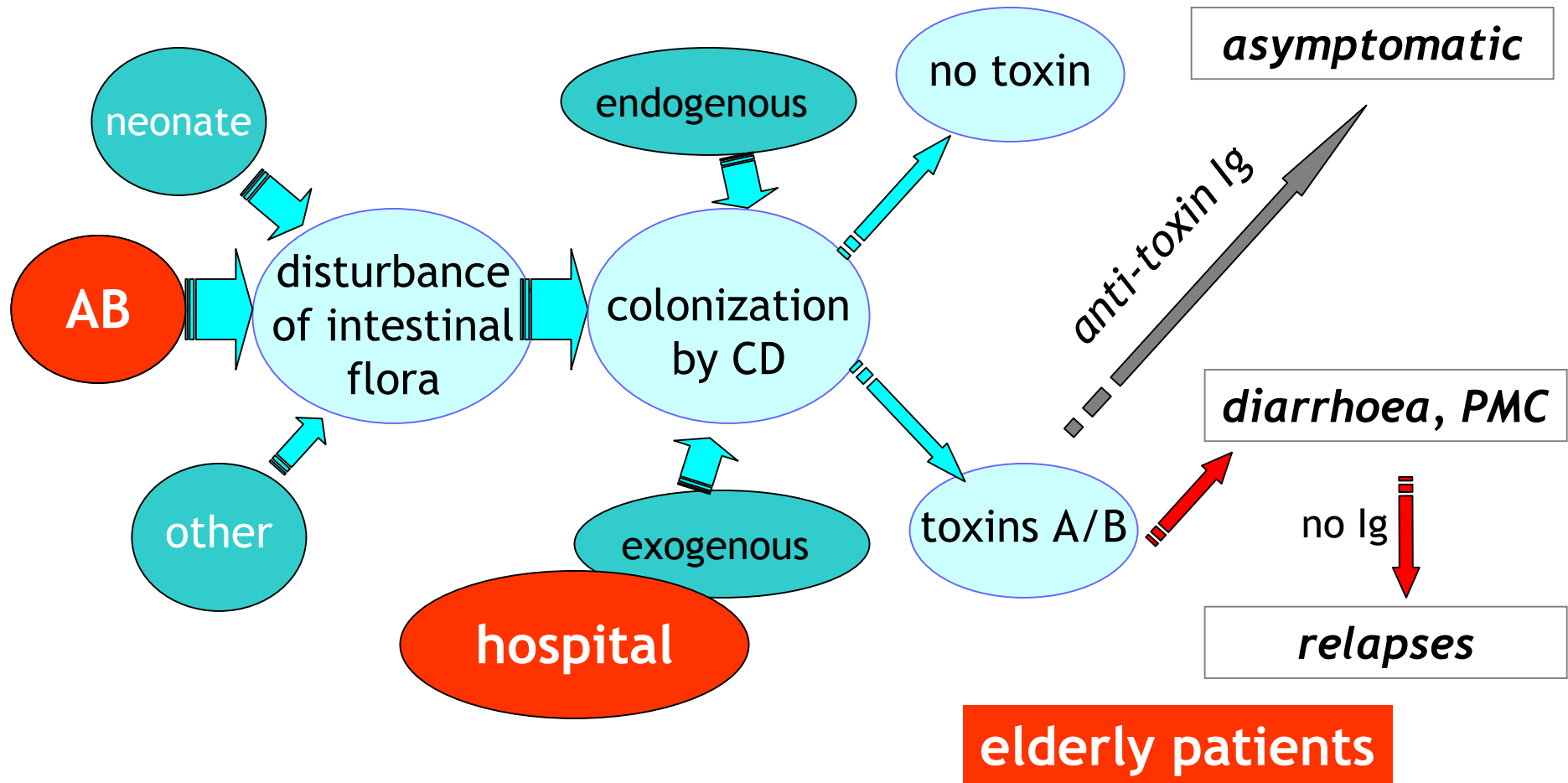


# Les souches hyper-virulentes de *Clostridium difficile*

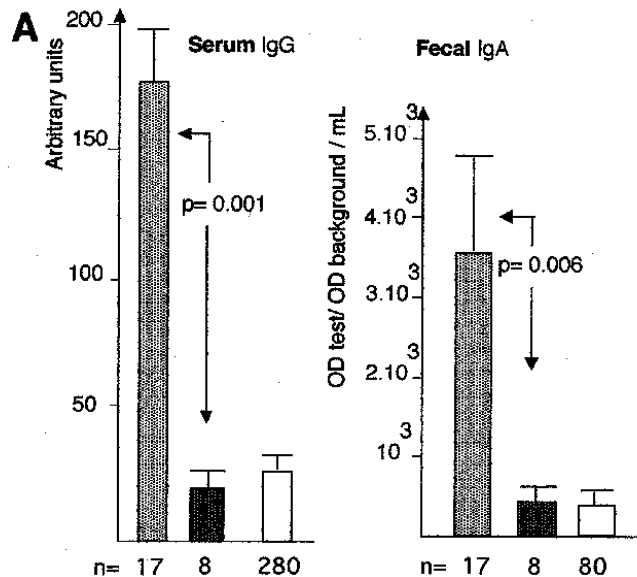
Michel Delmée






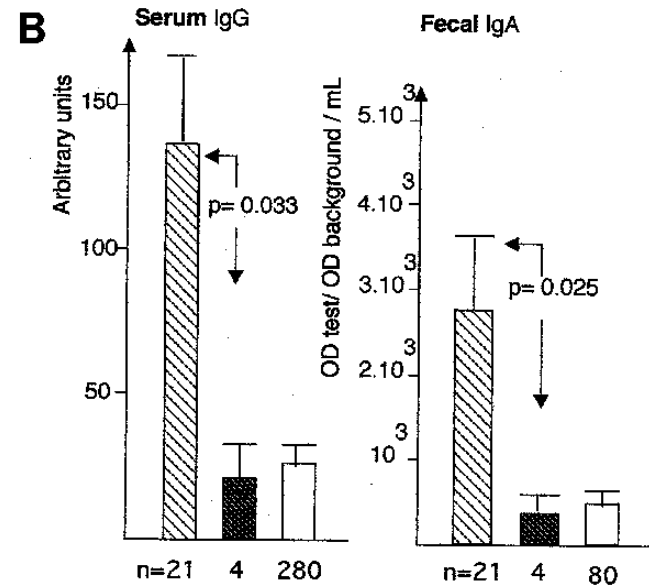
# CDAD : Physiopathology






# Immune response in CDAD



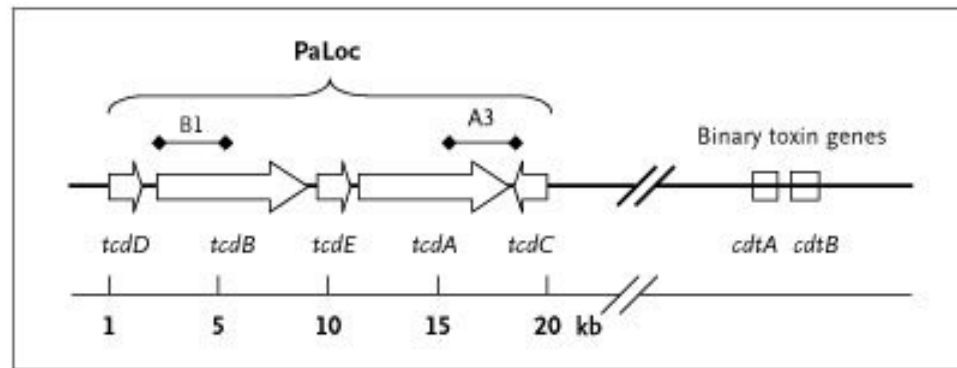
 diarrhea of less than 2 w.  
 diarrhea of more than 2 w  
 controls



