

## Role of Hospital Surfaces in the Spread of Emerging HA Pathogens: *C. difficile*, Norovirus, *Acinetobacter*

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Disclosure: ASP and Clorox

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## Role of Surfaces in Spread of HA Pathogens

- Factors that facilitate environmental transmission
  - *Acinetobacter*
  - Norovirus
  - *C. difficile*

## The Role of the Environment in Disease Transmission

- Over the past decade there has been a growing appreciation that environmental contamination makes a contribution to HAI with MRSA, VRE, *Acinetobacter*, norovirus, and *C. difficile*
- Surface disinfection practices are currently not effective in eliminating environmental contamination
- Some organisms have better ability to persist in the inanimate environment than others (e.g. *C. difficile*, MRSA, VRE, *Acinetobacter*)
- Inadequate terminal cleaning of rooms occupied by patients with MDR pathogens places the next patients in these rooms at increased risk of acquiring these organisms

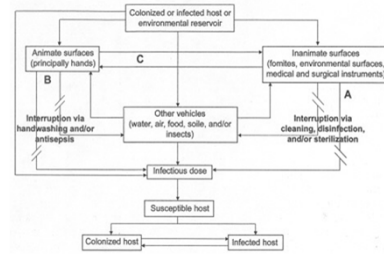
## KEY PATHOGENS WHERE ENVIRONMENTAL SURFACES PLAY A ROLE IN TRANSMISSION

- MRSA
- VRE
- *Clostridium difficile*
- *Acinetobacter* spp.
- Norovirus
- Rotavirus
- SARS

## FACTORS THAT FACILITATE ENVIRONMENTAL TRANSMISSION

- Survive in the environment for days to weeks to months
- Frequently contaminate the environmental surfaces in rooms of colonized or infected patients
- Transiently colonize the hands of healthcare personnel
- Transmitted by healthcare personnel
- Cause outbreaks in which environmental transmission was deemed to play a role
- Improved surface cleaning/disinfection reduces disease incidence
- Admission to a room previously occupied by a patient with the pathogen of interest is a risk factor for disease for the newly admitted patient to develop colonization/infection

## TRANSMISSION MECHANISMS INVOLVING THE SURFACE ENVIRONMENT



Rutala WA, Weber DJ. In: "SHEA Practical Healthcare Epidemiology" (Lautenbach E, Woeltje KF, Malani PN, eds), 3<sup>rd</sup> ed, 2010.

## KEY PATHOGENS WHERE ENVIRONMENTAL SURFACES PLAY A ROLE IN TRANSMISSION

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## ENVIRONMENTAL SURVIVAL OF KEY PATHOGENS

Pathogen	Survival	Environmental Data
MRSA	Days to weeks	2-3+
VRE	Days to weeks	3+
<i>Acinetobacter</i>	Days to weeks	2-3+
<i>C. difficile</i>	Months (spores)	3+
Norovirus	Days to weeks	3+

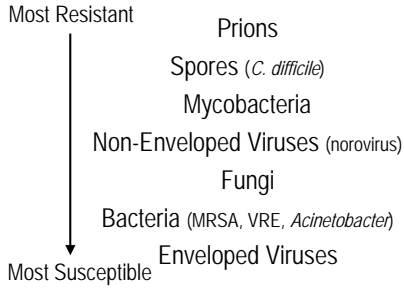
Adapted from Hota B, et al. Clin Infect Dis 2004;39:1182-9 and Kramer A, et al. BMC Infectious Diseases 2006;6:130



## Environmental Surface Disinfection

### Product and Practice

### Decreasing Order of Resistance of Microorganisms to Disinfectants/Sterilants



### Proving that Environmental Contamination Leads to Nosocomial Infections

- Demonstration of microbial persistence in the environment: *In vitro* studies and environmental samples
- Demonstration of frequent environmental contamination
- Demonstration of HCW hand contamination
- Relationship between level of environmental contamination and hand contamination
- Demonstration of person-to-person transmission (molecular link)
- Demonstration that being housed in a room previously occupied by a patient with the pathogen of interest is a risk factor for disease
- Demonstration that improved surface cleaning/disinfection reduces disease incidence

