik M, Monnet DL, van Dissel JT, Kuipper EJ, ECDIS Study Group. 2011. Clostridium difficile infection in Europe: a hospital-based survey. Lancet 377:63–73. http://dx.doi.org/10.1016/S0140-6736(10)61266-4.
3. Spigaglia P, Barbanti F, Morandi M, Moro ML, Mastrantonio P. 2016. Diagnostic testing for Clostridium difficile in Italian microbio-
logical laboratories. Anaerobe 37:29–33. http://dx.doi.org/10.1016/j.anaerobe.2015.11.002.
4. Kim J, Kang JO, Pai H, Choi TY. 2012. Association between PCR ribotypes and antimicrobial susceptibility among Clostridium difficile isolates from healthcare-associated infections in South Korea. Int J Antimicrob Agents 40:24–29. http://dx.doi.org/10.1016/j.ijantimicag.2012.03.015.
5. Senoh M, Kato H, Fukuda T, Niikawa A, Hori Y, Hagiya H, Ito Y, Miki H, Abe Y, Furuta K, Takeuchi H, Tajima H, Tominaga H, Satomura H, Kato H, Morita S, Tanada A, Hara T, Kawada M, Sato Y, Takahashi M, Higuchi A, Nakajima T, Wakahatsu Y, Toyokawa M, Ueda A, Roberts P, Miyajima F, Shibayama K. 2015. Predominance of PCR-ribotypes, 018 (smz) and 369 (trf) of Clostridium difficile in Japan: a potential relationship with other global circulating strains? J Med Microbiol 64:1226–1236. http://dx.doi.org/10.1099/jmm.0.000149.
6. Baldan R, Trovato A, Bianchini V, Biancardi A, Cichero P, Mazzotti M, Nizzero P, Moro M, Ossi C, Scarpellini P, Crillo DM. 2015. Clostridium difficile PCR ribotype 018, a successful epidemic genotype. J Clin Microbiol 53:2575–2580. http://dx.doi.org/10.1128/JCM.00533-15.
7. Zerbino DR, Birney E. 2008. Velvet: algorithms for de novo short read assembly using de Bruijn graphs. Genome Res 18:821–829. http://dx.doi.org/10.1101/gr.074492.107.
8. Delcher AL, Bratke KA, Powers EC, Salzberg SL. 2007. Identifying bacterial genes and endosymbiont DNA with Glimmer. Bioinformatics 23:673–679. http://dx.doi.org/10.1093/bioinformatics/btn009.
9. Kearse M, Moir R, Wilson A, Stones-Havas S, Cheung M, Sturrock S, Buxton S, Cooper A, Markowitz S, Duran C, Thierer T, Ashton B, Meintjes P, Drummond A. 2012. Geneious basic: an integrated and extendable desktop software platform for the organization and analysis of sequence data. Bioinformatics 28:1647–1649. http://dx.doi.org/10.1093/bioinformatics/bts199.
10. Spigaglia P, Barbanti F, Mastrantonio P, Brazier JS, Barbut F, Delmé M, Kuiper E, Poxtom IR, European Study Group on Clostridium difficile (ESGCD). 2008. Fluoroquinolone resistance in Clostridium difficile isolates from a prospective study of C. difficile infections in Europe. J Med Microbiol 57:784–789.
11. Huang H, Weintraub A, Fang H, Nord CE. 2009. Antimicrobial resistance in Clostridium difficile. Int J Antimicrob Agents 34:516–522. http://dx.doi.org/10.1016/j.ijantimicag.2009.09.012.
12. Pecavar V, Blaschitz M, Hufnagl P, Zeininger J, Fiedler A, Allerberger F, Maass M, Indra A. 2012. High-resolution melting analysis of the single nucleotide polymorphism hot-spot region in the rpoB gene as an indicator of reduced susceptibility to rifaximin in Clostridium difficile. J Med Microbiol 61:780–785. http://dx.doi.org/10.1099/jmm.0.041087-0.
13. Saujet L, Pereira FC, Henrques AO, Martin-Verstraete I. 2014. The regulatory network controlling spore formation in Clostridium difficile. FEMS Microbiol Lett 358:1–10. http://dx.doi.org/10.1111/1574-6968.12540.
14. Dingle KE, Didelot X, Ansari MA, Eyre DW, Vaughan A, Griffiths D, Ip CLC, Batty EM, Golubchik T, Bowden R, Jolley KA, Hood DW, Fawley WN, Walker AS, Peto TE, Wilcox MH, Crook DW. 2013. Recombinational switching of the Clostridium difficile S-layer and a novel glycosylation gene cluster revealed by large-scale whole-genome sequencing. J Infect Dis 207:675–686. http://dx.doi.org/10.1093/infdis/jis734.
15. Spigaglia P, Barketi-Klai A, Collignon A, Mastrantonio P, Barbanti F, Rupnik M, Janezic S, Kansau J. 2013. Surface-layer (S-layer) of human and animal Clostridium difficile strains and their behaviour in adherence to epithelial cells and intestinal colonization. J Med Microbiol 62:1386–1393. http://dx.doi.org/10.1099/jmm.0.056556-0.