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► To cite this version:

S. Sardi, P. Garcia, C. Zandotti, A. Chanteloup, E. Baptiste, et al.. First genome sequence of *Cellulomonas hominis* isolated from cerebrospinal fluid in the context of sudden infant death syndrome. *New Microbes and New Infections*, 2020, 33, pp.100623. 10.1016/j.nmni.2019.100623 . hal-02446274

HAL Id: hal-02446274

<https://amu.hal.science/hal-02446274v1>

Submitted on 16 Sep 2024

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First genome sequence of *Cellulomonas hominis* isolated from cerebrospinal fluid in the context of sudden infant death syndrome

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Keywords: *Cellulomonas*, cerebrospinal fluid, co-infection, genome sequencing, sudden infant death syndrome

Original Submission: 7 June 2019; **Revised Submission:** 21 October 2019; **Accepted:** 30 October 2019

Article published online: 29 November 2019

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A 2-month-old infant was found dead in Marseille, France, with hypotonic, pale skin and in respiratory arrest. No vaccination had been carried out, and the baby was breast-fed. Investigations were performed to elucidate the reasons for the sudden death. An autopsy was performed and acute pulmonary lesions were observed with emphysema, indicating mechanical asphyxia syndrome and an absence of malformation elements or underlying visceral pathologies involved in the mechanism of death. Toxicology analyses were negative. Based on the autopsy and on the pathologist analyses, it was concluded that the infant died unexpectedly from a sleeping (ventral) accident in a context of pharyngeal infection.

At the same time, investigating the laboratory values, we found the following: hyperleukocytosis in the order of 36 G/L (reference range: 6–18 G/L), hyper-lymphocytosis, predominantly B lymphocytes, with 32 G/L (reference range: 2–11 G/L), absence of biological inflammatory syndrome and absence of abnormality of haemoglobin. In parallel, molecular detection of *Staphylococcus aureus* and a rhinovirus were found positive in the nasopharynx. In the cerebrospinal fluid (CSF), there were 135 mononuclear cells and 20 red blood cells, and its appearance was limpid. The CSF was inoculated on PVX polyvitex chocolate agar (nutrient base enriched in factor X (hemin) and V (NAD)), COS Columbia agar + 5% sheep blood (COS) under CO₂ atmosphere, COS in

anaerobic conditions and were co-cultured on HEL and CE cells. All returned negative, except those on the COS agar plate after 48 hours of incubation at 37°C. The identification of this bacterium by 16S rRNA sequencing revealed a close similarity with *Cellulomonas hominis* strain Marseille-P2416 (LT223648.1) with 98.92% similar identity (Fig. 1). This last strain was isolated from human stool. However, its genome is not available for a genomic comparison. Genome sequencing was performed by paired-end strategy with Illumina Mi-seq technology. Genome assembly was carried out with the hybrid spades program [1] using only the paired-end strategy and standard parameters. A draft genome of 4 071 551 bp was obtained with 105 scaffolds and with a GC content close to 75%. A total of 3740 genes were predicted, plus 50 tRNA, and we detected one gene of 16S rRNA, one for 23S rRNA and one for 5SrRNA. Annotation was performed using PROKKA software [2] and brief analyses of BLAST protein confirmed that the isolated strain was a *Cellulomonas*; we named the isolated strain IHU. This represents the first draft genome available for *C. hominis* in a public database (embl website under number CABEGA010000001–CABEGA010000105). The literature contains few cases with CSF isolation of *Cellulomonas*, especially *C. hominis* [3]. Members of this species are Gram-positive, pleomorphic motile bacilli, lacking spores and capsules. Colonies are small and pale yellow [4]. No data were found concerning ages or clinical context of their discoveries in the CSF. Regarding sudden infant death syndrome, it is now clear that it is frequently associated (about 50%) with mild respiratory infections and increased interleukin-6 expression in the CSF [5]. Herein, the infant presents a complex clinical presentation with the presence of rhinovirus

Tree scale: 0.01

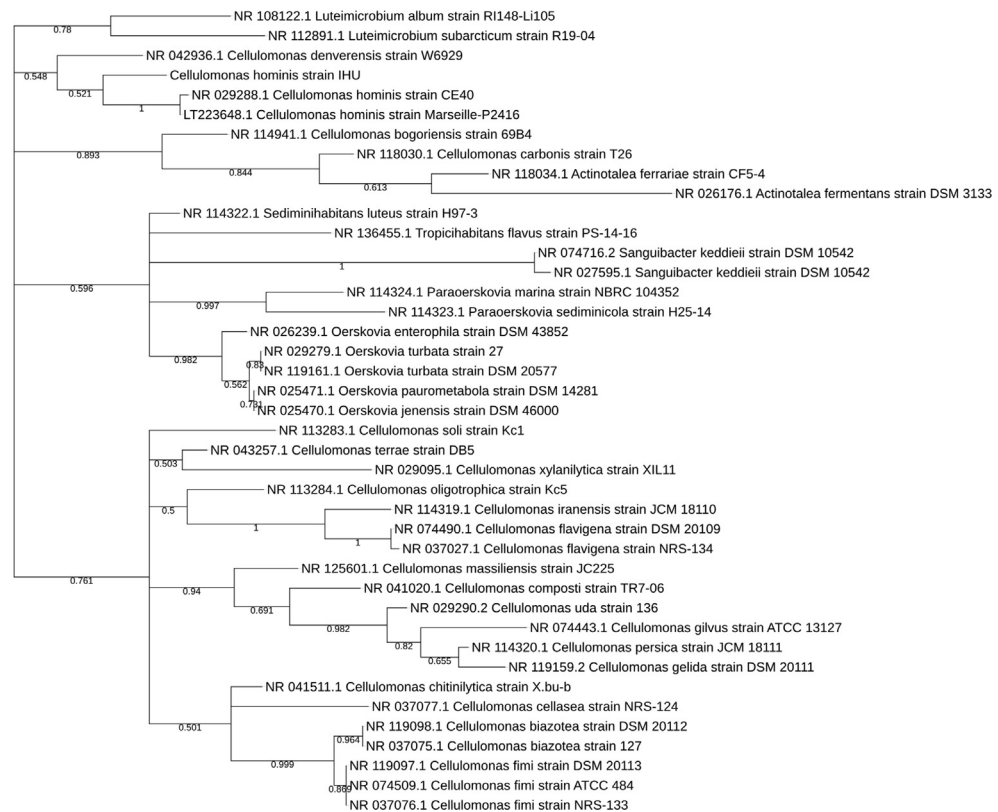


FIG. 1. Phylogenetic position of *Cellulomonas hominis* strain IHU. Maximum-likelihood tree based on the 16S rRNA genes of 41 bacteria sequences. Bootstrap values are indicated in the tree. MEGA 6 package was used to perform the entire analysis: alignment with MUSCLE and tree with Juke–Cantor model using 1000 replicates and the visualization was done using the iTOL program online.

in the nasopharynx compatible with the most common history of SIDS, but also with CSF signs of infection, suggesting that *C. hominis* could have played a role in death in this case.

‘Investissements d’avenir (Investments for the Future)’ programme with the reference ANR-10-IAHU-03 (Méditerranée Infection) and by Région Provence Alpes Côte d’Azur and European funding FEDER PRIMI.

Disclosures

All authors have nothing to disclose.

Conflict of interest

The authors have nothing to disclose.

Acknowledgments

This work was supported by a grant from the French State managed by the National Research Agency under the

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