### DAZO Solution (AKA – Goo)



### Thoroughness of Environmental Cleaning Carling and coworkers, SHEA 2010

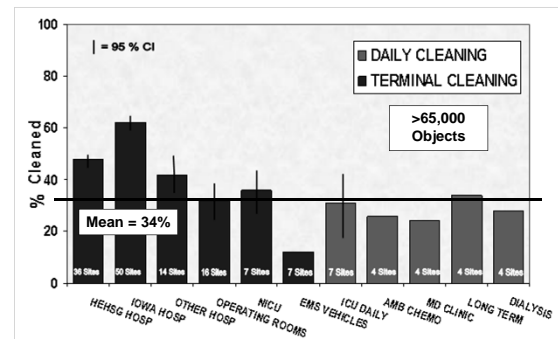


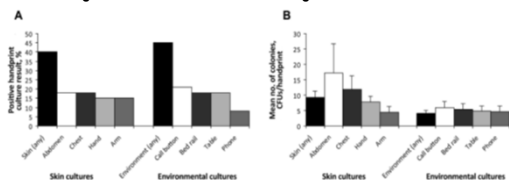
TABLE. Rates of Cleaning for 14 Types of High-Risk Objects

Object	Percentage cleaned		95% CI
	Mean ± SD	Range	
Sink	82 ± 12	57-97	77-88
Toilet seat	76 ± 18	40-98	68-84
Tray table	77 ± 15	53-100	71-84
Bedside table	64 ± 22	23-100	54-73
Toilet handle	60 ± 22	23-89	50-69
Side rail	60 ± 21	25-96	51-69
Call box	50 ± 19	9-90	42-58
Telephone	49 ± 16	18-86	42-56
Chair	48 ± 28	11-100	35-61
Toilet door knobs	28 ± 22	0-82	18-37
Toilet hand hold	28 ± 23	0-90	18-38
Bedpan cleaner	25 ± 18	0-79	17-33
Room door knobs	23 ± 19	2-73	15-31
Bathroom light switch	20 ± 21	0-81	11-30

NOTE. CI, confidence interval.

### FREQUENCY OF ACQUISITION OF MRSA ON GLOVED HANDS AFTER CONTACT WITH SKIN AND ENVIRONMENTAL SITES

No significant difference on contamination rates of gloved hands after contact with skin or environmental surfaces (40% vs 45%; p=0.59), that is, touching the environment results in hand/glove contamination



Stiefel U, et al. ICHE 2011;32:185-187

### Risk of Acquiring MRSA, VRE, and *C. difficile* from Prior Room Occupants

- Admission to a room previously occupied by an MRSA-positive patient or VRE-positive patient significantly increased the odds of acquisition for MRSA and VRE (although this route is a minor contributor to overall transmission). Huang et al. Arch Intern Med 2006;166:1945.
- Prior environmental contamination, whether measured via environmental cultures or prior room occupancy by VRE-colonized patients, increases the risk of acquisition of VRE. Drees et al. Clin Infect Dis 2008;46:678.
- Prior room occupant with CDAD is a significant risk for CDAD acquisition. Shaughnessy et al. ICH 2011;32:201

### Reduction in Acquisition of VRE after Enforcement of Routine Environmental Cleaning Measures

Hayden et al. Clin Infect Dis 2006;42:1552

- Acquisition of VRE decreased significantly after baseline and remained stable; when environmental cleaning was significantly improved in periods 2-4. Adherence to hand hygiene increased in period 2 but high adherence did not persist and lowest in period 3. Thus, environmental cleaning was considered to be the sole intervention.

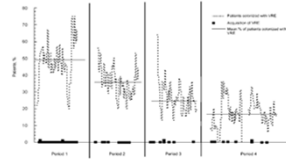


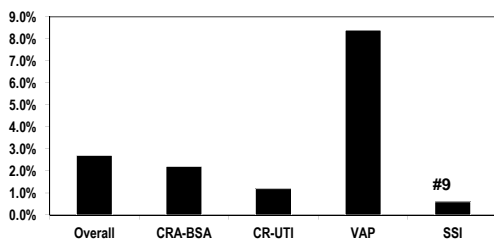
Figure 1. Daily percentage of patients colonized with vancomycin-resistant enterococcus (VRE), daily acquisition of rectal colonization with VRE, and mean percentage of patients colonized with VRE by period. Period 1 was a baseline period (5 March-1 May 2001); duration 58 days. Period 2 included environmental hygiene intervention (21 May-27 July 2001; duration 58 days). Period 3 was a "washout" period in which there was no intervention (23 August-30 October 2001; duration 57 days). Period 4 included hand hygiene intervention (8 November-1 February 2002; duration 52 days).

