



HAL
open science

Clostridium difficile forms variable biofilms on abiotic surface

Véronique Pantaleon, Marc Monot, Catherine Eckert, Sandra Hoys, Anne Collignon, Claire Janoir, Thomas Candela

► **To cite this version:**

Véronique Pantaleon, Marc Monot, Catherine Eckert, Sandra Hoys, Anne Collignon, et al.. Clostridium difficile forms variable biofilms on abiotic surface. Anaerobe, 2018, 53, pp.34-37. 10.1016/j.anaerobe.2018.05.006 . pasteur-02549240

HAL Id: pasteur-02549240

<https://pasteur.hal.science/pasteur-02549240v1>

Submitted on 13 Dec 2023

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial 4.0 International License

1 *Clostridium difficile* forms variable biofilms on abiotic surface

2

3 Pantaléon V.¹, Monot M.^{2,3,4}, Eckert C.^{5,6}, Hoys S.¹, Collignon A.¹, Janoir C.¹, Candela T.^{1*}

4 ¹EA4043 Unité Bactéries Pathogènes et Santé (UBaPS), Université Paris-Sud, Université Paris-
5 Saclay, 92290 Châtenay-Malabry, France.

6 ²Département de Microbiologie, Institut Pasteur, Laboratoire Pathogenèse des bactéries
7 anaérobies, Paris, France

8 ³Département de Microbiologie et d'infectiologie, Faculté de Médecine et des Sciences de la
9 Santé, Université de Sherbrooke, Sherbrooke, QC, Canada.

10 ⁴Université Paris Diderot, Sorbonne Paris Cité, Paris, France.

11 ⁵National Reference Laboratory for *C. difficile*, Assistance Publique-Hôpitaux de Paris,
12 Hôpital Saint-Antoine, Paris, France.

13 ⁶Sorbonne Université, Centre d'immunologie et des maladies infectieuses-Paris, Cimi-Paris,
14 Département de Bactériologie, AP-HP, Hôpitaux Universitaires de l'Est Parisien, F-75012
15 Paris, France

16 *Corresponding author

17

18 Keywords : *Clostridium difficile*; biofilm; motility

19 Abstract

20 *Clostridium difficile* can form biofilms. Thirty-seven strains were characterized for their
21 ability to form a biofilm, adhesion on an inert surface and hydrophobicity. No correlation
22 between the ability to form a biofilm and the strain virulence was highlighted. However, non-
23 motile strains were not able to form a high biofilm.

24

25 *Clostridium difficile*, an anaerobic Gram-positive spore-forming bacterium, is responsible
26 for a wide spectrum of infections ranging from mild/severe diarrhea to life-threatening
27 pseudomembranous colitis. The evolution of *C. difficile* infection (CDI) depends on both strain
28 virulence and host response. *C. difficile* clinical relevance has significantly increased since 2003
29 with the emergence of hypervirulent PCR-ribotype 027 strains that have been involved in
30 outbreaks, and that have been associated with severe disease in both North America and Europe.
31 *C. difficile* causes at least 450,000 infections leading to 29,000 deaths per year in the United
32 States of America (USA) [1]. The US Centers for Disease Control has designated this pathogen
33 as the pre-eminent of three “Urgent Threats” to US healthcare. The increasing incidence of CDI
34 and the high rate of recurrent disease have a major impact on healthcare costs. Because patient
35 contamination occurs from *C. difficile* spores present in the environment, increased spore
36 dissemination during outbreaks significantly complicates approaches to control infection [2, 3].
37 Antibiotic exposure leading to dysbiosis of gut microbiota is with advanced age and
38 hospitalization a major risk factor associated with CDI [4]. The major challenge is the
39 recurrence of CDI. Up to 35% of patients may undergo a recurrence after the first CDI episode
40 [5]. This rate increases after first recurrence. In case of relapse with the same *C. difficile* strain,
41 reinfection or persistence of *C. difficile* in the intestine may occur. Persistence may be the result
42 of germination/sporulation cycles or biofilm formation [6, 7]. Recently, *C. difficile* was
43 proposed to form a biofilm structure in the caecum and colon in a mono-associated mouse
44 model [7]. In addition, *C. difficile* is also able to reside in multicellular communities in mice,
45 but also in human periodontal-disease-associated biofilm [6, 8]. Similarly, in vitro *C. difficile*
46 is able to form a thick biofilm in the presence of *Fingoldia magna* [9]. In vitro, biofilm may
47 protect *C. difficile* from different antibiotics, such as vancomycin and metronidazole [10, 11].
48 In contrary, some antibiotics, such as surotomycin and fidaxomicin, were shown to be able to
49 disrupt the biofilm structure [12]. Biofilm matrix is composed of eDNA, polysaccharides and