 one episode of diarrhea  
 relapsing diarrhea  
 controls

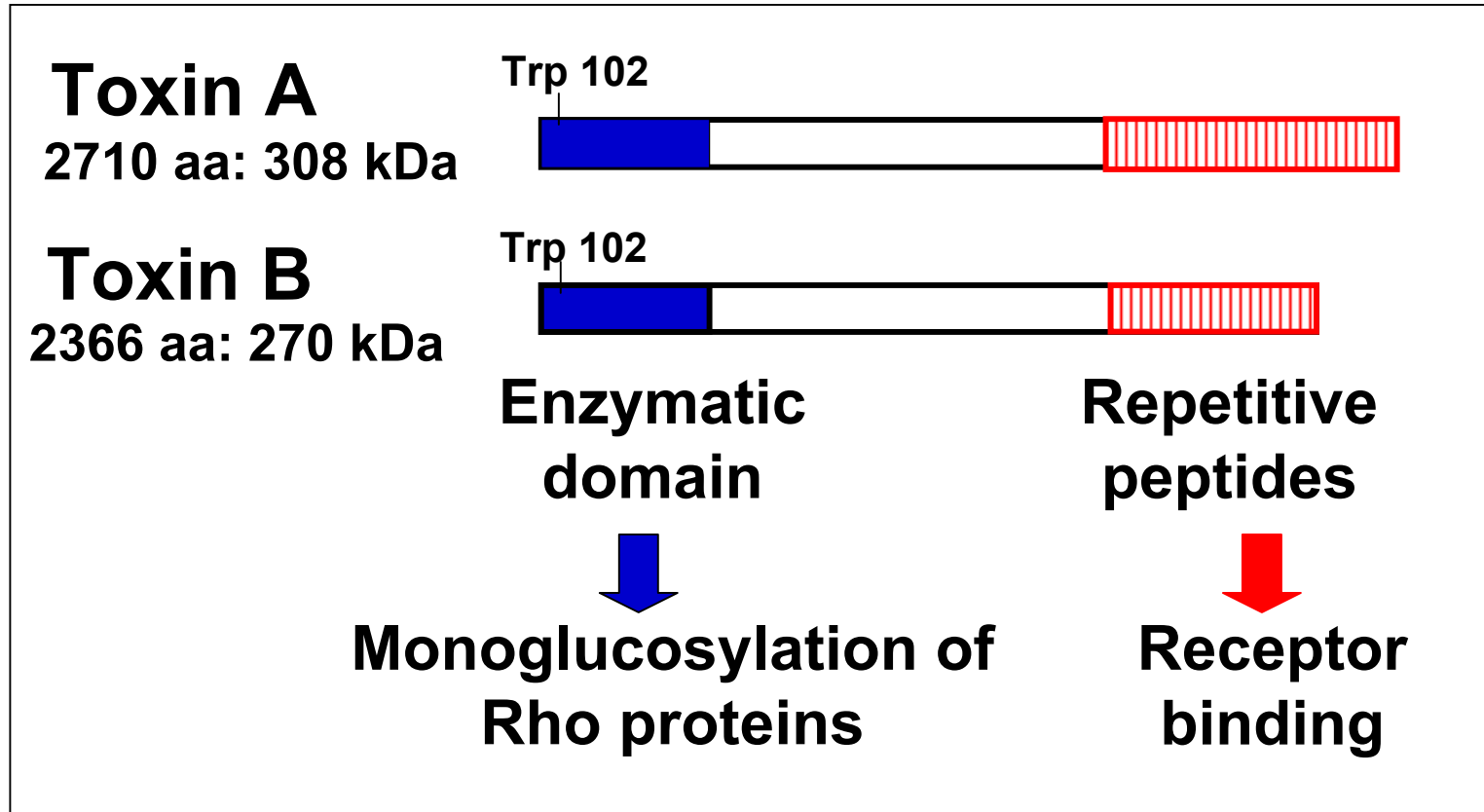
Warny et al. Infect. Immun. 1994;62:384

# Pathogenicity locus (PaLoc)

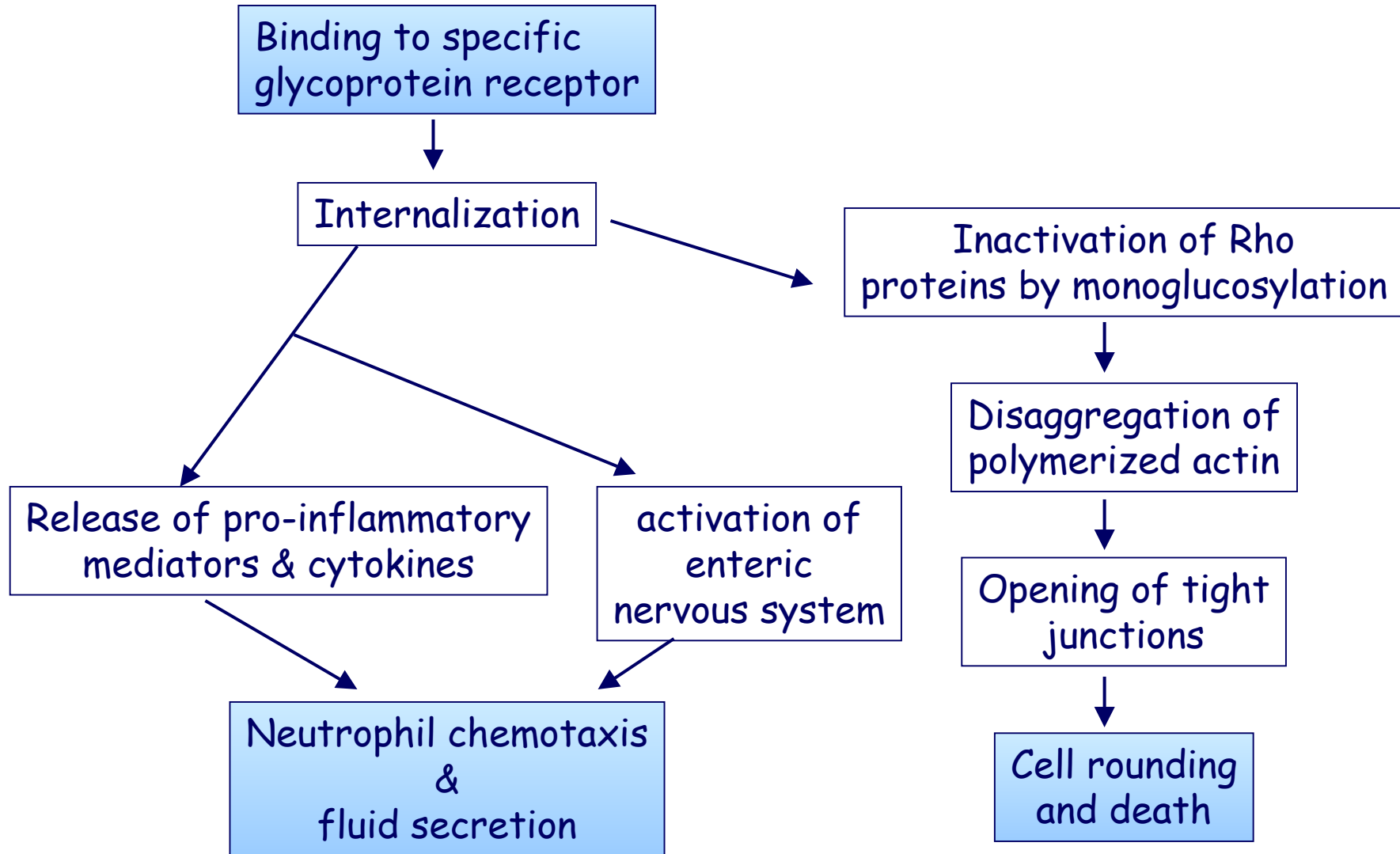


McDonald et al. NEJM, 2005; 353:2433-41

# *C. difficile* Toxin A and Toxin B Are Glycosyltransferases



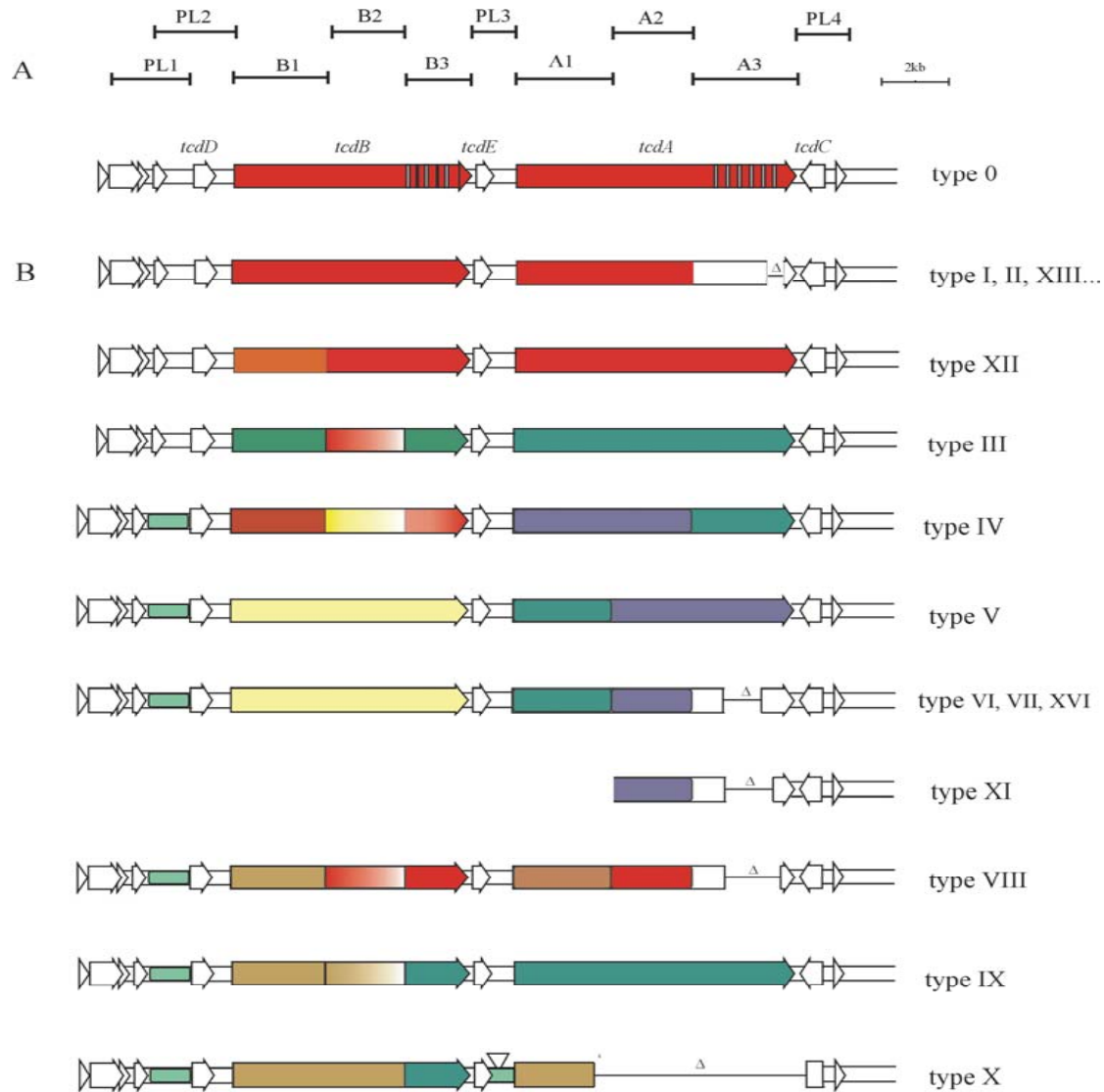
# Toxins mode of action



# Biological effects of *C. difficile* toxins

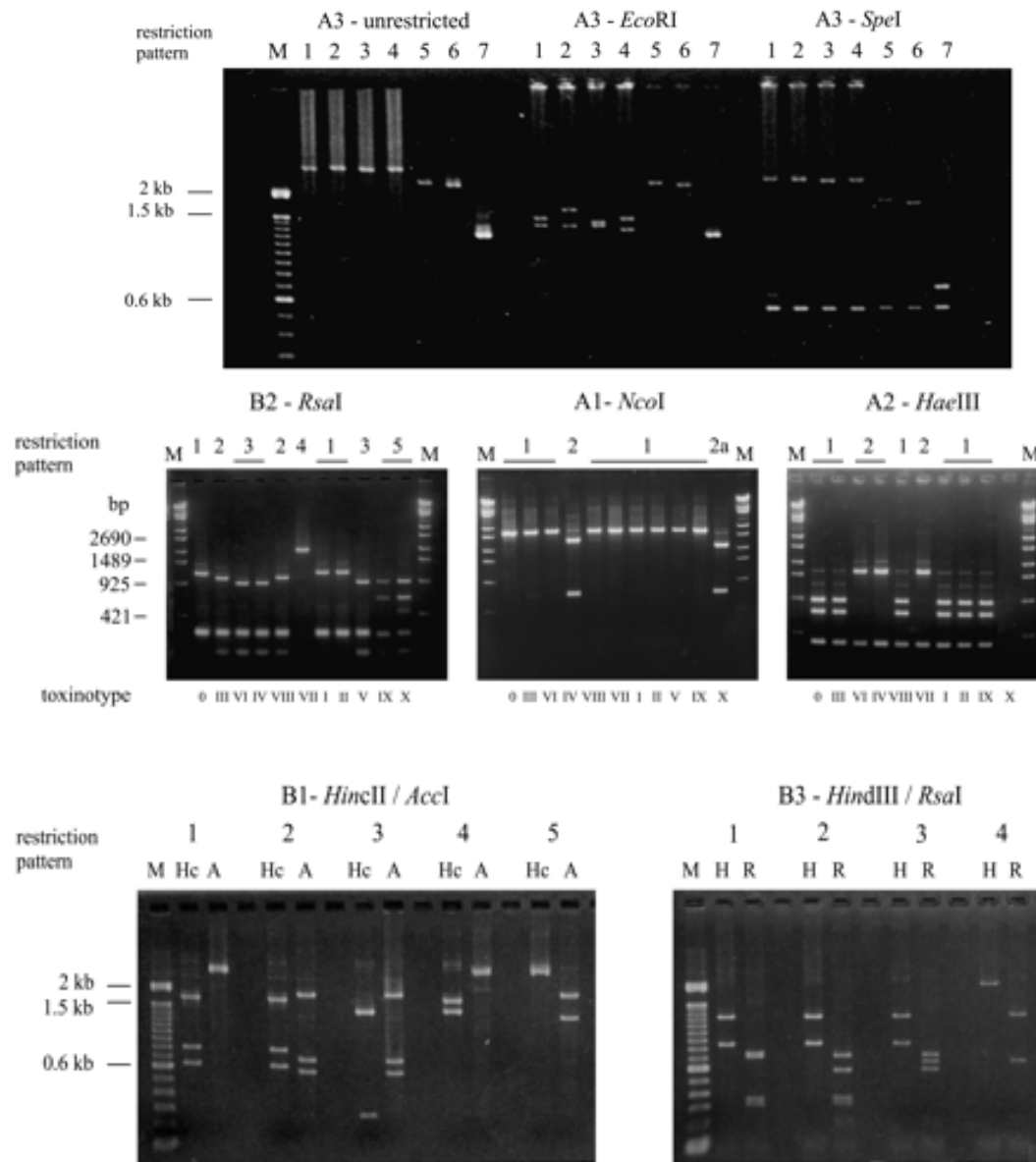
	Toxin A	Toxin B
• Enteritis in rodents	yes	no
• Mucosal damage in human colon	$10^{-8}$ M	$10^{-9}$ M
• Cytotoxicity in cultured cells	$10^{-10}$ M	$10^{-13}$ M

# toxintypage



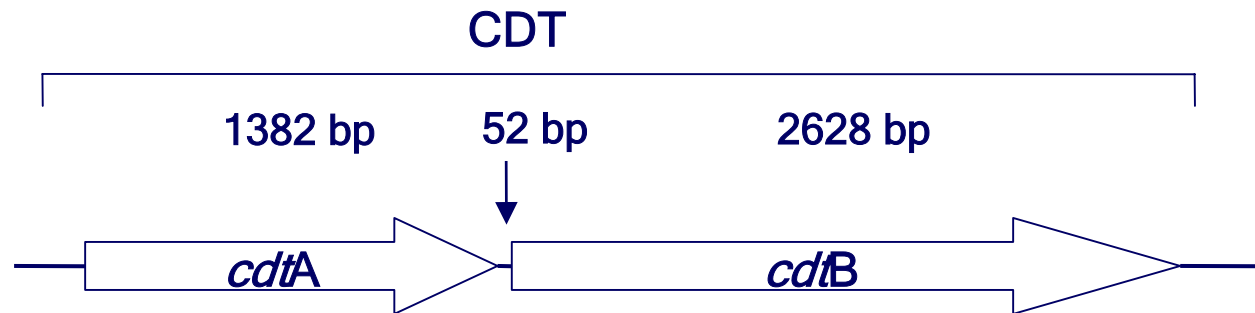


# toxintypage



Jeminfest 27/04/2006

# CDT locus for binary toxin CDT



# Binary toxin

- ❑ A-B exo-toxin
- ❑ Actin specific adenosine diphosphate-ribosyltransferase
- ❑ Encoded by *cdtA* (enzymatic component) and *cdtB* (binding component)
- ❑ Related to *C. perfringens* type E and *C. spiroforme* toxins
- ❑ 0.5 to 5% of strains
- ❑ May increase the severity of the disease  
Barbut et al. JMM 2005;54:181-5
- ❑ Not pathogenic by itself in hamster model  
Geric et al. JID 2006;193:1143-50

# *C. difficile* toxin production types

	large clostridial toxins		binary toxin
	TcdB	TcdA	CDT
type 1	+	+	-
type 2	+	+	+
type 3	+	-	-
type 4	+	-	+
type 5	-	-	+
type 6	-	-	-

## *Clostridium difficile*-associated diarrhea in a region of Quebec from 1991 to 2003: a changing pattern of disease severity

Jacques Pépin, Louis Valiquette, Marie-Eve Alary, Philippe Villemure, Anick Pelletier, Karine Forget, Karine Pépin, Daniel Chouinard

CMAJ, 2004

## Emergence of Fluoroquinolones as the Predominant Risk Factor for *Clostridium difficile*-Associated Diarrhea: A Cohort Study during an Epidemic in Quebec

Jacques Pépin,<sup>1</sup> Nathalie Saheb,<sup>2</sup> Marie-Andrée Coulombe,<sup>1</sup> Marie-Eve Alary,<sup>1</sup> Marie-Pier Corriveau,<sup>1</sup> Simon Authier,<sup>1</sup> Michel Leblanc,<sup>2</sup> Geneviève Rivard,<sup>1</sup> Mathieu Bettez,<sup>1</sup> Valérie Primeau,<sup>1</sup> Martin Naudon,<sup>1</sup> Claude-Émilie Jacob,<sup>1</sup> and Luc Lanthier<sup>2</sup>

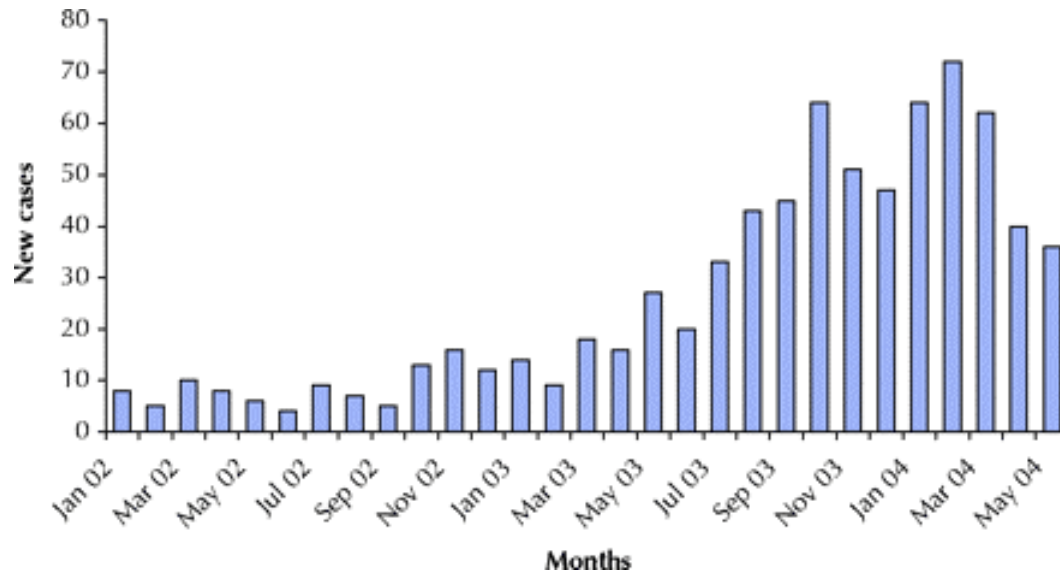
Clin. Infect. Dis., 2005

## A LARGE OUTBREAK OF *CLOSTRIDIUM DIFFICILE*-ASSOCIATED DISEASE WITH AN UNEXPECTED PROPORTION OF DEATHS AND COLECTOMIES AT A TEACHING HOSPITAL FOLLOWING INCREASED FLUOROQUINOLONE USE

Carlene A. Muto, MD, MS; Marian Pokrywka, MPH, BS, CIC; Kathleen Shutt, MS; Aaron B. Mendelsohn, PhD; Kathy Nouri, MPH, RN, BSN, CIC; Kathy Posey, MPH, BS, CIC; Terri Roberts, BS, CIC; Karen Croyle, Sharon Krystofiak, MPH, MS, CIC; Sujata Patel-Brown, BS; A. William Pasculle, ScD; David L. Paterson, MD; Melissa Saul, MS; Lee H. Harrison, MD

Infect. Control Hosp. Epidemiol., 2005

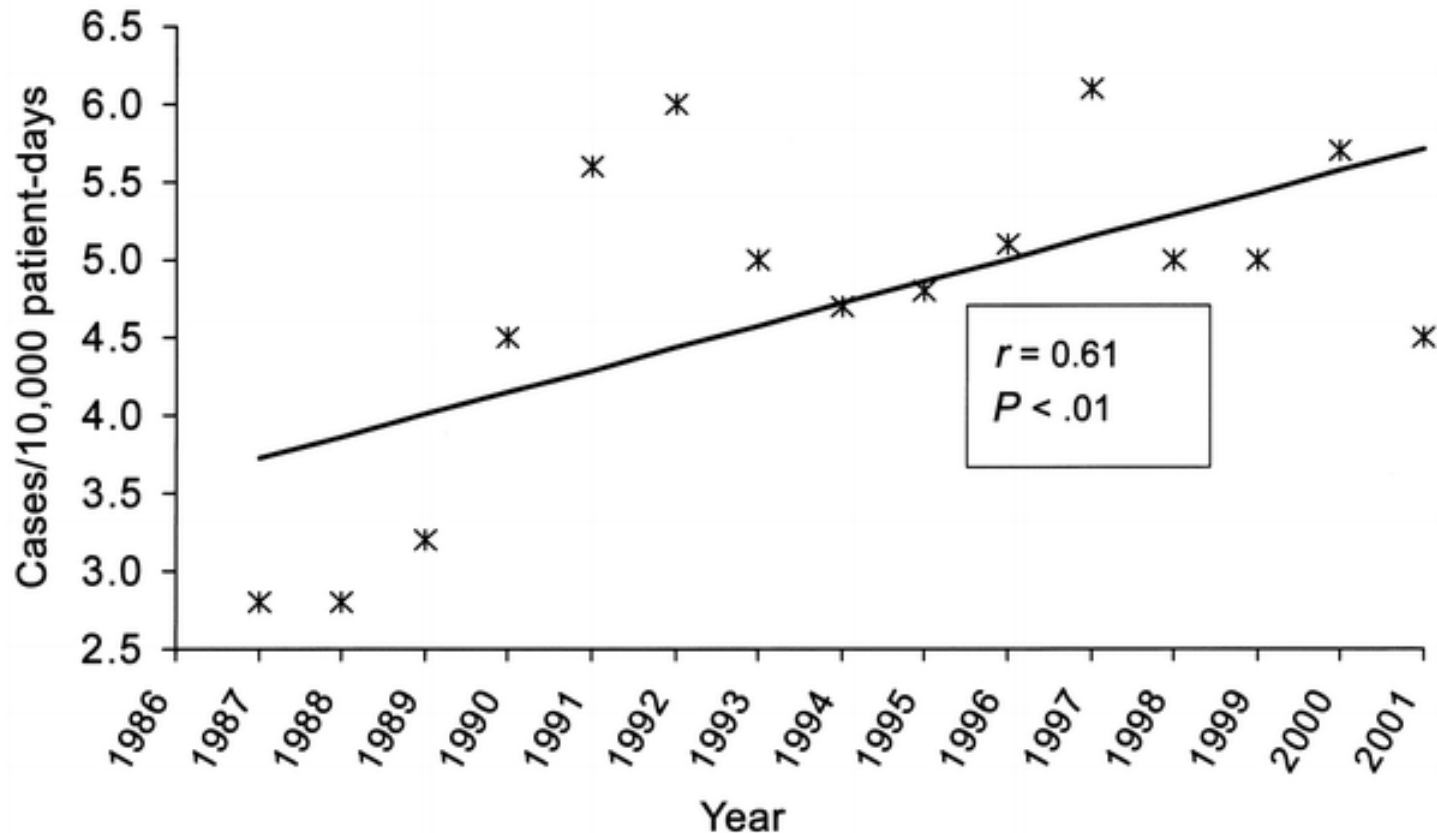
# North America outbreak



New cases of nosocomial and community-acquired *Clostridium difficile*-associated diarrhea (CDAD; diagnosed by positive cytotoxin assay result) reported by the microbiology laboratory at the Centre hospitalier universitaire de Sherbrooke.