## Acinetobacter

### ACINETOBACTER AS A HOSPITAL PATHOGEN

- Gram-negative aerobic bacillus
- Common nosocomial pathogen; outbreaks of MDR *Acinetobacter*
- Pathogenic: High attributable mortality (Falagas M, et al. Crit Care 2007;11:134)
  - Hospitalized patients: 8-23%
  - ICU patients: 10-43%
- Ubiquitous in nature and hospital environment
  - Found on healthy human skin
  - Found in the environment
- Survives in the environment for a prolonged period of time
- Often multidrug resistant

### Prevalence of *Acinetobacter* in Device-Related HAIs, NHSN, 2006-2007



### *Acinetobacter* Contamination of the Environment

- *Acinetobacter* isolated from curtains, slings, patient-lift equipment, door handles, and computer keyboards (Wilks et al. ICH 2006;27:654)
- *A. baumannii* isolated from 3% of 252 environmental samples: 2/6 stethoscopes, 1/12 patient records, 4/23 curtains, 1/23 OR lights (Young et al. ICH 2007;28:1247)
- *A. baumannii* isolated from 41.4% of 70 environmental cultures: 9 headboards, 2 foot of bed, 6 resident desks, 8 external surface ET tube (Markogiannakis et al. ICH 2008;29:410)
- *Acinetobacter* isolated from environmental surfaces on 2 occasions (Shelburne et al. J Clin Microbiol 2008;46:198)
- *A. baumannii* isolated from 21 environmental samples: 4 ventilator surfaces, 4 bedside curtains, 1 bed rail (Chang et al. ICH 2009;30:34)
- CRAB-isolated from 24/135 (17.9%) environmental samples and 7/65 (10.9%) of HCWs; genetically related (Choi et al. JKMS 2010;25:999)



## The Discovery of Norwalk Virus

Dr. Al Kapikian, NIH



EM Scope used to discover Norwalk virus in 1972

## NOROVIRUS: MICROBIOLOGY AND EPIDEMIOLOGY

- Classified as a calicivirus: RNA virus, non-enveloped
- Prevalence
  - Causes an estimated 23 million infections per year in the US
  - Results in 50,000 hospitalizations per year (310 fatalities)
  - Accounts for >90% of nonbacterial and ~50% of all-cause epidemic gastroenteritis
- Infectious dose: 10-100 viruses (ID<sub>50</sub> = 18 viruses)
- Fecal-oral transmission (shedding for up to 2-3 weeks)
  - Direct contact and via fomites/surfaces; food and water
- Droplet transmission? (via ingestion of airborne droplets of virus-containing particles)
- HA outbreaks involve patients and staff with high attack rates

## Factors Leading to Environmental Transmission of Norovirus

- Stable in the environment
- Low inoculating dose
- Frequent contamination of the environment
- Susceptible population (limited immunity)
- Relatively resistant to disinfectants
- Common source of infectious gastroenteritis

## Primary Modes of Transmission of Norovirus Outbreaks England & Wales 1992-2000

Setting of Outbreak	Foodborne*	Person-to-Person*	Other / Unknown*	No*
Hospital	10 (1.3)	716 (95.0)	28 (3.7)	754
Residential facilities	33 (4.5)	658 (91.0)	32 (4.4)	723
School	4 (5.5)	65 (89.0)	4 (5.5)	73
Food Outlet	70 (66.7)	23 (21.9)	12 (11.4)	105
Hotel	42 (28.6)	94 (63.9)	11 (7.5)	147
Other	25 (33.8)	43 (58.1)	6 (8.1)	74
<b>Total</b>	<b>184 (9.9)</b>	<b>1599 (85.2)</b>	<b>93 (5.0)</b>	<b>1877</b>

\* number of outbreaks (% of all outbreaks in setting)