50 proteins. Toxins were found in the biofilm matrix and may facilitate infection [10, 13]. *C.*
51 *difficile* has also been shown to sporulate in biofilm [13]. Moreover, spores were reported to be
52 found preferentially within biofilm structures in a chemostat gut model [14]. *C. difficile* life in
53 biofilm may therefore be crucial for its in vivo persistence. However, in vivo and in vitro
54 biofilm studies of *C. difficile* are still poorly explored with respect to other bacterial species (for
55 reviews [15, 16]) [15]. In vitro, only 7 different strains of *C. difficile* were described (mostly
56 the 630 and the R20291) to form a qualitatively weak biofilm [13, 17-22]. Here, we tested 37
57 strains from different origins to assess their ability to form in vitro biofilms to detect any
58 correlation with strain characteristics.

59 All the strains used in this study were isolated from infected patients except 2 that came
60 from poultry or rabbit (Table 1). They were all tested for their capacity to form a biofilm (72h
61 at 37°C, in 24-well polystyrene plates) on abiotic surface as described in Pantaleon *et al.* [19]
62 and quantified by crystal violet staining (Figure 1). Data are representative of three independent
63 experiments each performed in triplicate. According to their ability to form a biofilm, strains
64 were classed into three groups that are significantly different (Bartlett test of homogeneity of
65 variances, <http://marne.u707.jussieu.fr/biostatgv/>): the low-biofilm formers ($OD_{570} < 1.1$), the
66 moderate-biofilm formers ($1.1 < OD_{570} < 3.5$) and the high-biofilm formers ($OD_{570} > 3.5$). The
67 strain 94-1155 formed the highest biofilm with a mean $OD_{570} = 10.759$ and the strain T5 formed
68 the smallest biofilm with a mean $OD_{570} = 0.218$. 48.5% of the strains form a low-biofilm, 21.5%
69 a medium-biofilm and 30% a high-biofilm. Three strains were isolated from patient with relapse
70 (95-1078, 96-631 and 94-1155), and we note that they belong to the medium and high-biofilm
71 formers. These results suggest that biofilm is highly variable among *C. difficile* strains.

72 To investigate whether the ability to form a biofilm may be due to their PCR-ribotype
73 and sequence type (ST), they were determined for each strain (Table 1). The PCR-ribotype was
74 performed according to the protocol of Bidet *et al* [23] and the multi-locus sequence type
75 (MLST) was performed as previously described [24]. The toxinotype when known was also
76 reported in Table 1. Some strains were closely related, according to their ST, ribotype and
77 toxinotype. However, they have differences in their sequences and are therefore different:
78 R20291/CD196 [25], E16/T61, E15/T11, T15/E25 [26]. No statistical test could be performed
79 due to the low number of strains available for each typing method (ribotype and ST). However,
80 our results suggest that biofilm is variable among *C. difficile* strains, e.g. ribotypes 005 or 020
81 formed medium and high biofilms; strains belonging to ST19 formed low, medium and high
82 biofilms. Similarly, our results suggest that there is no correlation between the *C. difficile* strain
83 clinical symptoms during infection and the ability to form a biofilm.

84 The initial adhesion on an inert support may be involved in biofilm formation [27].
85 Initial adhesion of the 37 strains was therefore assessed on an abiotic surface (24-well
86 polystyrene plates, 1h at 37°C) as described in Pantaleon *et al.* (Figure 2) [19]. Data are
87 representative of three independent experiments each performed in triplicate. The mean of
88 adhesion for all the tested strains was 2.56% with a maximum of 8.89% (T6 strain) and a
89 minimum of 0.41% (T42 strain). No correlation was observed between adhesion of the strains
90 and their ability to form a biofilm (Pearson method: correlation coefficient = 0.01, pvalue =
91 0.96).