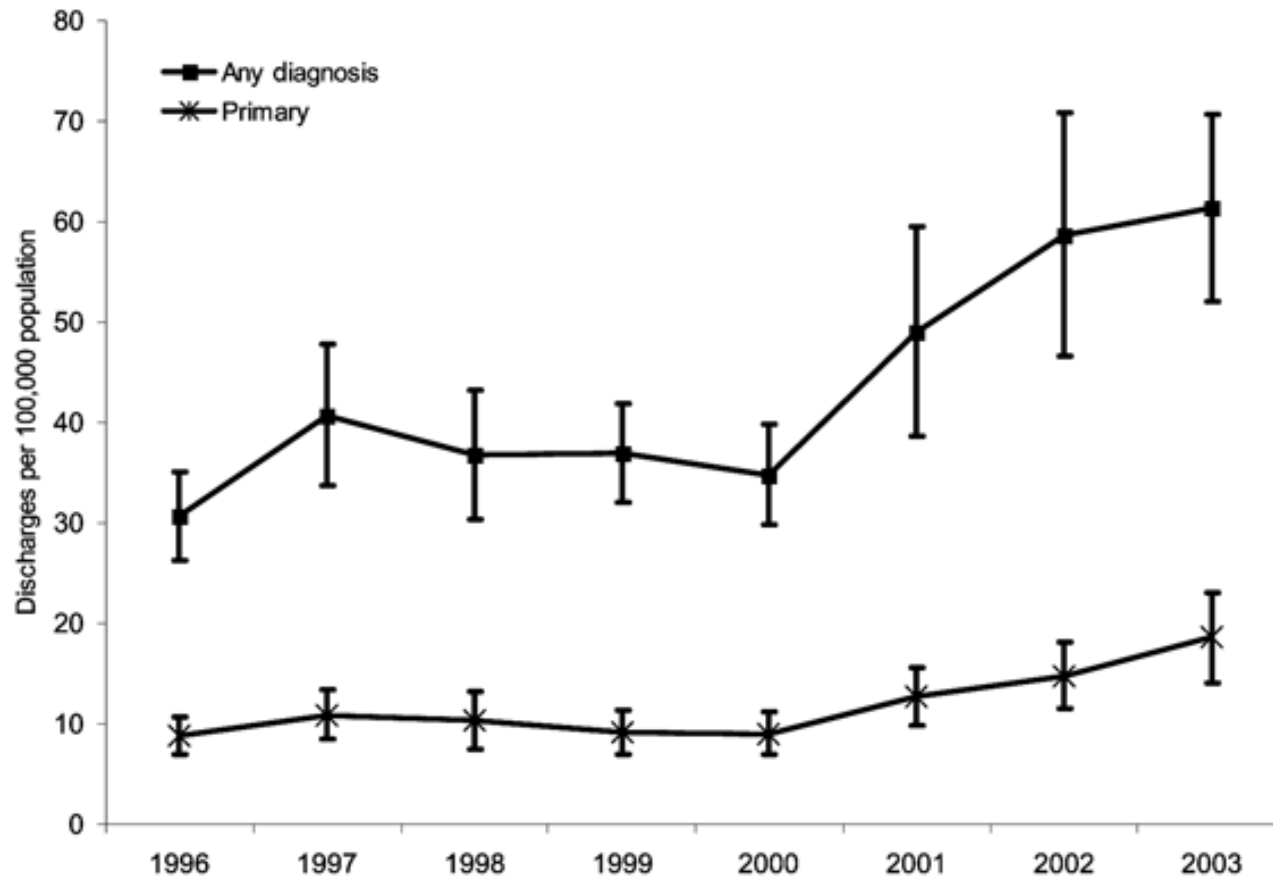
Valiquette et al. CMAJ 2004 171: 27-29

# USA 1987-2001



Annual CDAD rates for hospitals with >500 beds, by intensive care unit surveillance component (NNISS, 1987 2001).

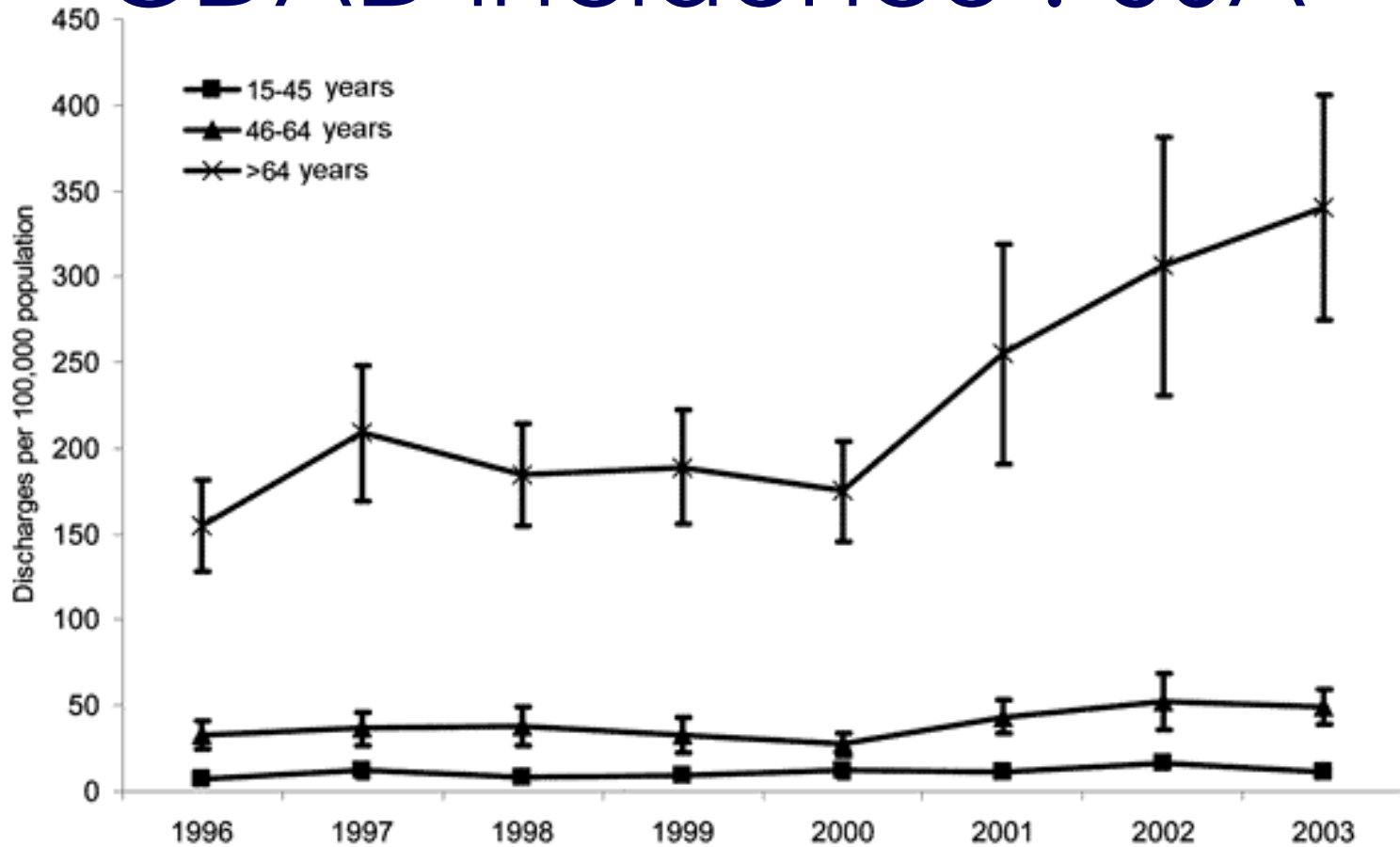
# CDAD incidence : USA



National estimates of US short-stay hospital discharges with *Clostridium difficile* listed as primary or as any diagnosis.

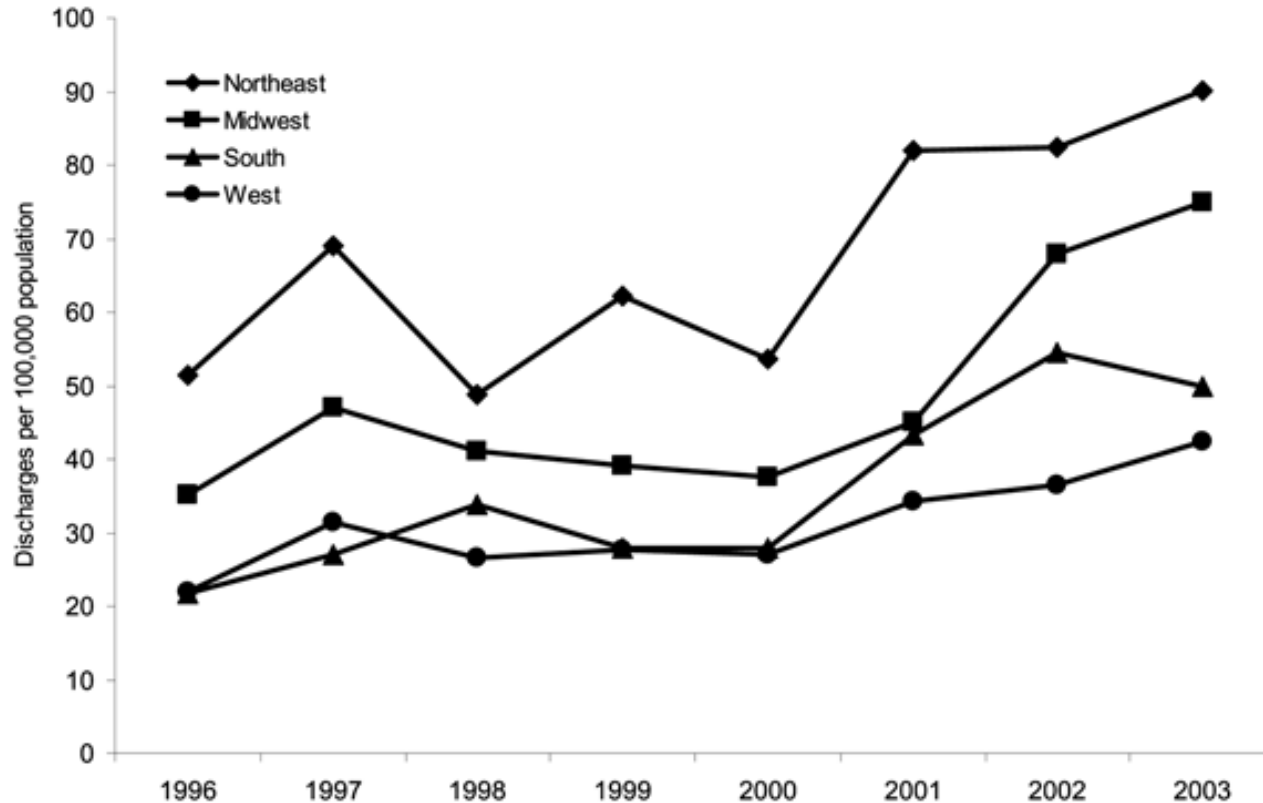


# CDAD incidence : USA



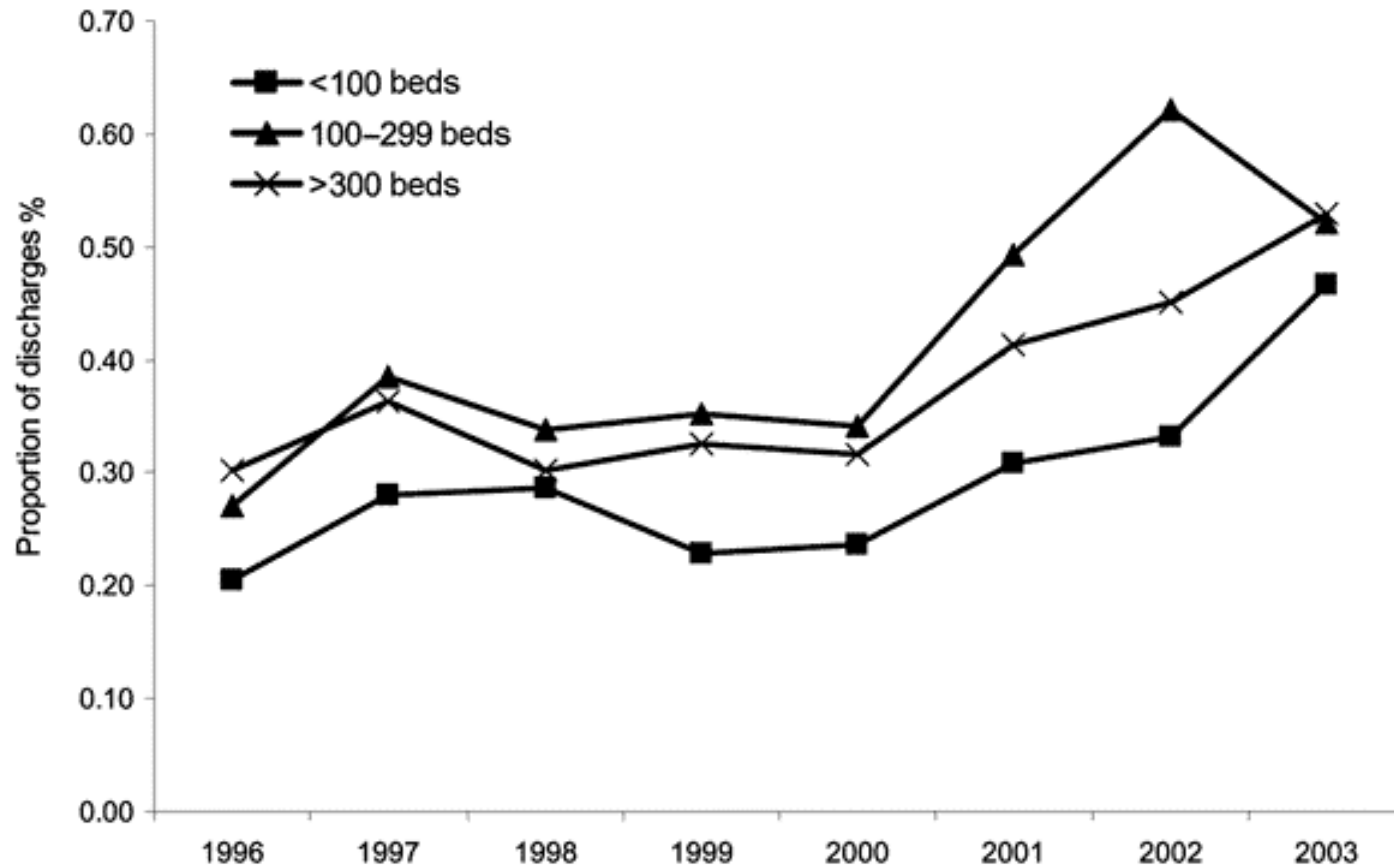
Rates of US short-stay hospital discharges with *Clostridium difficile* listed as any diagnosis, by age..

# CDAD incidence : USA



Rates of US short-stay hospital discharges with *Clostridium difficile* listed as any diagnosis, by region

# CDAD incidence : USA



Rates of US short-stay hospital discharges with *Clostridium difficile* listed as any diagnosis, by hospital size

# CDAD incidence and severity

**Table 1: Reports of increases in incidence and severity of *Clostridium difficile*-associated diarrhea (CDAD)**

Source	Period	Outcome measure	Results
US National Nosocomial Infection Surveillance System <sup>9</sup>	1987–2001	CDAD among patients admitted to intensive care unit in hospitals with > 500 beds	Doubling of incidence
UK Health Protection Agency Communicable Disease Surveillance Centre <sup>10</sup>	1986–2001	Voluntary laboratory reporting system of stools positive for <i>C. difficile</i> toxin	From < 2000 positive test results per year in 1986/87 to > 12 000 per year in 2000/01
Oregon <sup>11</sup>	1994–2000	All-cause 90-day mortality among patients with CDAD	3.5% in a previous 10-year cohort v. 15.3% in 1994–2000 cohort
		Colectomy	7.6% in 1994–2000 cohort
Pittsburgh <sup>12</sup>	1989–2000	Fulminant colitis	From 0% to 3.2% (mean 1.6% over 10 years)

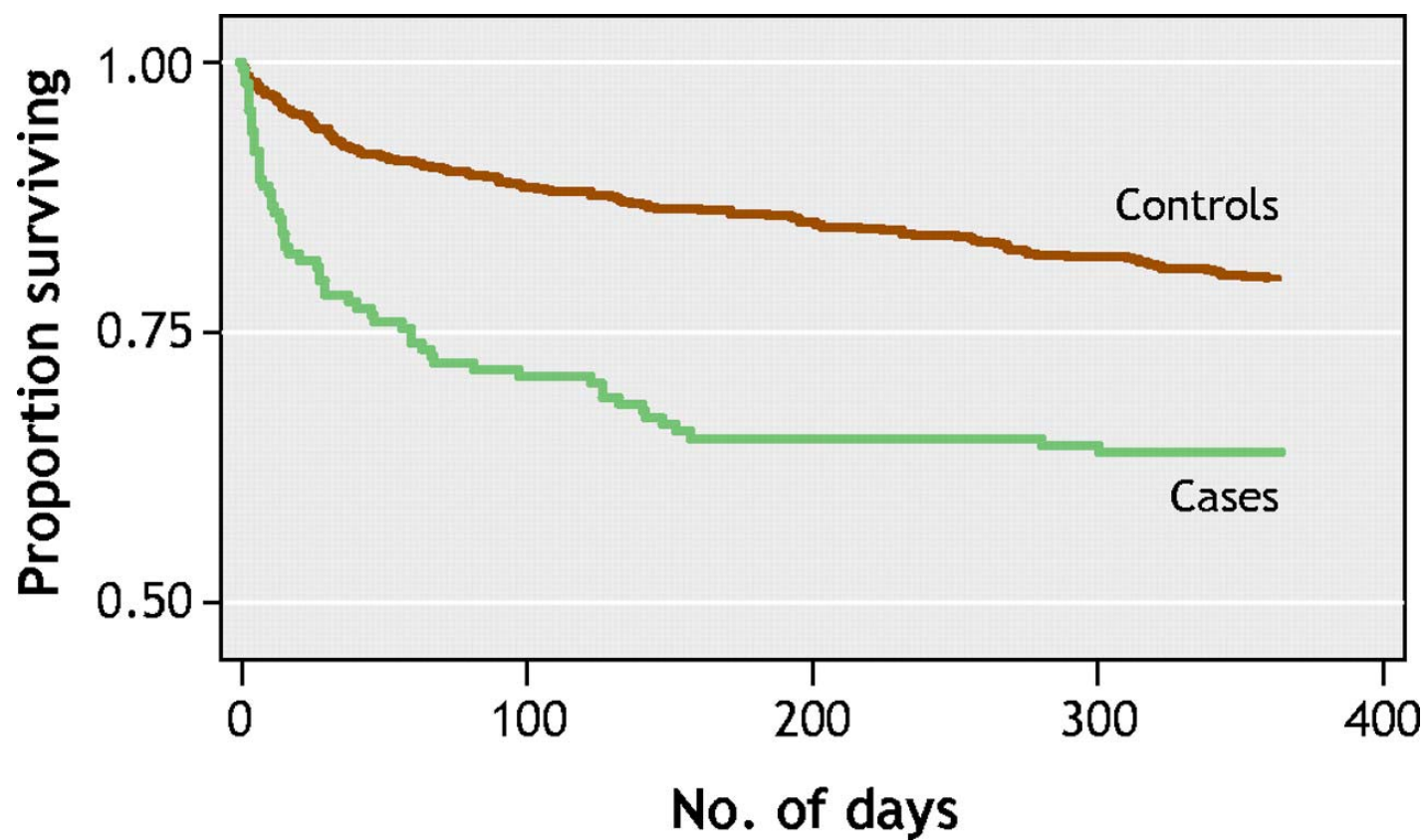
# CDAD incidence : Canada

**Table 2.** Age-Specific Incidence and Mortality Attributed to *Clostridium difficile*-Associated Diarrhea.

Age yr	No. of Cases	No. of Cases/ 1000 Admissions*	Attributable 30-Day Mortality Rate %†
<40	76	3.5	2.6
41–50	85	11.2	1.2
51–60	191	20.0	3.2
61–70	272	24.4	5.1
71–80	523	38.3	6.2
81–90	458	54.5	10.2
>90	114	74.4	14.0

\* Values are based on 1719 episodes of nosocomial *C. difficile*-associated diarrhea.