## Hospital Outbreaks

- Attack rate: 62% (13/21) for patients and 46% (16/35) for staff (Green et al. J Hosp Infect 1998;39:39)
- Number ill: 77 persons (28 patients and 49 staff) (Leuenberger et al. Swiss Med Weekly 2007;137:57)
- Attack rate: 21% (20 of 92) of all patients admitted to the pediatric oncology unit (Simon et al. Scand J Gastro 2006;41:693)
- Attack rate: 75% (3 of 4) of patients and 26% (10 of 38) staff (Weber et al. ICHE 2005;26:841)

## Environmental Contamination

- Hospital-11/36 (31%) environmental swabs were positive by RT-PCR. Positive swabs were from lockers, curtains and commodes and confined to the immediate environment of symptomatic patients (Green et al. J Hosp Infect 1998;39:39)
- Rehabilitation Center-Norovirus detected from patients and three environmental specimens (physiotherapy instrument handle, toilet seat [2-room of symptomatic guest, public toilet]) RT-PCR (Kuusi et al. Epid Infect 2002;129:133-138)
- LTCF-5/10 (50%) of the environmental samples were positive for norovirus by RT-PCR (Wu et al. ICHE 2005;26:802)

## Environmental Survival

- At 20°C a 9-log<sub>10</sub> reduction of FCV between 21-28 days in a dried state (Douttree et al. J Hosp Infect 1999;41:51)
- HuNV was detected by RT-PCR on stainless steel, ceramic, and formica surfaces for 7 days (D'Souza D et al. Int J Food Microbiol 2006;108:84-91)
- MNV survived more than 40 days on diaper material, on gauze, and in a stool suspension (JungEun L et al. Appl Environ Microbiol 2008;74:2111-17)
- FCV can survive up to 3 days on telephone buttons and receivers, 1-2 days on a computer mouse, and 8-12 hours on a keyboard (Clay S et al. AJIC 2006;34:41-3)  
FCV, feline calicivirus; HuNV, human norovirus; MNV, mouse norovirus

## Role of the Environment

1. Prolonged outbreaks on ships suggest norovirus survives well
2. Outbreak of GE affected more than 300 people who attended a concert hall over a 5-day period. Norwalk-like virus (NLV) confirmed in fecal samples by RT-PCR. The index case was a concert attendee who vomited in the auditorium. GI illness occurred among members of 8/15 school parties who attended the following day. Disinfection procedure was poor. Evans et al. Epid Infect 2002;129:355
3. Extensive environmental contamination of hospital wards  
Studies suggest transmission most likely occurred through direct contact with contaminated fomites.

## Surface Disinfection

- School outbreak of NLV-cleaning with QUAT preparations made no impact on the course of the outbreak. The outbreak stopped after the school closed for 4 days and was cleaned using chlorine-based agents (Marks et al. Epid Inf 2003;131:727)
- Detergent-based cleaning to produce a visibly clean surface consistently failed to eliminate norovirus contamination. A hypochlorite/detergent formulation of 5,000 ppm chlorine was sufficient to decontaminate surfaces. (Barker et al. J Hosp Infect 2004;58:42)

## Inactivation of Murine and Human Norovirus

Disinfectant, 1 min	MNV Log <sub>10</sub> Reduction	HNV Log <sub>10</sub> Reduction
70% Ethanol	>4 (3.3 at 15sec)	2
70% Isopropyl alcohol	4.2	2.2
65% Ethanol + QUAT	>2	3.6
79% Ethanol + QUAT	3.4	3.6
Chlorine (5,000ppm)	4	3
Chlorine (24,000ppm)	2.4	4.3
Phenolic, QUAT, Ag, 3% H <sub>2</sub> O <sub>2</sub>	≤1	≤1 (2.1 QUAT)
0.5% Accel H <sub>2</sub> O <sub>2</sub>	3.9	2.8

Rutala WA, Folan MP, Tallon LA, Lyman WH, Park GW, Sobsey MD, Weber DJ. 2007

## Inactivation of Murine and Human Norovirus

Antiseptic, 1 min	MNV Log <sub>10</sub> Reduction	HNV Log <sub>10</sub> Reduction
Ethanol Hand Spray	3.2	0.4
Ethanol Based Rub	1.9	2.1
Iodophor (10%)	0.8	0.5
4% CHG	0.1	0.3
0.5% Triclosan	1.3	0.2
1% PCMX	0	2.4