92 The surface hydrophobicity properties of bacteria are also usually involved in the
93 adhesion and the biofilm formation [28]. The properties of surface hydrophobicity of the 37
94 *C. difficile* strains were studied using the hexadecane method [19] (Table 1). The mean of
95 hydrophobicity was 18.74% with a maximum of 71.95% (VPI11186 strain) and a minimum of
96 6.84% (CD1 strain). The three strains forming the smallest biofilms (T5, 4485 and T20) and
97 the three strains forming the highest biofilms (T14, 3751 and 94-1155) had a similar surface
98 hydrophobicity mean. This result suggests that the surface hydrophobicity of *C. difficile* do not
99 correlate (Pearson method: correlation coefficient = 0.07, pvalue = 0.67) with their ability to
100 form biofilms.

101 The motility of bacteria has also been described as a feature of biofilm formation [29].
102 The motility of all strains was analysed using BHI agar 0.3% in test tubes. The agar medium
103 was inoculated with one colony and placed in anaerobic chamber at 37°C for 24 to 48 hours.
104 As expected, all the strains were motile except 6 (E1, T5, T20, IT1106, CD15 and 4485) which
105 belong to the 078, 079 and 126 ribotypes. These six strains belong to the ST49 (Table 1). The
106 strains E1, T5 and T20 have been sequenced [26] and they all possess the *fliC* and *fliD* genes,
107 but not the F3 locus, which does code for the motor and basal body of the flagellum. The six
108 non-motile strains belong to the low-biofilm former group. This result suggests that motility or

109 the flagellum presence is involved in biofilm formation after 3 days (Pearson method:
110 correlation coefficient = 0.33, pvalue = 0.05). Other studies, using mutant strains affecting
111 directly or indirectly either the flagellum or the motility have shown that biofilm was increased
112 in the mutant in comparison with the parental strains [21, 22, 30]. This apparent discrepancy
113 may be due to a difference in the global regulation due to the absence of the F3 region, and
114 probably *fliC* expression which may also impact the regulatory mechanisms and formation of
115 biofilm. Indeed, FliC has been proposed to be responsible for pleiotropic gene regulation
116 [31].

117 In conclusion, our study presents the ability to form a biofilm of 37 different strains.
118 The importance of motility in *C. difficile* biofilm was highlighted. However, no correlation
119 between their ability to form a biofilm with neither the origin nor the clinical symptoms of *C.*
120 *difficile* strains was observed.