† Values are based on data from 1703 patients with nosocomial *C. difficile*-associated diarrhea.



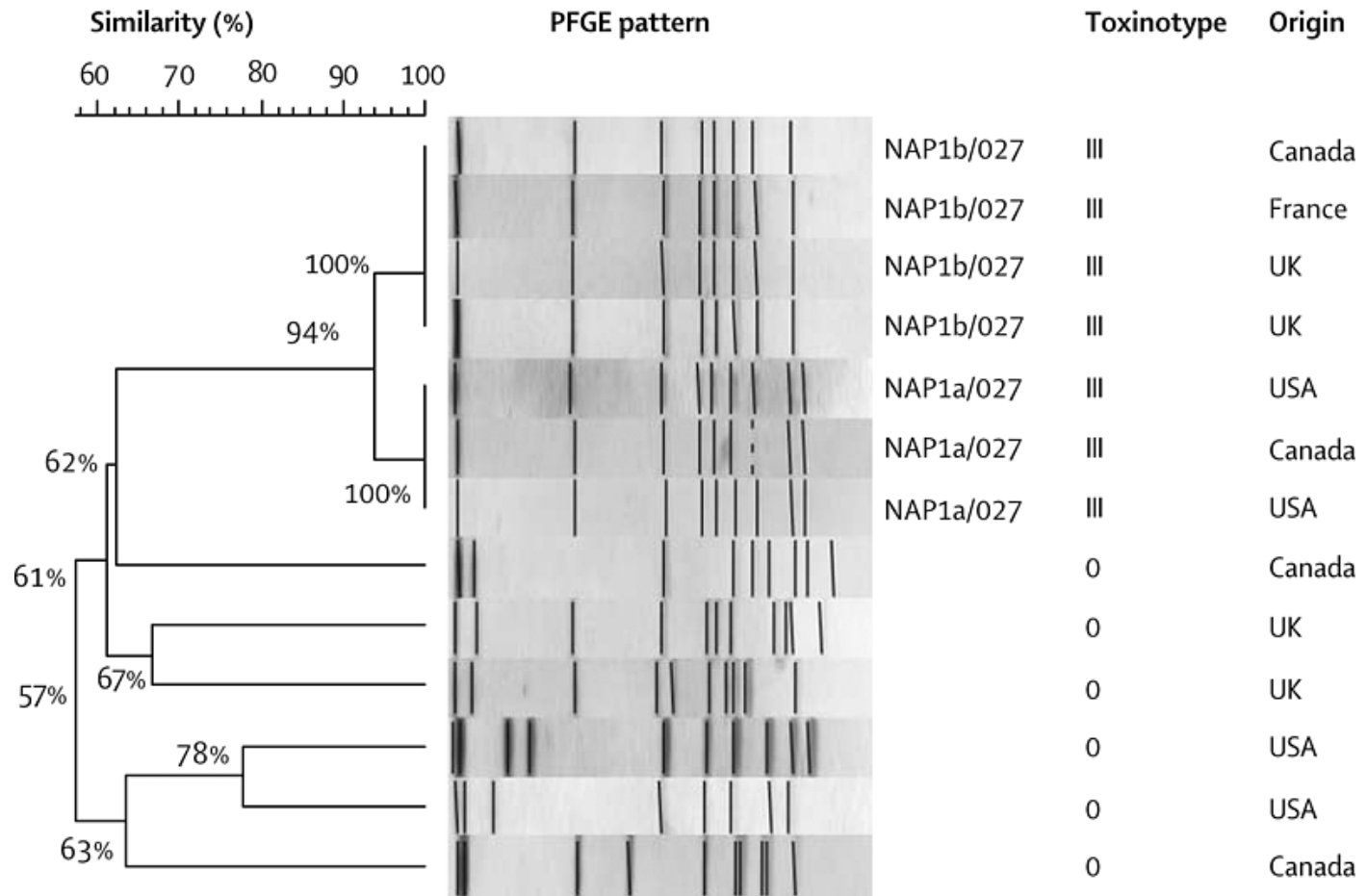
# NAP1/027 isolate

**Table 1.** Isolates of *Clostridium difficile* According to Health Care Facility and the Proportion of Isolates Belonging to the BI/NAP1 Strain.

Health Care Facility	Date of Onset of Outbreak	No. of Isolates Tested	BI/NAP1 Strain
			no. (%)
Georgia	Oct. 2001	46	29 (63)
Illinois	July 2003	14	6 (43)
Maine, Facility A	March 2002	13	9 (69)
Maine, Facility B	July 2003	48	30 (62)
New Jersey	June 2003	12	9 (75)
Oregon*	April 2002	30	3 (10)
Pennsylvania, Facility A	2000–2001	18	7 (39)
Pennsylvania, Facility B	Oct. 2003	6	3 (50)
Total		187	96 (51)

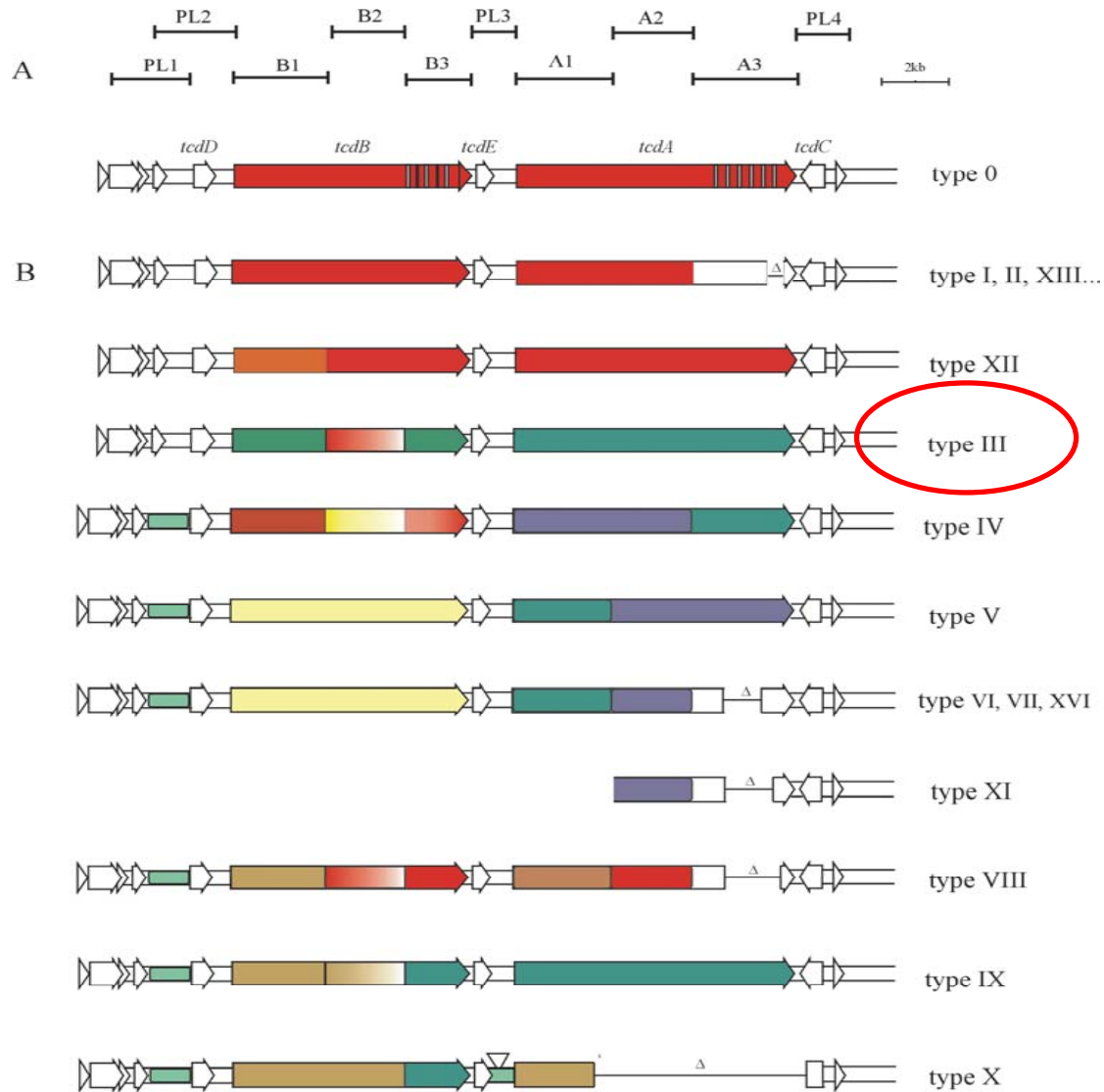
\* Isolates were not collected until after the peak of the outbreak.