## Guideline for the Prevention of Norovirus Outbreaks in Healthcare, HICPAC, 2011

- **Avoid exposure to vomitus or diarrhea. Place patients with suspected norovirus on Contact Precautions in a single room (IB)**
  - Continue Precautions for at least 48 hours after symptom resolution (IB)
  - Use longer isolation times for patients with comorbidities (II) or <2 yrs (II)
- **Consider minimizing patient movements within a ward (II)**
  - Consider restricting movement outside the involved ward unless essential (II)
  - Consider closure of wards to new admissions (II)
- **Exclude ill personnel (IB)**
- **During outbreaks, use soap and water for hand hygiene (IB)**
- **Clean and disinfect patient care areas and frequently touched surfaces during outbreaks 3x daily using EPA approved healthcare product (IB)**
- **Clean surfaces and patient equipment prior to disinfection. Use product with an EPA approved claim against norovirus (IC)**

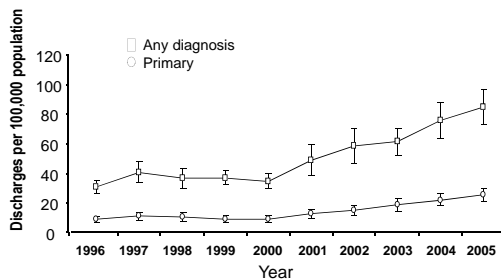
MacCannell T, et al. <http://www.cdc.gov/hicpac/pdf/norovirus/Norovirus-Guideline-2011.pdf>

# *C. difficile*

## *C. difficile*: Microbiology and Epidemiology

- Gram-positive bacillus: Strict anaerobe, spore-former
- Colonizes human GI tract
- Increasing prevalence and incidence
- New epidemic strain that hyperproduces toxins A and B
- Introduction of CDI from the community into hospitals
- High morbidity and mortality in elderly
- Inability to effectively treat fulminant CDI
- Absence of a treatment that will prevent recurrence of CDAD
- Inability to prevent CDAD

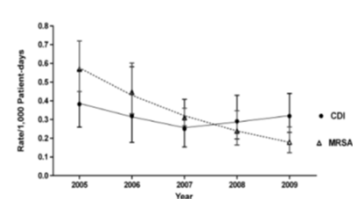
## Rates of *C. difficile* Disease in U.S. Hospitals, 2000 - 2005



From McDonald LC, et al. *Emerg Infect Dis.* 2006;12(3):409-15 and unpublished CDC data

## CDI Now the Most Common Healthcare-Associated Pathogen

- Analysis of 10 community hospitals, 2005-2009, in the Duke DICON system



Miller BA, et al. *ICHE* 2011;32:387-390

## Factors Leading to Environmental Transmission of *C. difficile*

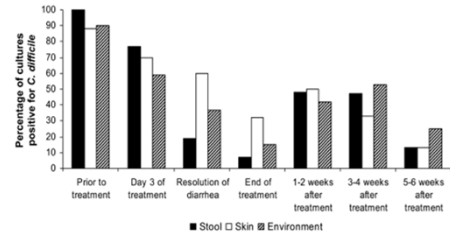
- Stable in the environment
- Low inoculating dose
- Common source of infectious gastroenteritis
- Frequent contamination of the environment
- Susceptible population (limited immunity)
- Relatively resistant to disinfectants

## Environmental Contamination

- 25% (117/466) of cultures positive (<10 CFU) for *C. difficile*. >90% of sites positive with incontinent patients. (Samore et al. *AJM* 1996;100:32)
- 31.4% of environmental cultures positive for *C. difficile*. (Kaatz et al. *AJE* 1988;127:1289)
- 9.3% (85/910) of environmental cultures positive (floors, toilets, toilet seats) for *C. difficile*. (Kim et al. *JID* 1981;143:42)
- 29% (62/216) environmental samples were positive for *C. difficile*. 29% (11/38) positive cultures in rooms occupied by asymptomatic patients and 49% (44/90) in rooms with patients who had CDAD. (*NEJM* 1989;320:204)
- 10% (110/1086) environmental samples were positive for *C. difficile* in case-associated areas and 2.5% (14/489) in areas with no known cases. (Fekety et al. *AJM* 1981;70:907)