121

122 Acknowledgments

123 S. Lambert is acknowledged for her technical help.

124

125 References

- 126 [1] F.C. Lessa, Y. Mu, W.M. Bamberg, Z.G. Beldavs, G.K. Dumyati, J.R. Dunn, et al. Burden
127 of *Clostridium difficile* infection in the United States. The New England journal of medicine
128 372 (2015) 825-34.
- 129 [2] M.J. Alam, A. Anu, S.T. Walk, K.W. Garey. Investigation of potentially pathogenic
130 *Clostridium difficile* contamination in household environs. *Anaerobe* 27 (2014) 31-3.
- 131 [3] M.P. Bauer, E.J. Kuijper. Potential sources of *Clostridium difficile* in human infection.
132 *Infectious disease clinics of North America* 29 (2015) 29-35.
- 133 [4] A.M. Seekatz, V.B. Young. *Clostridium difficile* and the microbiota. *The Journal of*
134 *clinical investigation* 124 (2014) 4182-9.
- 135 [5] J.W. Marsh, R. Arora, J.L. Schlackman, K.A. Shutt, S.R. Curry, L.H. Harrison.
136 Association of relapse of *Clostridium difficile* disease with BI/NAP1/027. *Journal of clinical*
137 *microbiology* 50 (2012) 4078-82.
- 138 [6] E.G. Semenyuk, V.A. Poroyko, P.F. Johnston, S.E. Jones, K.L. Knight, D.N. Gerding, et
139 al. Analysis of Bacterial Communities during *Clostridium difficile* Infection in the Mouse.
140 *Infection and immunity* 83 (2015) 4383-91.
- 141 [7] A.P. Soavelomandroso, F. Gaudin, S. Hoys, V. Nicolas, G. Vedantam, C. Janoir, et al.
142 Biofilm Structures in a Mono-Associated Mouse Model of *Clostridium difficile* Infection.
143 *Frontiers in microbiology* 8 (2017) 2086.
- 144 [8] A.P. Vieira Colombo, C.B. Magalhaes, F.A. Hartenbach, R. Martins do Souto, C. Maciel
145 da Silva-Boghossian. Periodontal-disease-associated biofilm: A reservoir for pathogens of
146 medical importance. *Microbial pathogenesis* 94 (2016) 27-34.
- 147 [9] G. Donelli, C. Vuotto, R. Cardines, P. Mastrantonio. Biofilm-growing intestinal
148 anaerobic bacteria. *FEMS Immunol Med Microbiol* 65 (2012) 318-25.
- 149 [10] T. Dapa, M. Unnikrishnan. Biofilm formation by *Clostridium difficile*. *Gut microbes*
150 4 (2013) 397-402.
- 151 [11] C. Vuotto, I. Moura, F. Barbanti, G. Donelli, P. Spigaglia. Subinhibitory
152 concentrations of metronidazole increase biofilm formation in *Clostridium difficile* strains.
153 *Pathogens and disease* 74 (2016).
- 154 [12] G.A. James, L. Chesnel, L. Boegli, E. deLancey Pulcini, S. Fisher, P.S. Stewart. Analysis
155 of *Clostridium difficile* biofilms: imaging and antimicrobial treatment. *The Journal of*
156 *antimicrobial chemotherapy* 73 (2018) 102-8.
- 157 [13] E.G. Semenyuk, M.L. Laning, J. Foley, P.F. Johnston, K.L. Knight, D.N. Gerding, et al.
158 Spore formation and toxin production in *Clostridium difficile* biofilms. *PLoS One* 9 (2014)
159 e87757.
- 160 [14] G.S. Crowther, C.H. Chilton, S.L. Todhunter, S. Nicholson, J. Freeman, S.D. Baines, et
161 al. Development and validation of a chemostat gut model to study both planktonic and
162 biofilm modes of growth of *Clostridium difficile* and human microbiota. *PLoS One* 9 (2014)
163 e88396.
- 164 [15] V. Pantaleon, S. Bouttier, A.P. Soavelomandroso, C. Janoir, T. Candela. Biofilms of
165 *Clostridium* species. *Anaerobe* 30 (2014) 193-8.
- 166 [16] C. Vuotto, G. Donelli, A. Buckley, C. Chilton. *Clostridium difficile* Biofilm. *Advances*
167 *in experimental medicine and biology* 1050 (2018) 97-115.
- 168 [17] T. Dapa, R. Leuzzi, Y.K. Ng, S.T. Baban, R. Adamo, S.A. Kuehne, et al. Multiple factors
169 modulate biofilm formation by the anaerobic pathogen *Clostridium difficile*. *J Bacteriol*
170 195 (2013) 545-55.