# NAP1/027 isolate

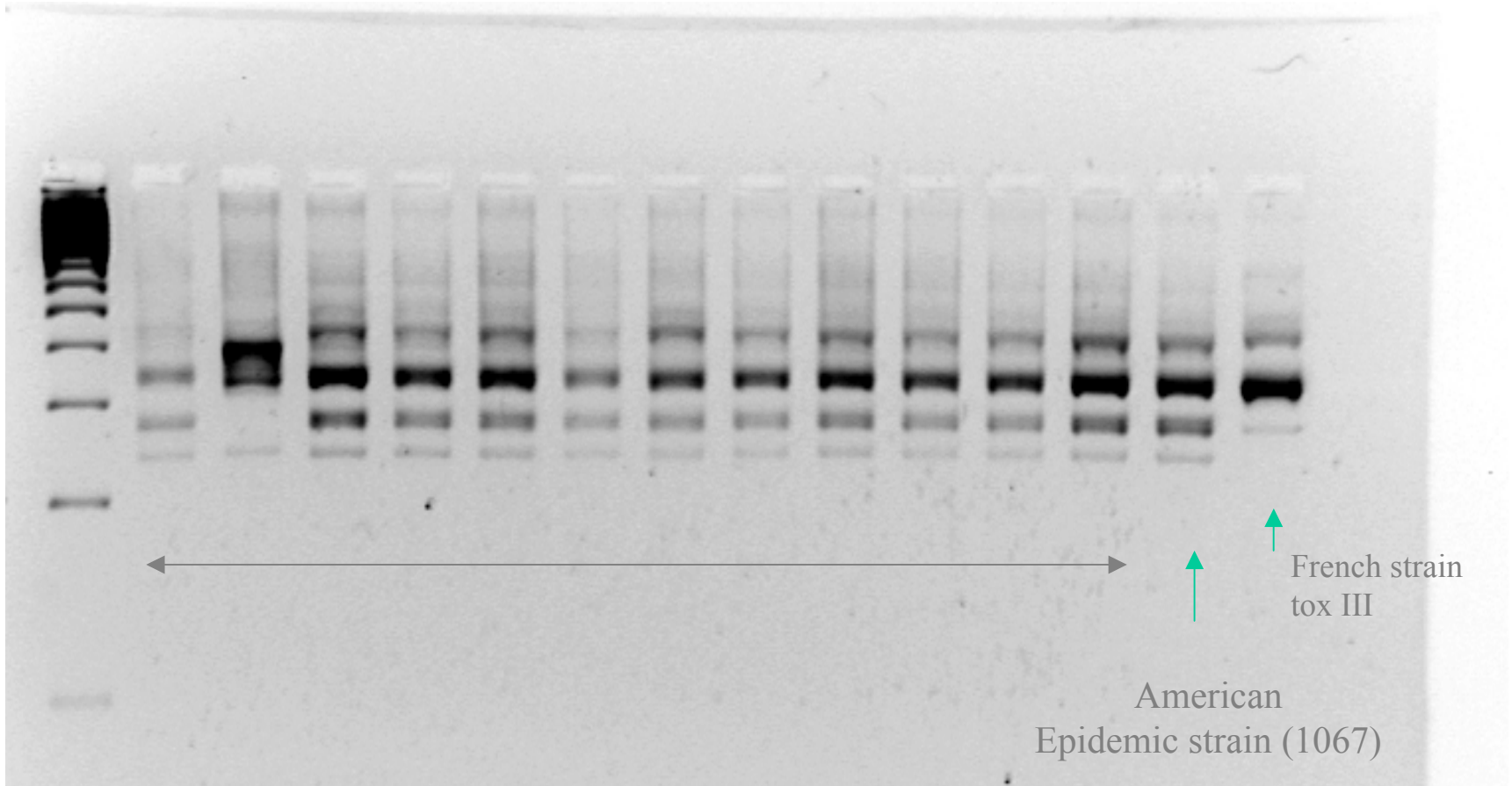




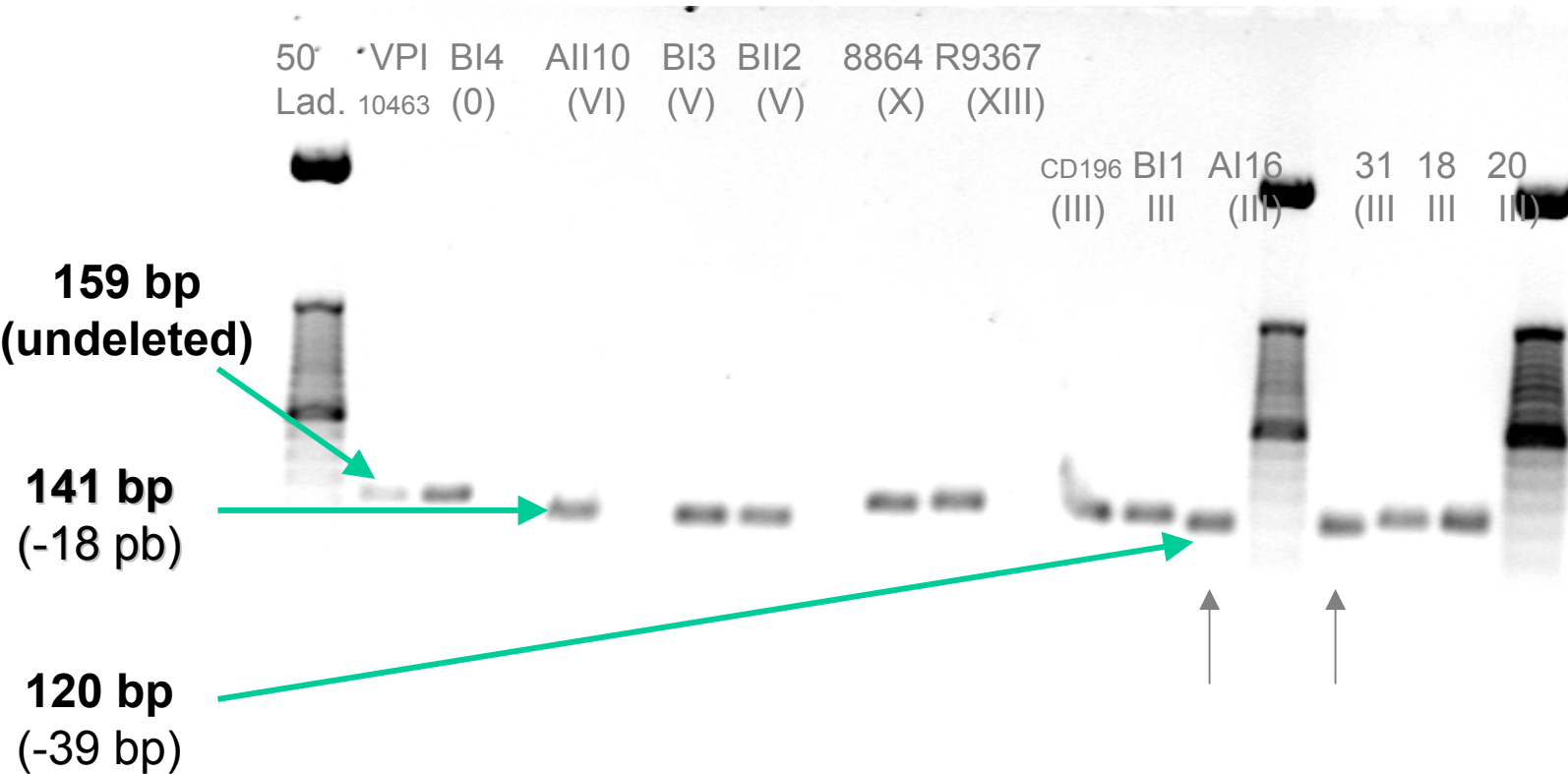
# toxinotyping



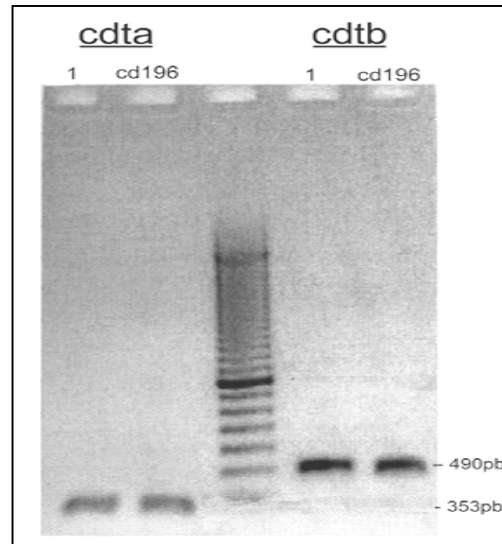
# ribotyping



# *tcdC* polymorphism

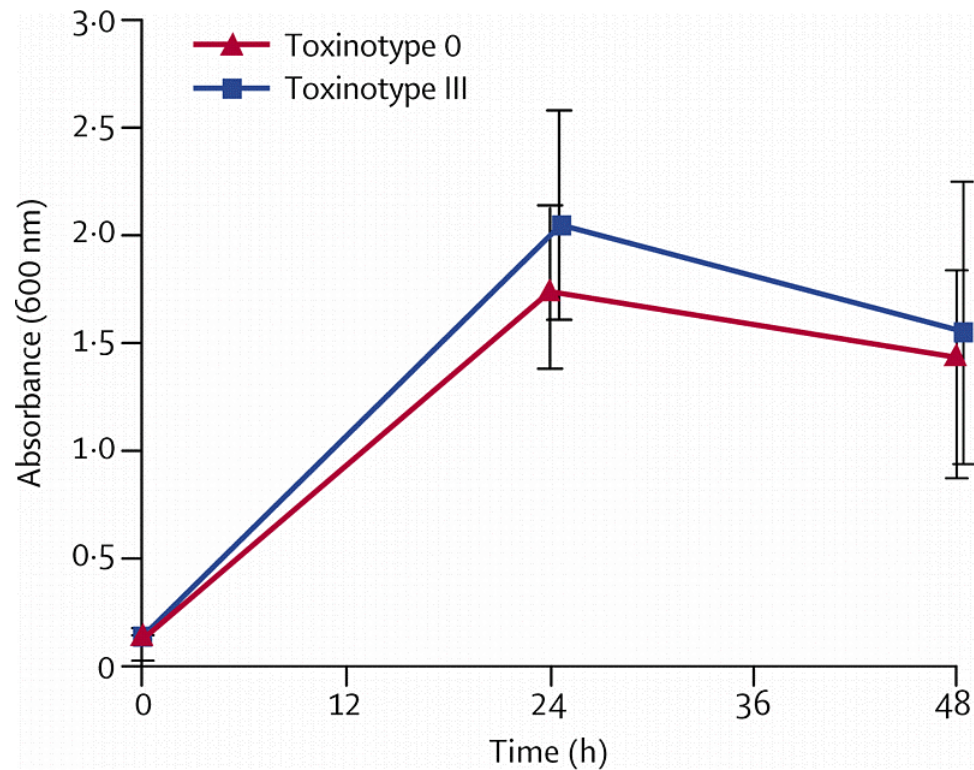


# Binary toxin



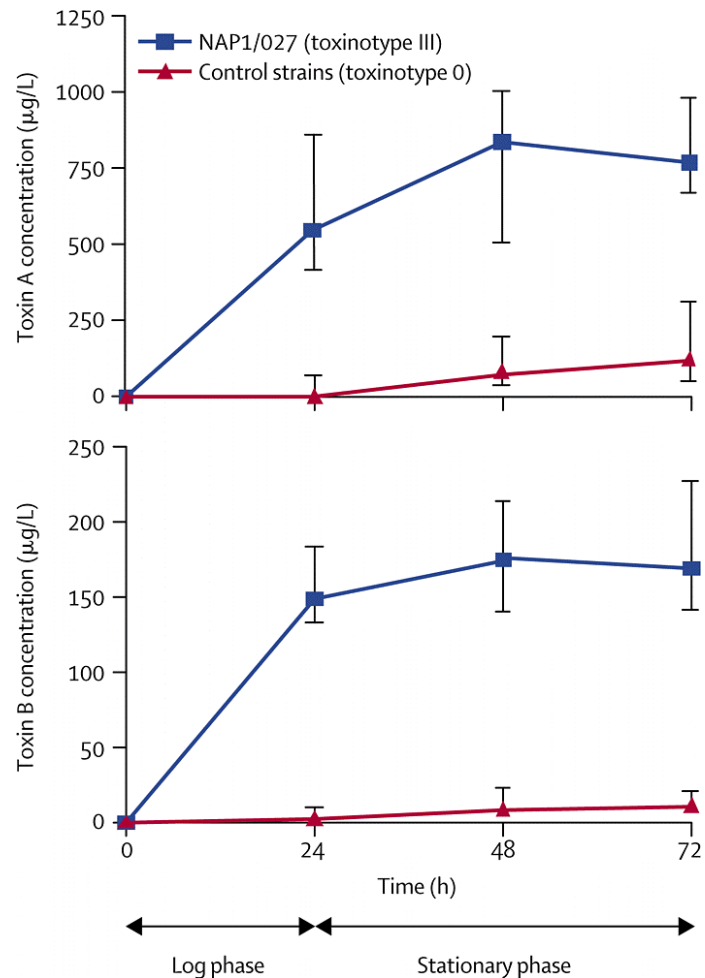
# NAP1/027 isolate

**Growth curves of  
toxintype 0 and  
toxintype III  
(NAP1/027)**

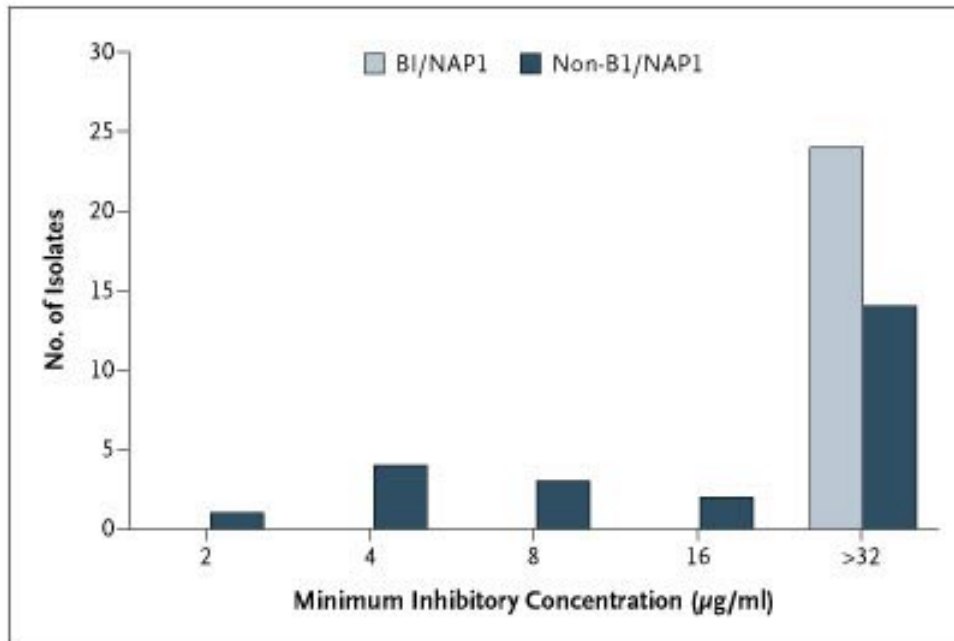


# NAP1/027 isolate

**In vitro production of  
toxins A and B by *C*  
*difficile* isolates**



# NAP1/027 isolate



Distribution of MIC of Levofloxacin for Current (Obtained after 2000) BI/NAP1 and Non-BI/NAP1 *Clostridium difficile* Isolates.

McDonald et al. NEJM, 2005; 353:2433-41

Table 3. Characteristics of Case Patients and Control Patients.*			
Characteristic	Case Patients (N=237)	Controls (N=237)	P Value
Age — yr			0.48
Median	75	75	
Interquartile range	66–82	66–82	
Male sex — no. (%)	115 (48.5)	126 (53.2)	0.3
Charlson index†	2.6±1.9	2.6±2.0	0.66
Ward			0.82
Medicine	133 (56.1)	142 (59.9)	
Surgery	78 (32.9)	70 (29.5)	
Geriatrics	17 (7.2)	15 (6.3)	
Oncology	9 (3.8)	10 (4.2)	
Community hospital — no. (%)	68 (28.7)	67 (28.3)	0.9
Days at risk for <i>C. difficile</i> -associated diarrhea			0.02
Median	13	16	
Interquartile range	6–25	8–29	
No. of antibiotics received	1.9±1.1	1.3±1.3	<0.001
Any exposure to antibiotics — no. (%)	188 (79.3)	141 (59.5)	<0.001
Cephalosporins	115 (48.5)	65 (27.4)	<0.001
Clindamycin	19 (8.0)	6 (2.5)	0.007
Fluoroquinolones	128 (54.0)	75 (31.6)	<0.001
Chemotherapy — no. (%)	17 (7.2)	13 (5.5)	0.45
Proton-pump inhibitors — no. (%)	112 (47.3)	111 (46.8)	0.92
Histamine H <sub>2</sub> -blockers — no. (%)	47 (19.8)	47 (19.8)	1.0
Enteral feeding — no. (%)	44 (18.6)	28 (11.8)	0.04

\* Plus-minus values are means ±SD.

† Scores for the Charlson index can range from 0 to 37, with higher scores indicating more coexisting conditions.



# NAP1/027 isolate

- ❑ Toxinotype III
- ❑ 18bp deletion *tcdC*
- ❑ Increased production tox A and B
- ❑ Ribotype 027/ PFGE NAP1
- ❑ Binary toxin
- ❑ FQ resistance

# EUROPEAN PROSPECTIVE STUDY OF *CLOSTRIDIUM DIFFICILE* STRAINS: PHENOTYPIC AND GENOTYPIC CHARACTERIZATION OF THE ISOLATES FROM DIFFERENT CLINICAL STATUS : INTERIM RESULTS

F. Barbut, P. Mastrantonio, M. Delmée, G. Ackermann,  
E. Bouza, C. Balmelli, D. Drudy, E. Kuijper, H. Ladas,  
E. Nagy, H. Pituch, M. Somolinos, E. Urban, M. Wullt, M. Yücesoy, M.  
Rupnik and I. Poxton

for the European Study Group on *Clostridium difficile*  
(*ESGCD*)

# OBJECTIVES

## Main objectives :

- ❑ To establish a well defined European collection of *C. difficile* strains
- ❑ To study and to compare the phenotypic and genotypic markers of isolates (*prevalence of toxinotype III ?*)
- ❑ To get an estimation of the incidence of *C. difficile* infections

## Other objectives :

- ❑ To correlate clinical presentations with phenotypic and genotypic features *C. difficile*.
- ❑ To get the baseline characteristics of *C. difficile* strains isolated in 2005 making further investigations possible to follow trends in antimicrobial susceptibility, serogroups, genotypic patterns...

# MATERIALS AND METHODS

## □ Design

- Prospective European-wide study on *C. difficile* strains isolated during a 2-month period with phenotypic and genotypic characterization of isolates
- Requirements for participating hospitals:
  - to have an on-site laboratory of microbiology
  - to systematically perform culture each time a test for *C. difficile* is requested.
  - to be able to detect toxins A or B from stools or from strains
  - to fill out a questionnaire (=data form) for each isolated CD strain (whatever the toxin result is)

# MATERIALS AND METHODS

## □ Inclusion criteria:

All the *C. difficile* strains (including the non toxigenic strains) isolated from **inpatients** (with community or nosocomial diarrhea)

## □ Exclusion criteria:

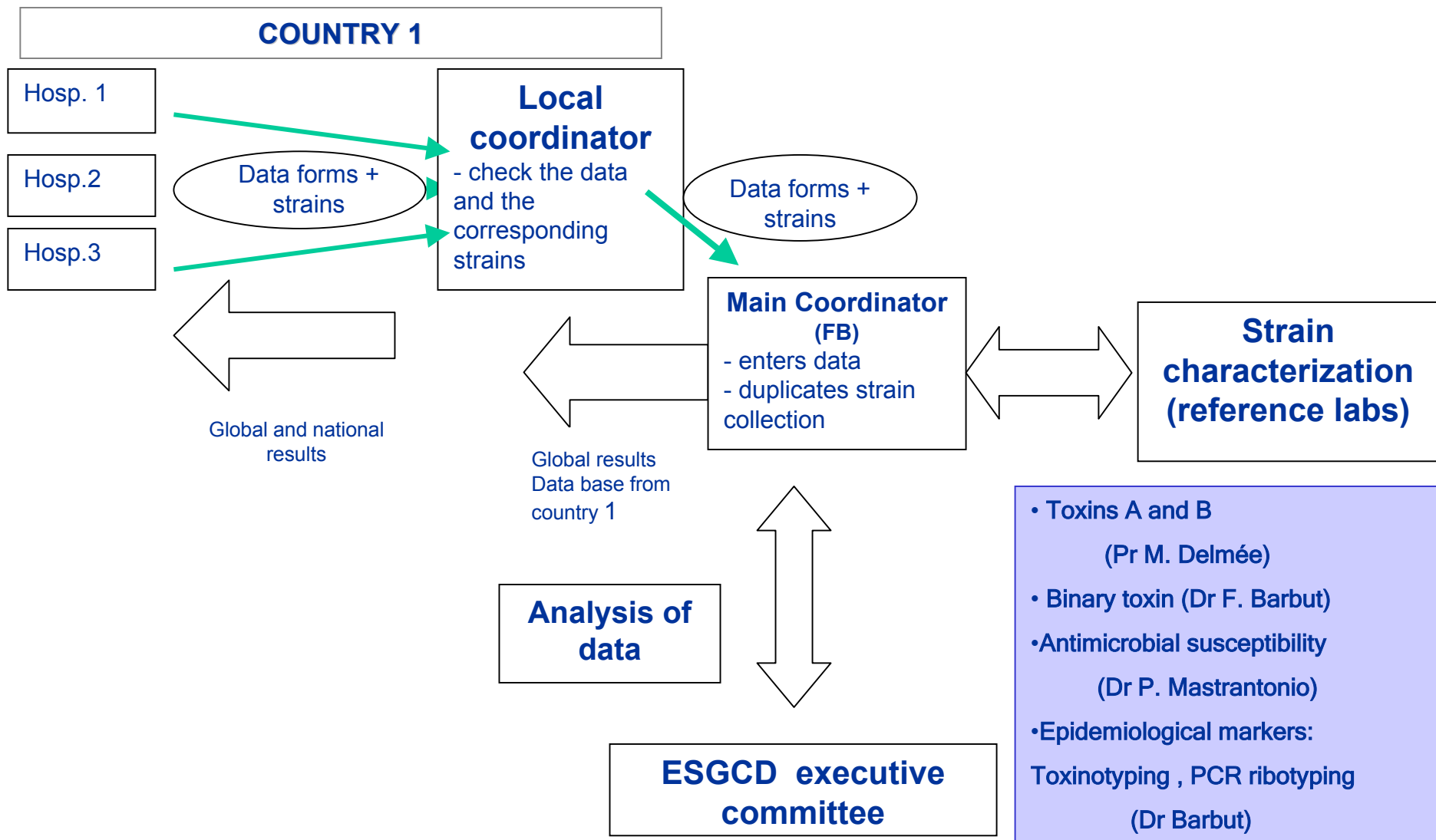
Strains from **outpatients** or **day-care patients**.

Strains from **children** under 2 years old

## □ Length of the study :

a 2 month-period (or 1 month for every hospital which reaches 30 *C. difficile* strains at the end of the first month) between April and June.