## PERCENT OF STOOL, SKIN, AND ENVIRONMENT CULTURES POSITIVE FOR *C. difficile*



Skin (chest and abdomen) and environment (bed rail, bedside table, call button, toilet seat)  
Sethi AK, et al. ICHE 2010;31:21-27

## FREQUENCY OF ENVIRONMENTAL CONTAMINATION AND RELATION TO HAND CONTAMINATION

- Study design: Prospective study, 1992
- Setting: Tertiary care hospital
- Methods: All patients with CDI assessed with environmental cultures
- Results
  - Environmental contamination frequently found (25% of sites) but higher if patients incontinent (>90%)
  - Level of contamination low (<10 colonies per plate)
  - Also contaminated: BP cuff, electronic thermometer, IV accurate control device and oximeter
  - ↑ hand cont ↑ surface cont

Site	All Rooms		Double Rooms	
	No. Positive/No. Tested (%)	Index Site (%)	No. Positive/No. Tested (%)	Roommate Site (%)
Floor	15/71 (48)	NA	NA	NA
Commodes	1/17 (4)	NA	NA	NA
Window sill	6/16 (38)	NA	NA	NA
Tablet	11/45 (24)	NA	NA	NA
Buzzer	11/57 (19)	6/19 (32)	1/17 (6)	1/17 (6)
Bedposts	11/56 (21)	4/22 (18)	2/14 (14)	2/14 (14)
Beetles	10/81 (12)	7/26 (27)	2/25 (8)	2/25 (8)
Total	81/303 (27)	17/65 (26)	5/96 (5)	5/96 (5)

NA = not applicable.

Environmental Sites Positive (%)	No. of Index Cases With Environmental Sites and Personnel Cultured (%)	No. of Positive Personnel/Personnel Cultured (%)
0	12	0/25
1-25	5	0/11
26-50	5	1/12 (8)
>50	6	9/25 (36)

Chi-square test for linear trend in proportions: P < 0.01.

Samore MH, et al. Am J Med 1996;100:32-40

## PERSISTENCE OF CLINICALLY RELEVANT BACTERIA ON DRY INANIMATE SURFACES

Type of bacterium	Duration of persistence (range)
<i>Acinetobacter</i> spp.	3 days to 5 months
<i>Bordetella pertussis</i>	3 - 5 days
<i>Campylobacter jejuni</i>	up to 6 days
<i>Clostridium difficile</i> (various)	7 months
<i>Escherichia coli</i> (various)	up to 13 months
<i>Staphylococcus aureus</i>	13 days
<i>Corynebacterium diptheriae</i>	2 days - 6 months
<i>Corynebacterium parvulosporosus</i>	1-8 days
<i>Escherichia coli</i>	1.5 hours - 16 months
<i>Enterococcus</i> spp. including VRE and VSE	5 days - 4 months
<i>Haemophilus influenzae</i>	12 days
<i>Helicobacter pylori</i>	2-90 minutes
<i>Klebsiella</i> spp.	2 hours to > 30 months
<i>Listeria</i> spp.	1 day - months
<i>Mycobacterium bovis</i>	> 2 months
<i>Mycobacterium tuberculosis</i>	1 day - 4 months
<i>Neisseria gonorrhoeae</i>	1 - 3 days
<i>Proteus vulgaris</i>	1 - 2 days
<i>Pseudomonas aeruginosa</i>	4 hours - 16 months; on dry floor: 5 weeks
<i>Salmonella</i> spp.	6 hours - 4 weeks
<i>Salmonella typhimurium</i>	10 days - 4.2 years
<i>Salmonella</i> spp.	1 day
<i>Serratia marcescens</i>	3 days - 2 months; on dry floor: 5 weeks
<i>Shigella</i> spp.	2 days - 3 months
<i>Staphylococcus aureus</i> , including MRSA	7 days - 7 months
<i>Streptococcus pneumoniae</i>	1 - 20 days
<i>Streptococcus pyogenes</i>	3 days - 4.5 months
<i>Vibrio cholerae</i>	1 - 7 days