- 171 [18] G.A. Maldarelli, K.H. Piepenbrink, A.J. Scott, J.A. Freiberg, Y. Song, Y. Achermann, et
172 al. Type IV pili promote early biofilm formation by *Clostridium difficile*. Pathogens and
173 disease 74 (2016).
- 174 [19] V. Pantaleon, A.P. Soavelomandroso, S. Bouttier, R. Briandet, B. Roxas, M. Chu, et al.
175 The *Clostridium difficile* Protease Cwp84 Modulates both Biofilm Formation and Cell-
176 Surface Properties. PLoS One 10 (2015) e0124971.
- 177 [20] E.B. Purcell, R.W. McKee, D.S. Courson, E.M. Garrett, S.M. McBride, R.E. Cheney, et
178 al. A Nutrient-Regulated Cyclic Diguanilate Phosphodiesterase Controls *Clostridium*
179 *difficile* Biofilm and Toxin Production during Stationary Phase. Infection and immunity 85
180 (2017).
- 181 [21] E. Valiente, L. Bouche, P. Hitchen, A. Faulds-Pain, M. Songane, L.F. Dawson, et al.
182 Role of Glycosyltransferases Modifying Type B Flagellin of Emerging Hypervirulent
183 *Clostridium difficile* Lineages and Their Impact on Motility and Biofilm Formation. The
184 Journal of biological chemistry 291 (2016) 25450-61.
- 185 [22] B.M. Walter, S.T. Cartman, N.P. Minton, M. Butala, M. Rupnik. The SOS Response
186 Master Regulator LexA Is Associated with Sporulation, Motility and Biofilm Formation in
187 *Clostridium difficile*. PLoS One 10 (2015) e0144763.
- 188 [23] P. Bidet, F. Barbut, V. Lalande, B. Burghoffer, J.C. Petit. Development of a new PCR-
189 ribotyping method for *Clostridium difficile* based on ribosomal RNA gene sequencing.
190 FEMS Microbiol Lett 175 (1999) 261-6.
- 191 [24] L. Lemee, A. Dhalluin, M. Pestel-Caron, J.F. Lemeland, J.L. Pons. Multilocus sequence
192 typing analysis of human and animal *Clostridium difficile* isolates of various toxigenic
193 types. Journal of clinical microbiology 42 (2004) 2609-17.
- 194 [25] R.A. Stabler, M. He, L. Dawson, M. Martin, E. Valiente, C. Corton, et al. Comparative
195 genome and phenotypic analysis of *Clostridium difficile* 027 strains provides insight into
196 the evolution of a hypervirulent bacterium. Genome biology 10 (2009) R102.
- 197 [26] H. Kurka, A. Ehrenreich, W. Ludwig, M. Monot, M. Rupnik, F. Barbut, et al. Sequence
198 similarity of *Clostridium difficile* strains by analysis of conserved genes and genome
199 content is reflected by their ribotype affiliation. PLoS One 9 (2014) e86535.
- 200 [27] V. Nguyen, E. Karunakaran, G. Collins, C.A. Biggs. Physicochemical analysis of initial
201 adhesion and biofilm formation of *Methanosarcina barkeri* on polymer support material.
202 Colloids and surfaces B, Biointerfaces 143 (2016) 518-25.
- 203 [28] A. Krasowska, K. Sigler. How microorganisms use hydrophobicity and what does
204 this mean for human needs? Frontiers in cellular and infection microbiology 4 (2014) 112.
- 205 [29] A. Houry, R. Briandet, S. Aymerich, M. Gohar. Involvement of motility and flagella
206 in *Bacillus cereus* biofilm formation. Microbiology 156 (2010) 1009-18.
- 207 [30] S. Jain, D. Smyth, B.M.G. O'Hagan, J.T. Heap, G. McMullan, N.P. Minton, et al.
208 Inactivation of the *dnaK* gene in *Clostridium difficile* 630 Deltaerm yields a temperature-
209 sensitive phenotype and increases biofilm-forming ability. Scientific reports 7 (2017)
210 17522.
- 211 [31] A. Barketi-Klai, M. Monot, S. Hoys, S. Lambert-Bordes, S.A. Kuehne, N. Minton, et al.
212 The flagellin FliC of *Clostridium difficile* is responsible for pleiotropic gene regulation
213 during in vivo infection. PLoS One 9 (2014) e96876.
- 214 [32] M. Sebahia, B.W. Wren, P. Mullany, N.F. Fairweather, N. Minton, R. Stabler, et al.
215 The multidrug-resistant human pathogen *Clostridium difficile* has a highly mobile, mosaic
216 genome. Nature genetics 38 (2006) 779-86.
- 217 [33] P. Spigaglia, A. Barketi-Klai, A. Collignon, P. Mastrantonio, F. Barbanti, M. Rupnik,
218 et al. Surface-layer (S-layer) of human and animal *Clostridium difficile* strains and their

219 behaviour in adherence to epithelial cells and intestinal colonization. J Med Microbiol 62
220 (2013) 1386-93.
221 [34] M. Rupnik, V. Avesani, M. Janc, C. von Eichel-Streiber, M. Delmee. A novel
222 toxinotyping scheme and correlation of toxinotypes with serogroups of *Clostridium*
223 *difficile* isolates. Journal of clinical microbiology 36 (1998) 2240-7.
224
225