# GENERAL ORGANIZATION OF THE STUDY



# PARTICIPATING COUNTRIES

14 countries, 38 hospitals, 486 strains

COUNTRY	Letter	No. Hospitals	Local coordinator	No. Strains
Belgium	A	3	M. Delmée	73
France	B	3	F. Barbut	53
Germany	C	3	G. Ackermann	86
Hungary	E	4	E. Nagy	48
GB	R	1	I. Poxton	9
Italy	F	3	P. Mastrantonio	27

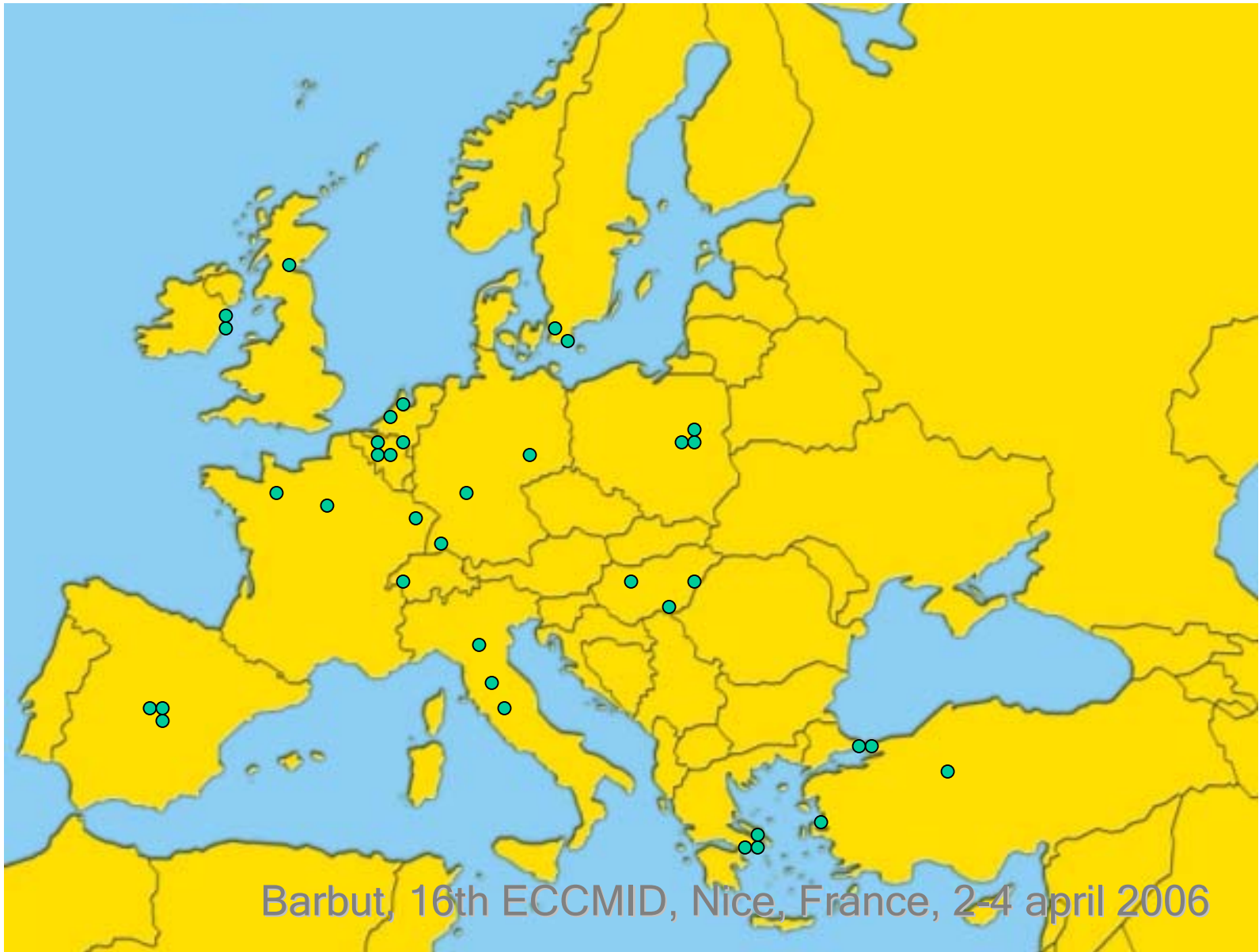
Barbut, 16th ECCMID, Nice, France, 2-4 april 2006

Seminfect 27/04/2006

COUNTRY	Letter	No. Hospitals	Local coordinator	No. Strains
Netherlands	G	3	E. Kuijper	29
Poland	H	3	H. Pituch	20
Spain	K	3	E. Bouza M. Somolinos	39
Switzerland	L	1	C. Balmelli	15
Sweden		3	M. Wult B. Andersson	22
Turkey	N	4	M. Yucesoy	9
Greece	P	3	H. Ladas	24
Ireland	D	1	D. Drudy	32



# Distribution of participating hospitals



Barbut, 16th ECCMID, Nice, France, 2-4 april 2006

# clinical data

Incidence varies widely (23 hosp.):  
 $2.45 \pm 1.8$  CDAD/10,000 patient-days  
(range : 0.14- 7.1)

Hospitals N :	0.13-0.14 CDAD/10,000 patient-days
Hospitals F:	0.5-1.2 «
Hospitals G, B:	2-7 «

USA : 12.1 CDAD /10,000 patients days (range 3-25.1)

(Sohn, ICHE 2005)

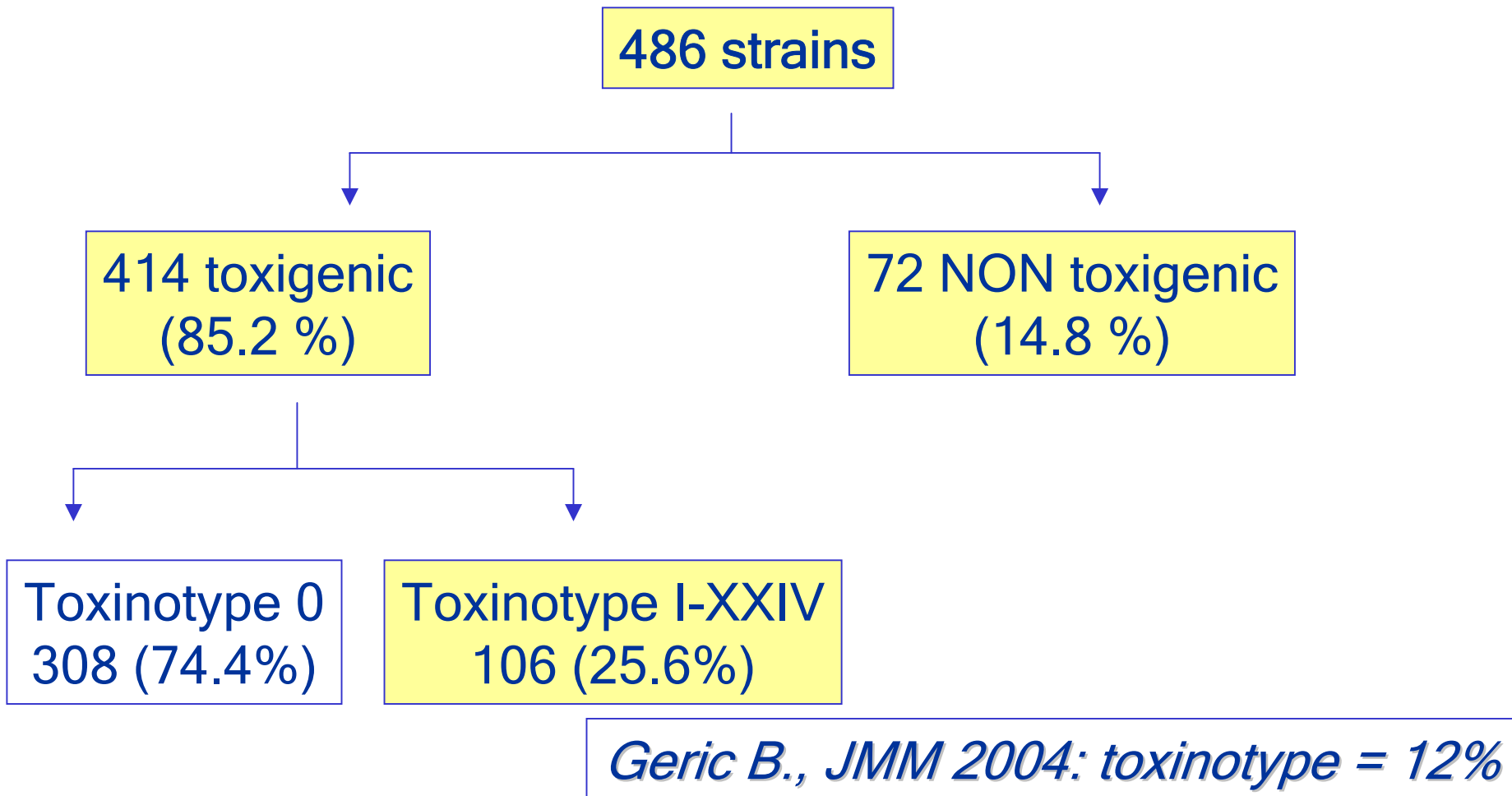
USA : 5 /10,000 patient-days

(Archibald, CDC, JID 2004)

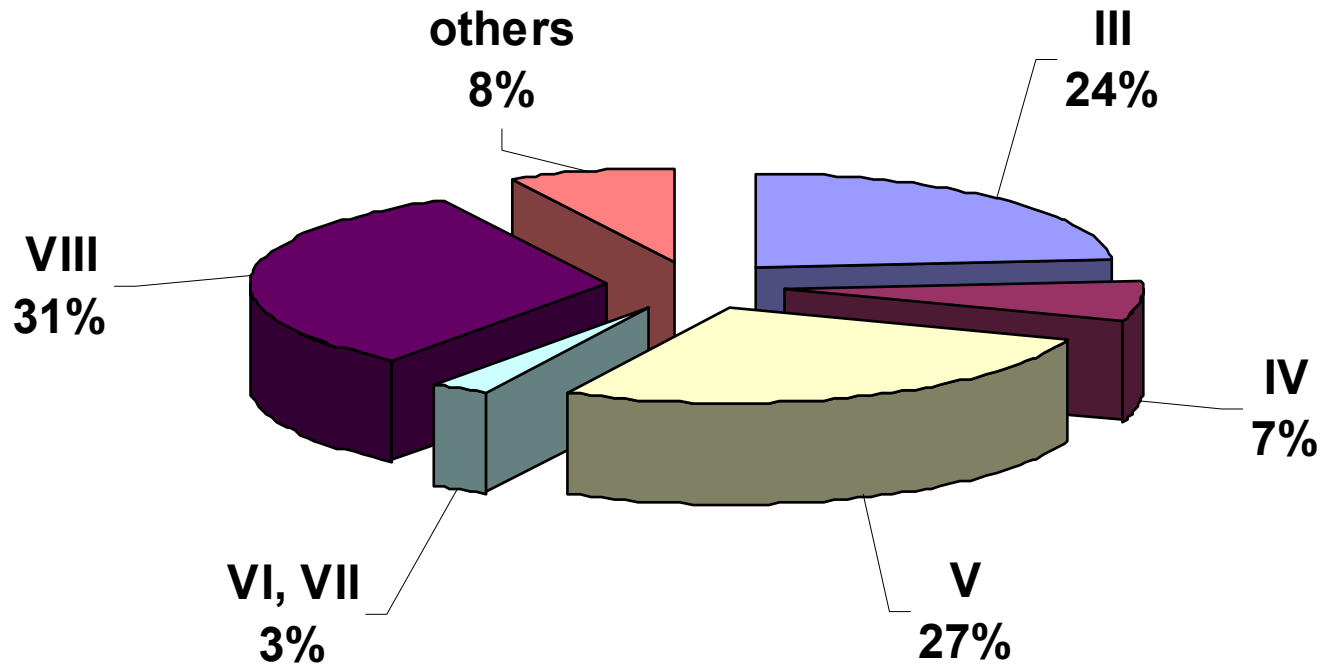
Québec : 12.8 /10,000 patients-days

Barbut, 16th ECCMID, Nice, France, 2-4 april 2006

# Toxigenicity



# Distribution of toxin variant strains (N=106)



USA (Geric, JMM 2004) : III (17%), V (6%), VIII (12%)

# ANTIMICROBIAL SUSCEPTIBILITY

ATB	MIC50 (mg/l)	MIC90 (mg/l)	Range (min. max)	% R
VA	0.5	0.75	0.25-2	0
MZ	0.06	0.125	0.012-0.75	0
EM	1	>256	0.047->256	46.7
CM	4	>256	0.016->256	50.0
TC	0.032	0.38	0.023->32	8.8
MX	0.5	>32	0.032->32	33.9

Seminfect 27/04/2006

Barbut, 16th ECCMID, Nice, France, 2-4 april 2006

# ANTIMICROBIAL SUSCEPTIBILITY

Country	No. strains	% R EM+CM+TC+MX	% S EM+CM+TC+MX
Belgium	72	1.4	33.3
France	50	2	42
Germany	85	8.2	37.6
Ireland	31	9.7	32.3
Hungary	47	17	55.3
Italy	27	0	14.8
Netherlands	31	0	45.2
Poland	19	5.3	26.3
Spain	39	0	12.8
Switzerland	15	0	80
Sweden	22	0	81.8
Greece	23	8.7	21.7
Turkey	9	11.1	77.8
UK	8	0	12.5

# Toxinotype III

- ❑ 25 strains isolated in 6 different countries
- ❑ 2 types of toxinotype III isolates by PCR-ribotyping
  - « 027 » (n=20) : AI13, AII6, AII9, AII12, AII25, AII27, AIII2, AIII3, AIII5, AIII8, AIII14, GII1, GII3, GII5, GII6, GII7, GII8, GII9, GII 10, DI12
  - Non « 027 » (n=5) : AI16, KIII3, BI1, MI27, MI23
- ❑ Epidemic « 027 » strains were clustered in 2 countries (Netherlands and Belgium) and isolated once in Ireland.
- ❑ Non « 027 » toxinotype III isolates were found once in France, Belgium, Spain and Sweden (2 strains)

# Toxinotype III

- ❑ Epidemic «027» strains (n=20):
  - MIC EM > 256 mg/l
  - MIC MX  $\geq$  12 mg/l (except 1 strain with MIC= 6 mg/l)(AIII8)
- ❑ Non «027» toxinotype III strains (n=5) :
  - MIC EM = 0.064->256 mg/l (1 strain R)
  - MIC MX < 2 mg/l
- ❑ Resistance to MX is not specific of the epidemic clone :  
31% of strains different from toxinotype III are resistant to MX



# Toxinotype VIII

- ❑ 34 strains isolated in 6 countries
- ❑ All the strains were A-B+
- ❑ High prevalence in countries:
  - Poland (HII) : 80% (4/5)
  - Poland (HI) : 46.7 % (5/11)
  - Ireland (DI) : 30.7% (4/13)
  - Sweden (MI) : 25% (3/12)
  - Germany (CIII) : 14.2% (7/49)
  - Ireland (DII) : 15.9% (3/19)
- ❑ PCR-ribotyping is under investigation

# Binary toxin

- ❑ Binary toxin was found in 72 strains (17.4% of toxigenic strains)
- ❑ Binary toxin was not detected in non toxigenic strains

<i>Stubbs (Wales):</i>	<i>6.4%</i>
<i>Goncalves (France) :</i>	<i>6%</i>
<i>Rupnik (Asia) :</i>	<i>1.6%</i>
<i>Geric (USA) :</i>	<i>6.1%</i>

# Conclusion

- ❑ So far, the epidemic strain toxinotype III, PCR -ribotype « 027 » has been isolated in 3 countries
- ❑ In 4 hospitals, this clone represents 30% to 70% of all the toxigenic isolates
- ❑ Resistance to MX is not a good screening method for detecting the epidemic clone
- ❑ There is a polymorphism among toxinotype III strains



## The hospital

St. Jansdal hospital in Harderwijk

General hospital, 341 beds

Number of patients admitted in 2004: 25,625

## The laboratory

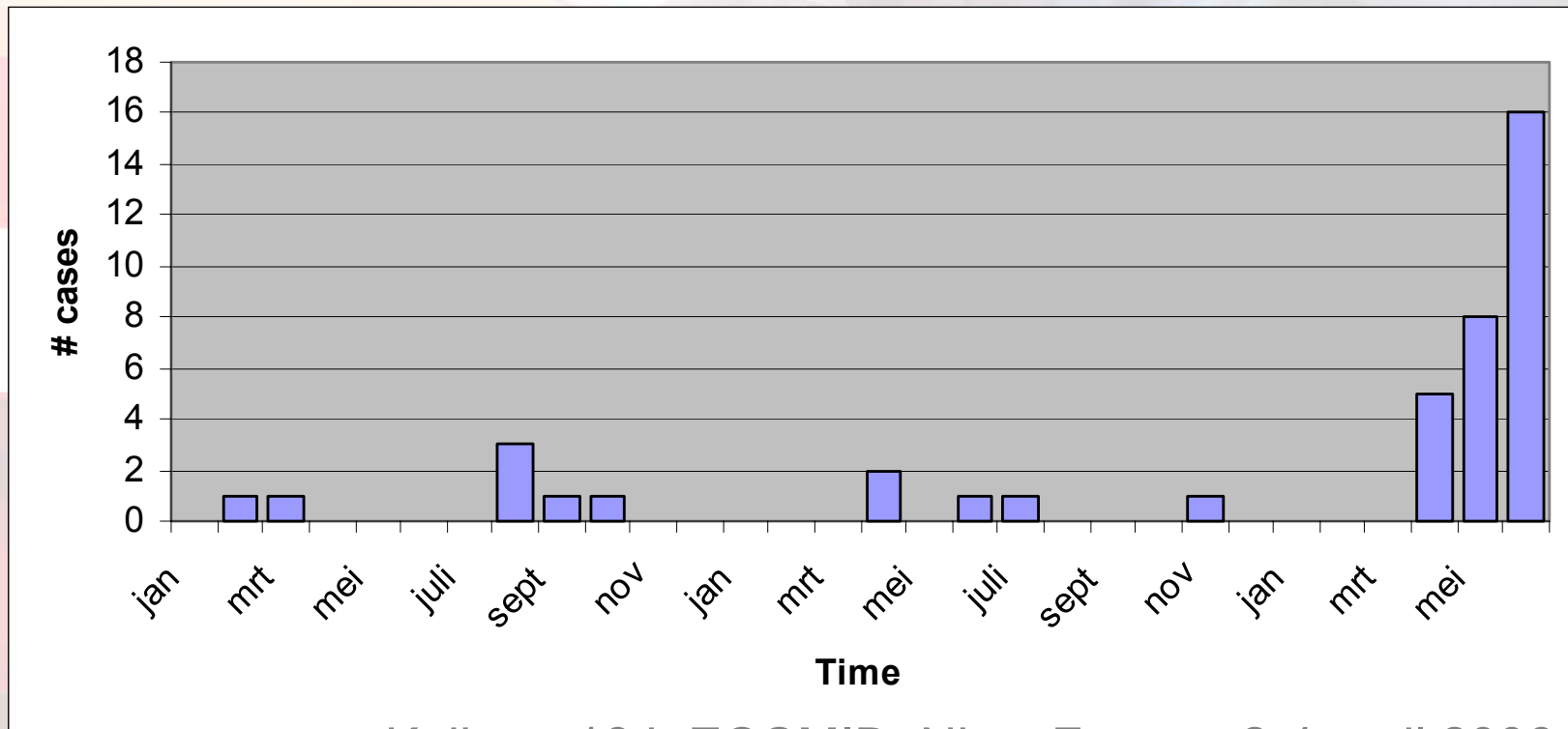
Before march 2005 testing for CDAD referred to Meander Medical Centre, Amersfoort (cytotoxicity test)