Kramer A, et al. BMC Infect Dis 2006;6:130

## EVALUATION OF HOSPITAL ROOM ASSIGNMENT AND ACQUISITION OF CDI

- Study design: Retrospective cohort analysis, 2005-2006
- Setting: Medical ICU at a tertiary care hospital
- Methods: All patients evaluated for diagnosis of CDI 48 hours after ICU admission and within 30 days after ICU discharge
- Results (acquisition of CDI)
  - Admission to room previously occupied by CDI = 11.0%
  - Admission to room not previously occupied by CDI = 4.6% (p=0.002)

TABLE 3. Multivariate Analysis of Risk Factors for Acquisition of *Clostridium difficile* Infection (CDI)

Risk factor	HR (95% CI)	P
Prior room occupied with CDI	2.57 (1.21-4.54)	.01
Gender age	1.00 (reference)	—
Higher APACHE II score	1.00 (1.00-1.01)	.06
Proton pump inhibitor use	1.11 (0.44-2.78)	.83
Antibiotic exposure	—	—
Norfloxacin	0.38 (0.05-2.72)	.33
Levofloxacin	1.08 (0.67-1.73)	.75
Ciprofloxacin	0.49 (0.15-1.67)	.23
Fluoroquinolones	1.17 (0.72-1.91)	.53
Clindamycin	0.45 (0.14-1.42)	.17
Third- or fourth-generation cephalosporins	1.17 (0.76-1.79)	.48
Carbapenems	1.05 (0.63-1.75)	.84
Piperacillin-tazobactam	1.31 (0.82-2.10)	.27
Other penicillin	0.47 (0.23-0.98)	.04
Metronidazole	1.31 (0.83-2.07)	.24
Vancomycin	—	—
Oral	1.38 (0.52-5.89)	.67
Intravenous	1.55 (0.88-2.73)	.13
Aminoglycosides	1.27 (0.78-2.06)	.35
Multiple (≥2 antibiotic classes)	1.28 (0.75-2.21)	.37

NOTE. APACHE, Acute Physiology and Chronic Health Evaluation; CI, confidence interval; HR, hazard ratio.

Shaughnessy MK, et al. ICHE 2011;32:201-206

## Disinfectants and Antiseptics

*C. difficile* spores at 10 and 20 min, Rutala et al, 2006

- ~4 log<sub>10</sub> reduction (5 *C. difficile* strains including BI-9)
  - Clorox, 1:10, ~6,000 ppm chlorine (but not 1:50, ~1,200 ppm)
  - Clorox Clean-up, ~19,100 ppm chlorine
  - Tilex, ~25,000 ppm chlorine
  - Steris 20 sterilant, 0.2% peracetic acid
  - Cidex, 2.4% glutaraldehyde
  - Cidex-OPA, 0.55% OPA
  - Wavicide, 2.65% glutaraldehyde
  - Aldahol, 3.4% glutaraldehyde and 26% alcohol

## DISINFECTANTS

No measurable activity (1 *C. difficile* strain, J9; spores at 20 min)

- Vesphene (phenolic)
- 70% isopropyl alcohol
- 95% ethanol
- 3% hydrogen peroxide
- Clorox disinfecting spray (65% ethanol, 0.6% QUAT)
- Lysol II disinfecting spray (79% ethanol, 0.1% QUAT)
- TBQ (0.06% QUAT); QUAT may increase sporulation capacity- (Lancet 2000;356:1324)
- Novaplus (10% povidone iodine)
- Accel (0.5% hydrogen peroxide)

Rutala W, Weber D, et al. 2006

## Environmental Surface Disinfection

Product-5000-6000ppm chlorine effective  
(or another sporicidal product)

## Effect of Hypochlorite on Environmental Contamination and Incidence of *C. difficile*

- Use of chlorine (500-1600 ppm) decreased surface contamination and the outbreak ended. Mean CFU/positive culture in outbreak 5.1, reduced to 2.0 with chlorine. (Kaatz et al. Am J Epid 1988;127:1289)
- In an intervention study, the incidence of CDAD for bone marrow transplant patients decreased significantly, from 8.6 to 3.3 cases per 1000 patient days after the environmental disinfection was switched from QUAT to 1:10 hypochlorite solution in the rooms of patients with CDAD. No reduction in CDAD rates was seen among NS-ICU and medicine patients for whom baseline rates were 3.0 and 1.3 cases per 1000-patient days. (Mayfield et al. Clin Inf Dis 2000;31:995)