Table 1

Strains	Origin (geography)	Origin (ecology)	Symptoms	Biofilm biomass (OD ₅₇₀)	Strain typing			Hydrophobicity	Motility	Références
					MLST	PCR-ribotype	Toxinotype			
E23	Europe	human	SCDI	0.359	36	001 ; 001/072*	0	12.30%	+	[26]
CD268	Italy	rabbit	/	0.648	33	001	ND	20.40%	+	Lab Collection
FM17	unknow	unknow	Unknown	0.314	19	002	ND	9.80%	+	Lab Collection
96-631	France	human	Relapse	3.509	21	003	ND	10.19%	+	Lab Collection
T15	France	human	Diarrhoea	1.15	19	005	0	15.51%	+	[26]
E25	France	human	Sepsis, Col	2.917	19	005	0	30.18%	+	[26]
94-1155	France	human	Relapse	10.759	19	005	ND	16.55%	+	Lab Collection
CD630	UK	human	PMC	1.039	27	012*	0*	18.50%	+	[32]
E14	Europe	human	SCDI	3.803	33	014	0	14.19%	+	[26]
P30	Italy	poultry	/	5.370	34	014/020*	0*	15.36%	+	[33]
4684/08	Italy	human	Unknown	5.599	33	014/020*	0*	15.90%	+	[33]
1064	Italy	human	Unknown	7.670	33	014/020*	0*	23.49%	+	[33]
CD49	France	human	Carriage	0.607	33	014/020/077	ND	22.58%	+	Lab Collection
E16	France	human	Perf, Peri	0.586	1	015	0	13.19%	+	[26]
T61	France	human	Diarrhoea	0.963	1	015	0	11.98%	+	[26]
E24	France	human (community)	SS	1.675	33	020	0	16.57%	+	[26]
T42	France	human	Diarrhoea	4.871	33	020	0	16.98%	+	Accession Number : PRJEB216

CD196	France	human	CDI, PMC	1.630	3	027*	III*	22.59%	+	[26]
R20291	UK	human	CDI	2.471	3	027*	III*	19.04%	+	[20]
VPI11186	USA	Unknown	Unknown	1.884	16	038*	ND	70.00%	+	Lab Collection
E15	France	human	MG, SS, Col	0.4	5	075	III	18.67%	+	[26]
T11	France	human	Diarrhoea	0.662	5	075	III	9.18%	+	[26]
T20	Ireland	human	CDI	0.312	49	078	V	20.16%	-	[26]
IT1106	Italy	human	Unknown	0.704	49	078*	V*	17.96%	-	[33]
4485	France	human	PMC	0.264	49	078/126	ND	14.54%	-	Lab Collection
CD15	France	human (HIV+)	Diarrhoea	0.561	49	078/126	ND	23.29%	-	Lab Collection
T5	Italy	human	CDI	0.218	49	079	V	22.66%	-	[26]
T6	Hungary	human	CDI,	4.255	33	095	0	11.61%	+	[26]
E12	UK	human	SCDI	0.502	26	106	0	19.87%	+	[26]
E1	Austria	human	SCDI	1.004	49	126	V	14.41%	-	[26]
T14	Ireland	human	CDI	7.747	26	156	0	32.51%	+	[26]
T10	Germany	human	CDI	3.115	36	165	0	11.13%	+	[26]
VPI10463	USA	human	PMC	1.047	11	ND	0*	11.04%	+	[34]
94-1458	France	human	Unknown	0.575	1	other	ND	19.86%	+	Lab Collection
95-1078	France	human	Relapse	2.517	~20	other	ND	18.50%	+	Lab Collection

Table 1: strains and characteristics

The upper part of the table reports the low-biofilm former, the middle part reports the medium-biofilm formers forming an intermediate biofilm and the lower part reports the high-biofilm formers. ND, not determined; CDI *Clostridium difficile* infection; SCDI Severe *Clostridium difficile* infection; Col, colectomy; HIV, human immunodeficiency virus; MG, Megacolon; Peri, Peritonitis; Perf, perforation; PMC, Pseudomembranous Colitis; SS, septic shock. * corresponds to the characteristics of the strains described in other studies (see references in the text). other: PCR-ribotypes that are not among the following ,001, 015, 017, 023, 056, 081, 087, 014/020/077, 003, 019, 046, 050, 117, 005, 018, 106, 131, 012, 029, 053, 075, 002, 070, 078/126

Figure legend

Figure 1: ability of the 37 strains to form a biofilm

Biomass of the 37 strains after 72h of incubation. Strains are divided (dashed lines) in three categories, low, medium and high-biofilm formers.

Figure 2: adhesion of the 37 strains on an inert surface

Percentage of initial adhesion of the 37 strains to a polystyrene inert surface. Error bars represent standard deviation. Strains are classed by low, medium and high-biofilm formers.