March: local introduction of a new diagnostic test in St Jansdal:

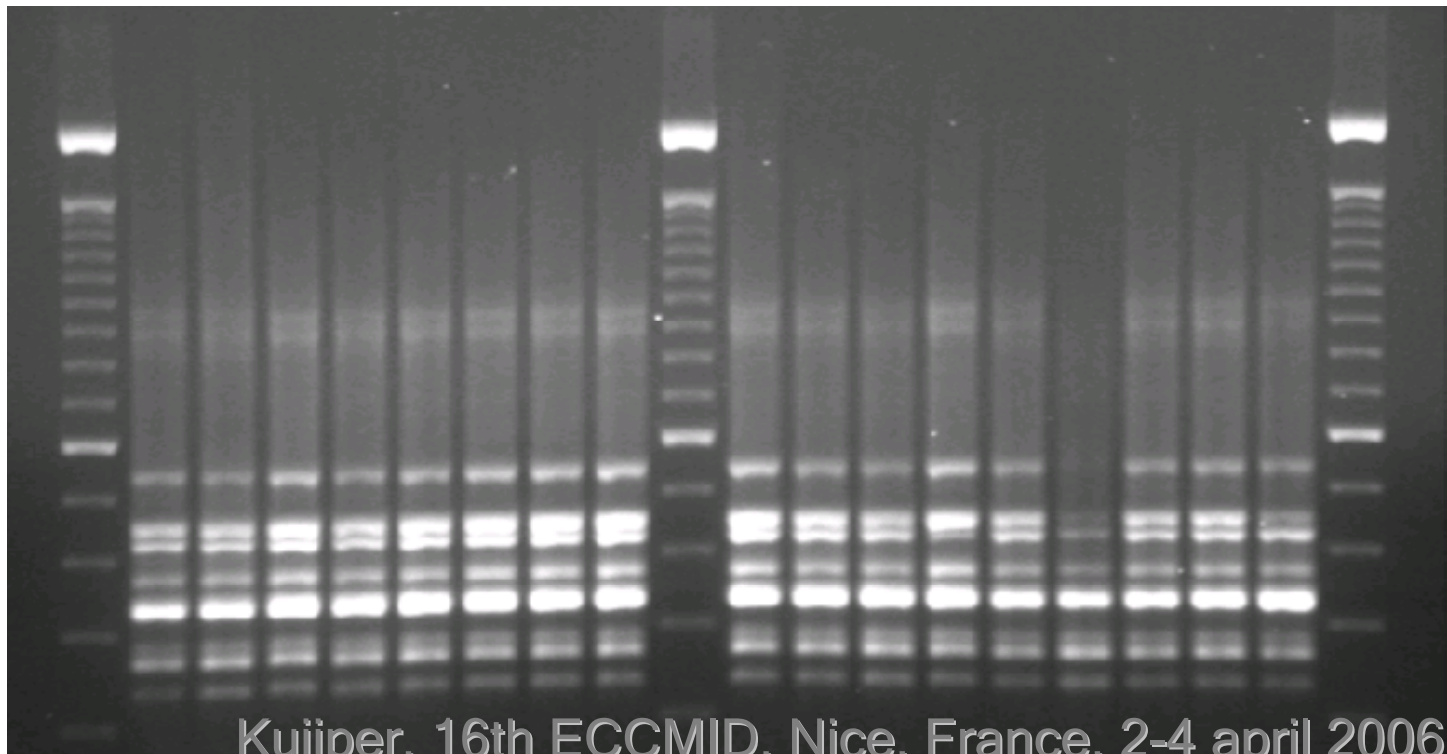
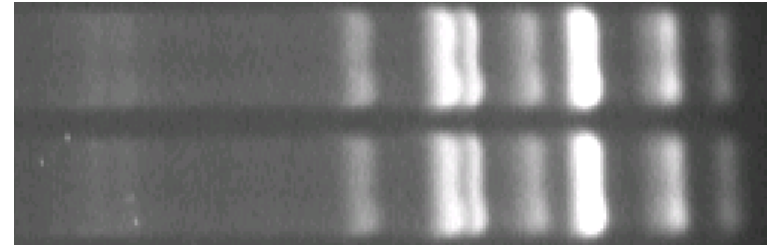
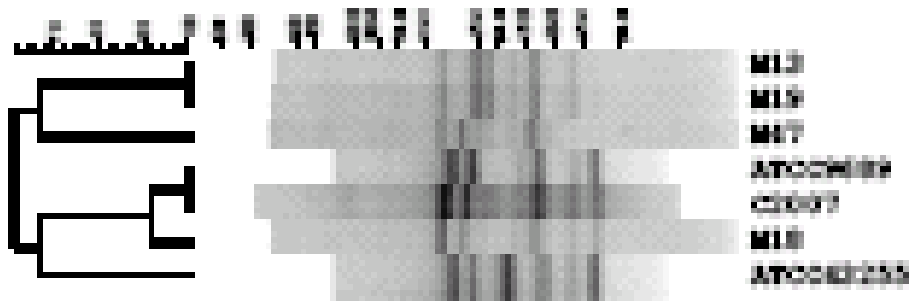
*Rapid enzyme-immuno-assay (ICTAB, Meridian)*



<b>Year</b>	<b># cases</b>	<b>incidence</b>
<b>2003</b>	<b>7</b>	<b>5.4 / 10.000 admissions</b>
<b>2004</b>	<b>5</b>	<b>3.8 / 10.000 admissions</b>
<b>April 2005</b>	<b>5</b>	<b>43 / 10.000 admissions</b>
<b>May 2005</b>	<b>8</b>	<b>79 / 10.000 admissions</b>
<b>June 2005</b>	<b>16</b>	<b>142 / 10.000 admissions</b>



# Ribotyping: 027

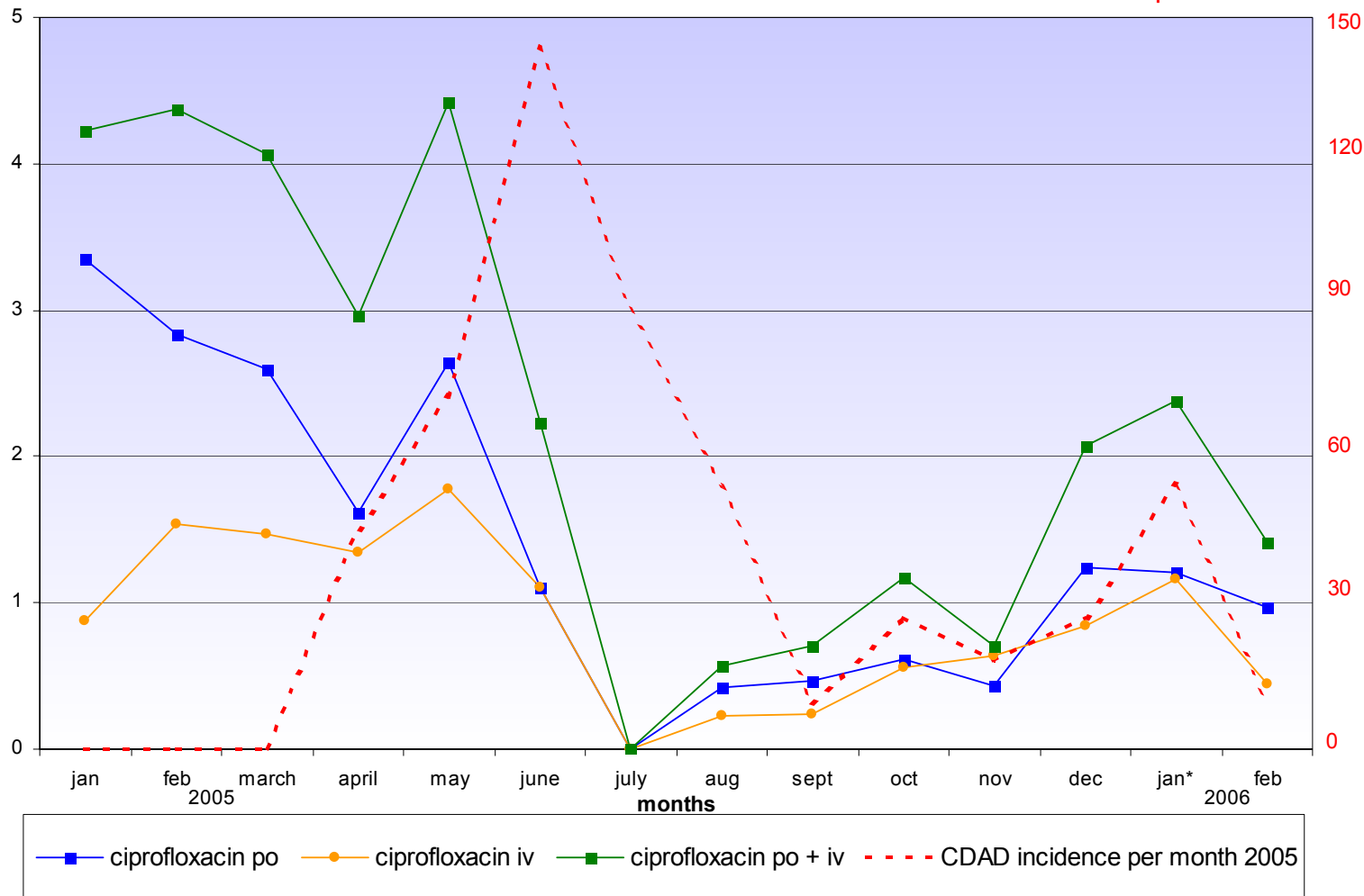


Kuijper, 16th ECCMID, Nice, France, 2-4 april 2006

# Use of ciprofloxacin in St. Jansdal hospital and patients with CDAD

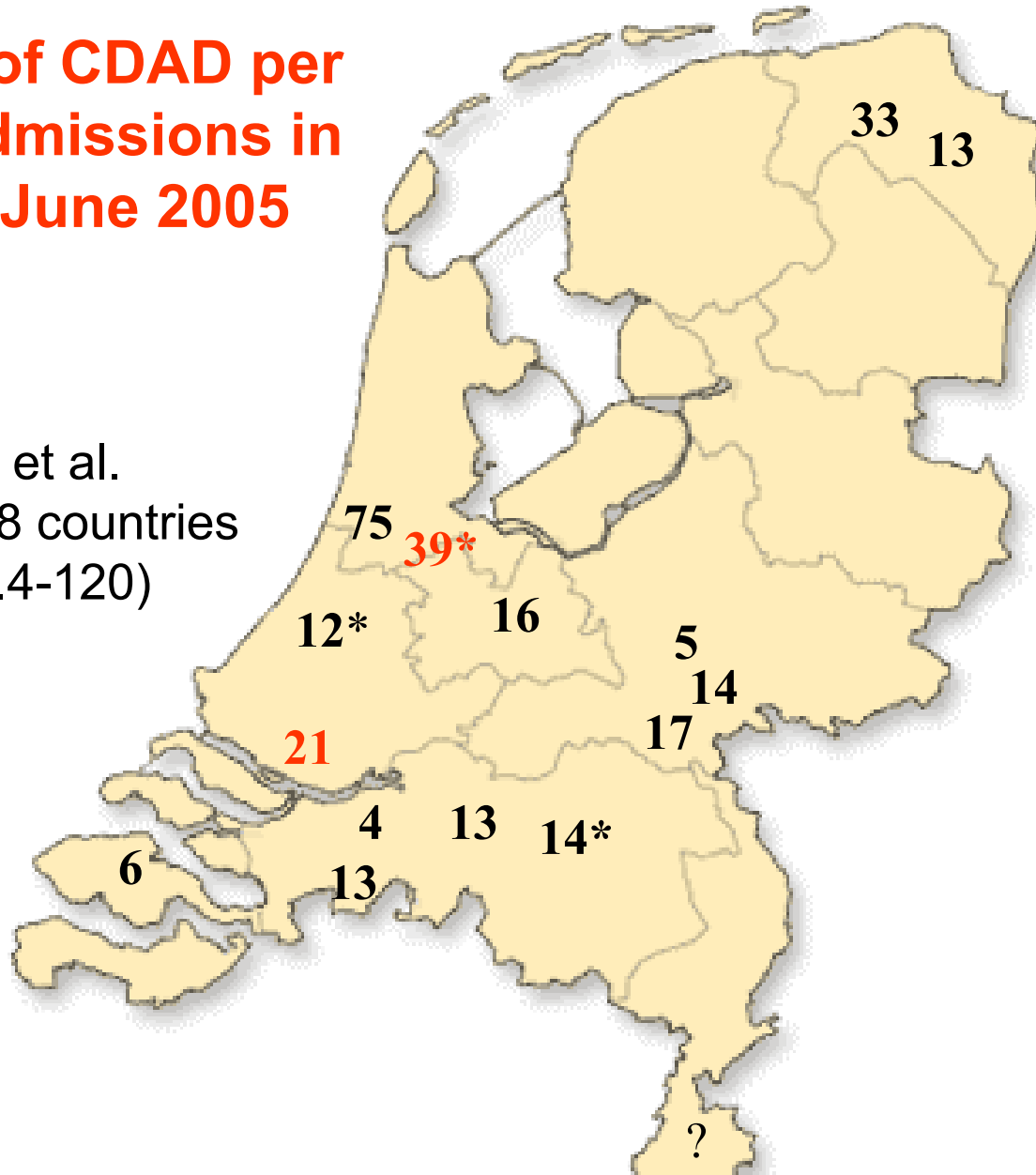
DDD/100 bed-days per month

CDAD incidence  
per 10 000 admissions



## Number of CDAD per 10,000 admissions in May and June 2005

2003, Barbut et al.  
N=112 labs, 8 countries  
11/10,000 (0.4-120)





# Situation at March 29th, 2006



1 isolate of 027



multiple isolates



# Epidemic ended at St. Jansdal:

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Strict hand hygiene with water and soap

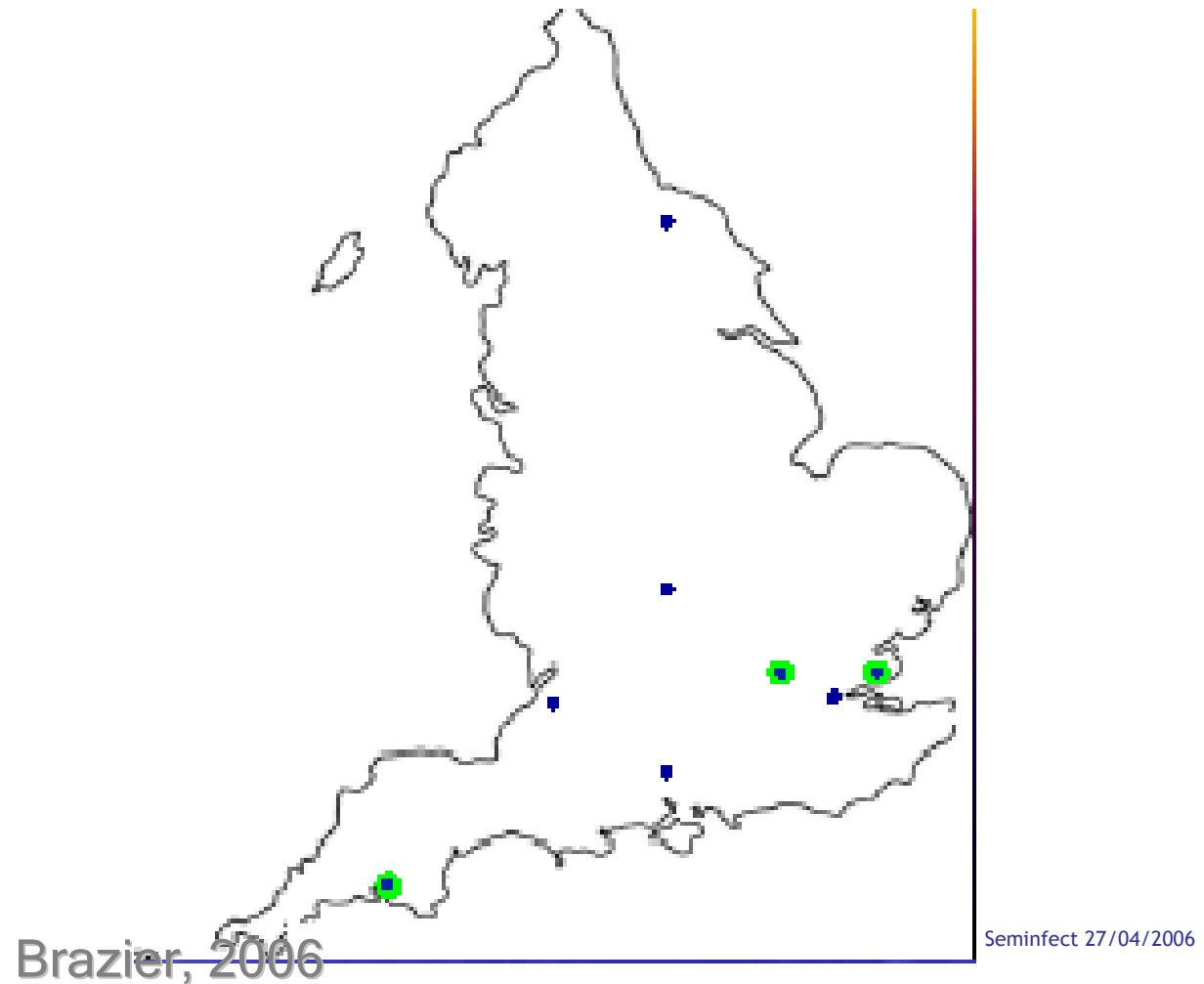
Gloves and apron

Cohorting of patients with CDAD

Complete banning of fluoroquinolones and restriction of cephalosporins

Effective environmental cleaning with chlorine containing disinfectants

# GB : known locations with PCR ribotype 027 (as of January 2006)



# 027 in Belgium

Eurosurveillance 2005;10 (10): 051020 - Microsoft Internet Explorer

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Adresse <http://www.eurosurveillance.org/ViewArticle.aspx?pid=205&tid=205&aid=205>

The full risk assessment, guidance for occupational risk in affected regions and travel advice is available on the ECDC website at <http://www.ecdc.eu.int>.