## Effect of Hypochlorite on Environmental Contamination and Incidence of *C. difficile*

- 35% of 1128 environmental cultures were positive for *C. difficile*. To determine how best to decontaminate, a cross-over study conducted. There was a significant decrease of *C. difficile* on one of two medicine wards (8.9 to 5.3 per 100 admissions) using hypochlorite (1,000 ppm) vs. detergent. (Wilcox et al. J Hosp Infect 2003;54:109)
- Acidified bleach (5,000 ppm) and the highest concentration of regular bleach tested (5,000 ppm) could inactivate all the spores in <10 minutes. (Perez et al. AJIC 2005;33:320)

## PROVING THAT ENVIRONMENTAL CONTAMINATION IS IMPORTANT IN *C. difficile* TRANSMISSION

- Environmental persistence (Kim et al. JID 1981;14342)
- Frequent environmental contamination (McFarland et al. NEJM 1989;320:204)
- Demonstration of HCW hand contamination (Samore et al. AJM 1996;100:32)
- Environmental  $\Rightarrow$  hand contamination (Samore et al. AJM 1996;100:32)
- Person-to-person transmission (Raxach et al. ICHE 2005;26:691)
- Transmission associated with environmental contamination (Samore et al. AJM 1996;100:32)
- CDI room a risk factor (Shaughnessy et al. IDSA/ICAAC. Abstract K-4194)
- Improved disinfection  $\Rightarrow$   $\Downarrow$  epidemic CDI (Kaatz et al. AJE 1988;127:1289)
- Improved disinfection  $\Rightarrow$   $\Downarrow$  hyperendemic CDI (Boyce et al. ICHE 2008;29:723)

## Evidence Supporting the Role of Environmental Contamination in Transmission of CDI

- *C. difficile* is able to survive for prolonged periods in the environment
- Environmental contamination is frequently found in the rooms of infected patients
- Contaminated environmental reservoirs have been demonstrated to be the source of an outbreak
- Contamination of healthcare personnel hands has been demonstrated
- Levels of environmental contamination are associated with the frequency of healthcare personnel hand contamination
- Prevalence of environmental contamination has been associated with the incidence of patient acquisition and infection
- Admission to a room previously occupied by an infected patient is associated with the risk of colonization or infection
- Environmental cleaning and disinfection has been demonstrated to reduce the hospital incidence of CDI

Weber DJ, Rutala WA. ICHE 2011;32:207-209

## CLINICAL PRACTICE GUIDELINES FOR *C. difficile*, SHEA & IDSA, 2010

- HCWs and visitors must use gloves (AI) and gowns (BIII) on entry to room
- Emphasize compliance with the practice of hand hygiene (AII)
- In a setting in which there is an outbreak or an increased CDI rate, instruct visitors and HCP to wash hands with soap (or antimicrobial soap) and water after caring for or contacting patients with CDI (BIII)
- Accommodate patients with CDI in a private room with Contact Precautions (BIII)
- Maintain Contact Precautions for the duration of diarrhea (CIII)
- Identification and removal of environmental sources of *C. difficile*, including replacement of electronic rectal thermometers with disposables, can reduce the incidence of CDI (BII)
- Use chlorine containing cleaning agents or other sporicidal agents in areas with increased rates of CDI (BII)
- Routine environmental screening for *C. difficile* is NOT recommended (CIII)

Cohen SH, et al. ICHE 2010;31:431-435

### UNC HEALTH CARE ISOLATION SIGN FOR PATIENTS WITH NOROVIRUS OR *C. difficile*

- Use term Contact-Enteric Precautions
- Requires gloves and gown when entering room
- Recommends hand hygiene with soap and water (instead of alcohol-based antiseptic)
- Information in English and Spanish



### Role of Hospital Surfaces in Transmission Summary

- Contaminated environment likely important for *Acinetobacter*, norovirus, and *C. difficile*
- Supportive data include: prior room occupant with pathogen a significant risk for acquisition; an improved surface cleaning/disinfection reduces disease incidence
- Adhere to proper room cleaning and disinfection protocols which may include monitoring, new methods for room decontamination and practice improvements (checklist, assignments, education)

### Role of Surfaces

- Factors that facilitate environmental transmission
  - *Acinetobacter*
  - Norovirus
  - *C. difficile*

Thank you