References:

1. ECDC. Occupational exposure - Current International Guidance on Reducing the Risk of Transmission to humans of Highly Pathogenic Avian Influenza in Birds. 13 October 2005 (<http://www.ecdc.eu.int>)

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**First Isolation of *Clostridium difficile* PCR ribotype 027, toxinotype III in Belgium**

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<sup>1</sup>Jan Yperman Ziekenhuis, Leper, Belgium  
<sup>2</sup>Department of Medical Microbiology, Reference laboratory for *Clostridium difficile*, Leiden University Medical Center, The Netherlands  
<sup>3</sup>Department of Medical Microbiology, Université Catholique de Louvain, Brussels, Belgium

Outbreaks of diarrhoea due to *Clostridium difficile* ribotype 027, toxinotype III have been reported in North America, United Kingdom, and the Netherlands [1-4], and this toxinotype has also been isolated from patients in Belgium. Recently, it has been suggested that the severity of the disease is associated with hyperproduction of toxins A and B by this new variant strain [5].

By 19 September 2005, four patients in the Jan Yperman hospital in Leper, southwest Belgium, had been infected. There was one death due to complications of *C. difficile*-associated diarrhoea and an underlying condition. All patients were female, aged over 70 and had spent longer than 2 weeks in hospital. Two patients were treated with quinolones, a third patient with a beta-lactam antibiotic and the fourth patient, who had a milder form, received no antibiotics at all. In the Jan Yperman hospital, the incidence of *C. difficile*-associated diarrhoea increased from 10 per 10 000 admissions in January - August 2005 to 33 per 10 000 patient admissions in September 2005.

The strain was characterised as PCR ribotype 027 and toxinotype III at the reference laboratory at Leiden University Medical Center. It also contained the binary toxin and had an 18bp deletion in a toxin regulator gene (*tcdC*). As determined by E-tests, the isolates were resistant to ciprofloxacin (MIC=32 mg/l) and susceptible to clindamycin (MIC=2 mg/l) and metronidazole (MIC=0.19 mg/ml). These characteristics are similar as the strain that has been isolated from outbreaks in the United States, Canada, the UK and the Netherlands.

Contact tracing did not reveal the origin of this strain. The hospital has taken additional infection control measures and used the guidelines recently published by Centre for Infectious Disease Control at the National Institute for Public Health and the Environment (RIVM) in Bilthoven (<http://www.rivm.nl>). Subsequently, the Health Inspectorate and the *Clostridium* Reference Centre in Brussels, Belgium, were informed.

**Acknowledgements:**  
Department of Medical Microbiology at the Leids Universitair Medisch Centrum (Leiden University Medical Center).

References:

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2. Outbreak of *Clostridium difficile* in a hospital in south east England. *COT weekly* 2005;15(24): news. (<http://www.hpa.org.uk/cot/archives/archive05/News/news2405.htm>)
3. McDonald C. *Clostridium difficile*: responding to a new threat from an old enemy. *Infect Control and Epidemiol*. 2005;26:672-5.
4. Kuijper EJ, Debast SB, van Kreghen P, Notermans OW, van den Broek PJ. *Clostridium difficile* ribotype 027, toxinotype III in the Netherlands. *Ned Tijdschr Geneesk*. 2005; 49:2087-9.
5. Warny H, Pepin J, Fang A, Karpman G, Thompson A, Brazer J, Frost E. McDonald Toxin production by an emerging strain of *Clostridium difficile* associated with outbreaks of severe disease in North America and Europe. *Lancet*. 2005;366:1079-84.

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**Internationally adopted children as a source for MRSA**

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<sup>1</sup>Department of medical microbiology, St. Olavs University Hospital, Trondheim, Norway  
<sup>2</sup>Department of Laboratory Medicine, Children's and Women's Health, Norwegian University of Science and Technology, Trondheim, Norway

We describe the import of methicillin-resistant *Staphylococcus aureus* (MRSA) to Norway by three internationally adopted children from Hunan province in south central China. The children had been living in different children's homes in Hunan, and came to times between January and August 2004. They had no contact with one another, either in Norway or, to our knowledge, in China. The first child, living with her adoptive family in northern Norway, was found to be carrying MRSA in a minor lesion on her abdomen, her adoptive mother had to undergo a surgical revision of an abscess in the leg, and MRSA was grown from this. On screening, the adoptive father was also found to be MRSA positive. Both parents are healthcare workers. Repeated eradication unsuccessful in all three patients, and the parents are on sick leave from work due to MRSA carriage.

The adoptive mother of the second girl, living in southern Norway, became pregnant after the adoption. She gave birth to a premature child from whom MRSA was cultivated in pus from the eye. The mother, the adopted child, and two other newborns and a nurse from the premature baby ward were screened and found to be colonised with the same MRSA strain. The third child, living in western Norway, was found to be MRSA positive in a minor lesion on her chin. Her mother is a nurse in the intensive care unit at the local hospital and has been colonised with MRSA.

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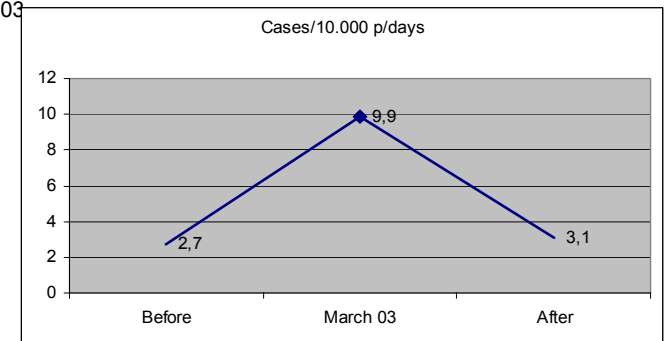
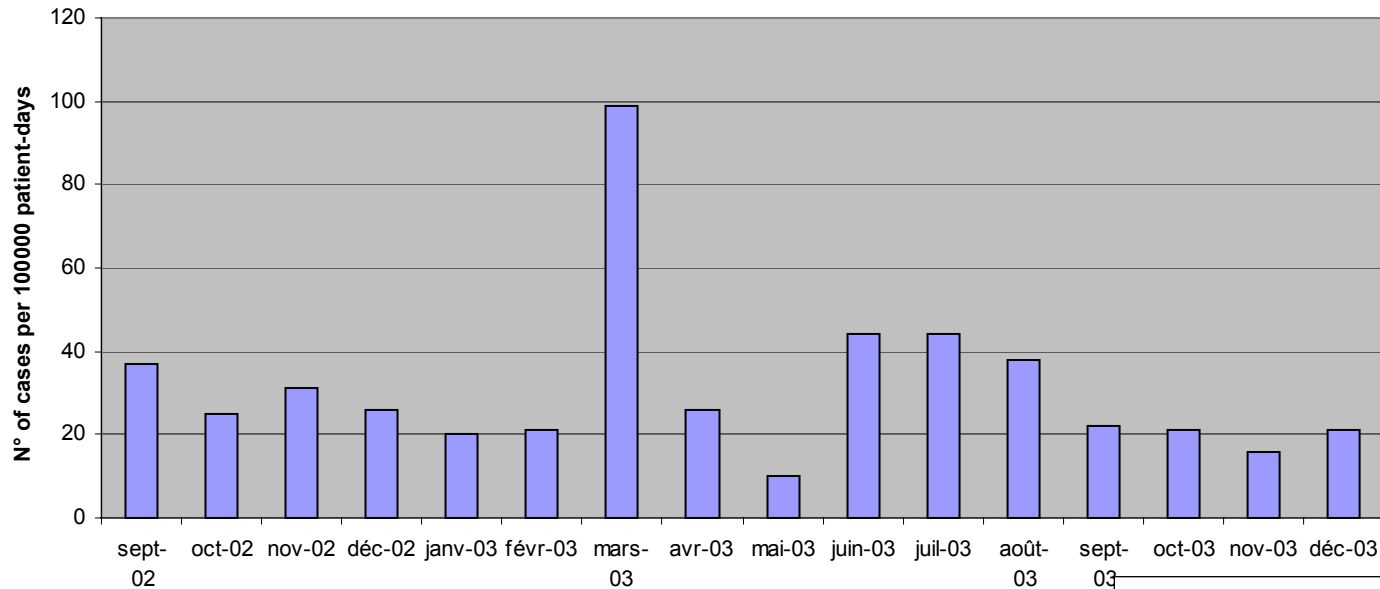
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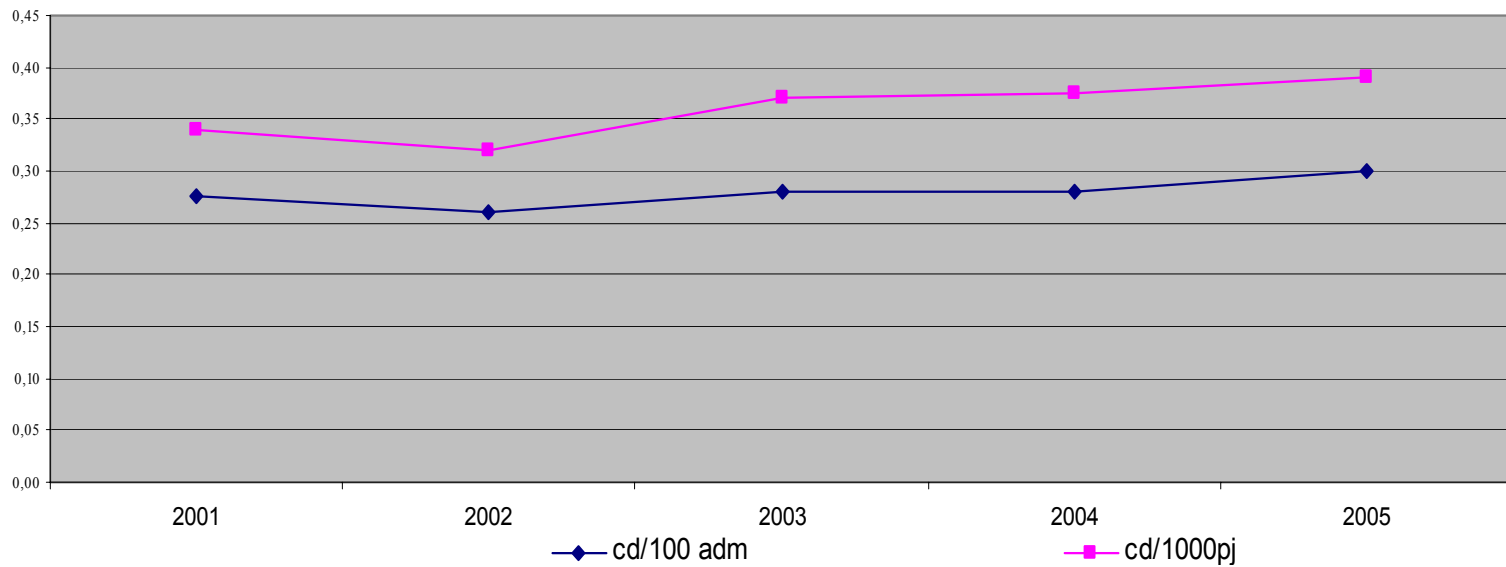
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# 027 in Belgium

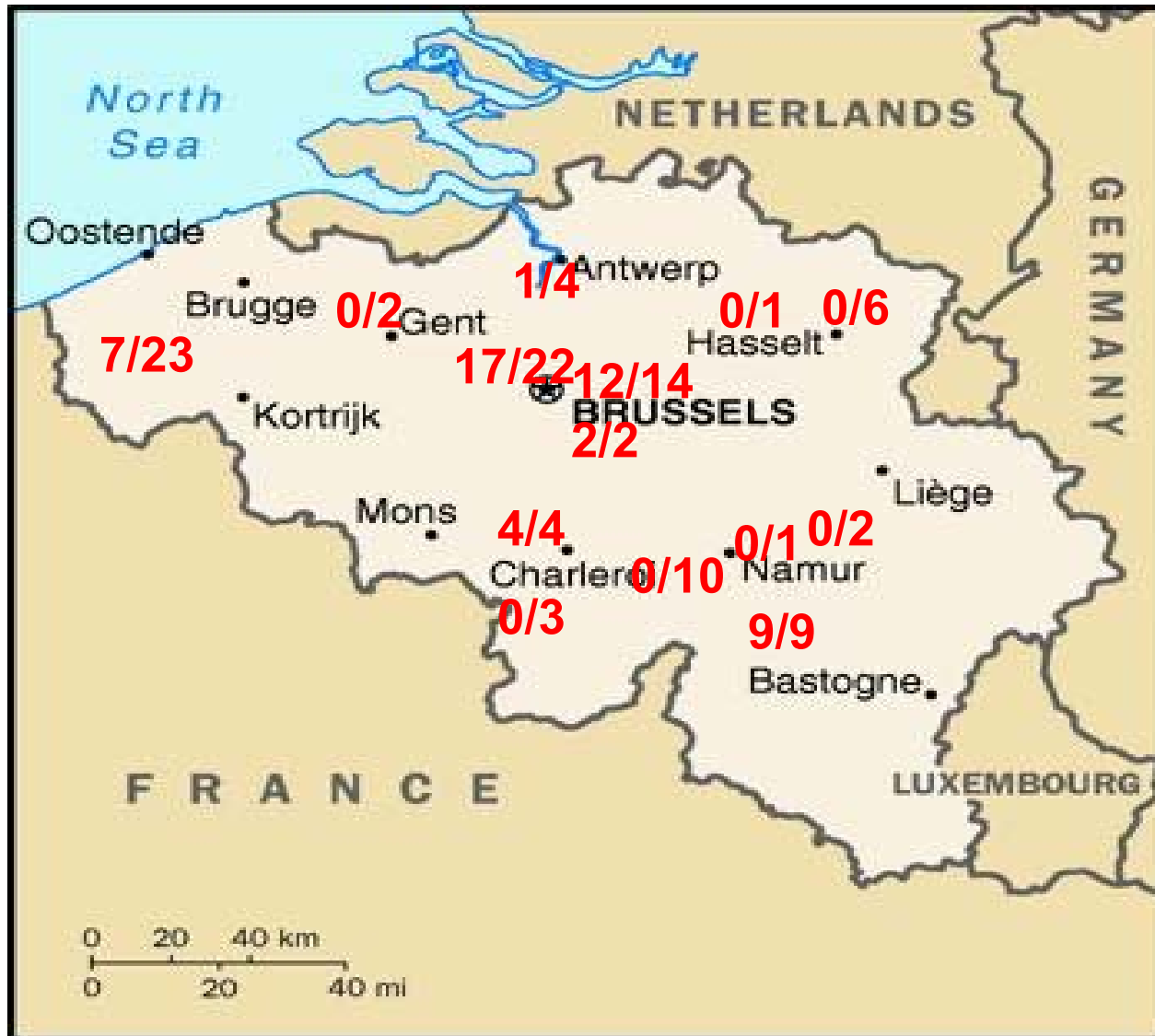
Incidence of positive *Clostridium difficile* toxine per 100000 patient-days



# Densité d'incidence de *Clostridium difficile*



# Belgium ref center 2006



n 027/  
total

52/103  
(50.5%)

# 027 : what to do ?

- ☐ Diagnosis
- ☐ Surveillance
- ☐ Treatment
- ☐ Prevention