

# Literature 'Bundles' In Infection Prevention & Control

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# Conflicts Of Interests:

None to declare

# Impact Factors (2007/2010) Of The Mainstream Infection Prevention & Control Literature. A Comparison ...

- S AFRICAN MEDICAL JOURNAL 1.268 >1.676
- BMC INFECTIOUS DISEASES 2.825
- AMERICAN JOURNAL OF INFECTION CONTROL 1.907 >3.036
- JOURNAL OF HOSPITAL INFECTION 2.47 >3.078
- INFECTIOUS CONTROL & HOSP EPIDEMIOLOGY 2.989 >3.571
- CURRENT OPINION IN INFECTIOUS DISEASES 4.754 >5.167
- JOURNAL OF ANTIMICROBIAL CHEMOTHERAPY 5.682 >4.659
- AIDS 5.842 >6.348
- EMERGING INFECTIOUS DISEASES 5.775 >6.859
- JOURNAL OF INFECTIOUS DISEASES 6.035 >6.288
- CLINICAL INFECTIOUS DISEASES 6.75 >8.186
- CRITICAL CARE MEDICINE 6.823 >6.250
- BMJ 9.723 >13.741
- LANCET INFECTIOUS DISEASES 12.058 > 16.144
- JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION 25.547 >30.011
- LANCET 28.638 >33.633
- NEW ENGLAND MEDICAL JOURNAL 52.589 >53.484

# Articles Chosen on the Basis of ...

- 1 An excuse to 'talk-the-talk' of bundles
- 2 Not necessarily groundbreaking but offering new research methodologies and knowledge in IPC
- 3 Introducing evaluation of new technologies to keep up-to-date with presentations by industry representatives
- 4 To ensure that duplication by other speakers is avoided in this session due to the peculiarities of papers chosen
- 5 Provide some 'fun' to IPCs who will appreciate the diversity of IPC research questions

# 1 The PPE “Bundle”

Trans-ocular Entry of Seasonal  
Influenza-Attenuated Virus aerosols  
& The Efficacy of N95 Respirators,  
Surgical Masks & Eye Protection In  
Humans

*JID 2011:204;193-9*

# Background:

- Efficacy of barrier precautions to prevent influenza transmission is poorly understood
- New testing methodology developed to allow accurate, reproducible delivery of defined mono-dispersed concentrations of live viruses to humans in fully controlled environment
- Study represents first data regarding the efficacy of commonly used barrier precautions against influenza

# Methods:

- 28 healthy participants (19 F, 9 M, av. age 30.5y) who did not receive seasonal flu vaccine & had no active flu before enrollment
- Assigned to 1 of 6 groups:
  - Group 1: no precautions (n=4)
  - Group 2: ocular exposure only (n=4) [half-mask respirator used]
  - Group 3: surgical mask, no eye protection (n=5)
  - Group 4: surgical mask, eye protection (n=5)
  - Group 5: fit-tested N95 respirator, no eye protection (n=5)
  - Group 6: fit-tested N95 respirator with eye protection (n=5)
- Exposed, in a test chamber, to cold-adapted live attenuated influenza vaccine (3 strains) particles (4.9 $\mu$ m) generated by a vibrating-orifice aerosol generator
- Endpoints: nasal washes, RT-PCR, viral culture

# Results:

- Group 1: no precautions – influenza detected in 4/4 (95% CI, 0-0.60)
  - Group 2: ocular exposure only – 3/4 (95% CI, 0.006-0.806)
  - Group 3: surgical mask, no eye protection – 5/5 (95% CI, 0-0.522)
  - Group 4: surgical mask, eye protection - 5/5 (95% CI, 0-0.522)
  - Group 5: fit-tested N95 respirator, no eye protection - 3/5 (95% CI, 0.053-0.853)
  - Group 6: fit-tested N95 respirator with eye protection – 1/5 (95% CI, 0.05-0.72)
- Amount of RT-RNA detected by PCR used for quantitative comparisons of the intervention groups: Significant differences of viral copies were detected between group1 (no mask) and all other groups ( $P<0.05$ ) except group 3 ( $P=0.62$ )

# Comments & conclusions 1:

- Several limitations:
  - Potential differences between vaccine strains, wild-type seasonal influenza, & pandemic H1N1
  - Limited sample size precluded *P* value calculations for group count outcomes [exact 95% CIs used to establish an estimate of certainty]
  - Limitation of models of masks & respirators tested
  - Scant knowledge about: (i) influenza viral particle load necessary to infect an individual, (ii) dispersal pattern of influenza produced by affected patients, (iii) differences between mechanically- versus humanly-generated aerosols

# Comments & conclusions 2:

- However ...
  - Novel approach to evaluate preventive qualities of barrier (PPE) precautions in a controlled testing environment, allowing manipulation of key variables such as viral load, particle size, temperature and humidity during human exposure
  - Provides first insights into trans-ocular transmission dynamics of influenza, allowing viral particles fast and easy access to URT, and efficacy of PPE tested
  - Study points to the need for combining effective respirator types with eye protection to successfully interrupt aerosol transmission of influenza

# Protecting Healthcare Workers From Pandemic Influenza: N95 Or Surgical Masks?

*Crit Care Med 2010;38(2):657-667*

# In Essence...

- Literature review undertaken for evidence superior protective value of N95 respirators or surgical masks for HCW against influenza. Total reviewed: 21 mask studies in H-C settings & 25 laboratory based publications
- Most studies used medium or lower evidence study design – important confounders include unrecognized impact of concurrent bundling of other IPC measures, mask compliance, contamination from improper doffing of RPE, and ocular inoculation
- Only 3 studies directly compared protective value of surgical masks with N95 respirators
- Laboratory studies show that N95 masks afford superior protection against particles of similar size to influenza

# So...

- Paucity of high-quality studies in H-C setting, WHO advocacy of what RPE is required for influenza is not entirely evidence-based
- Laboratory studies of potential airborne spread of influenza from shedding patients indicate that guidelines related to current 1 metre respiratory zone may need to be extended (2-3 metres) & include protection from ocular inoculation
- RPE alone is unlikely to protect effectively against respiratory pathogens unless combined with synergistic IPC techniques
- In the event of a pandemic, mask use alone will, in addition to IPC measures, be bundled with other measures such as prophylaxis, vaccination & other PPE including face shields/goggles

## 2 A “Bundle” Of Problematic Gram-positives

Impact Of Combined Low-Level  
Mupirocin And Genotypic Chlorhexidine  
Resistance On Persistent MRSA Carriage  
After Decolonization Therapy: A Case-  
control Study

*Clinical Infectious Diseases 2011;52(12):1422-  
1430*

# Background 1:

- Colonization with MRSA increases risk of adverse event outcomes with 10%-30% of carriers developing MRSA infection
- MRSA carriers also act as reservoirs in HCFs with risk of transmission to other patients
- Intranasal mupirocin and chlorhexidine washing are widely used to decolonize carriers.

But, increasing resistance to these agents reported

# Background 2:

- High-level mupirocin-R MRSA (MIC  $\geq 512$   $\mu\text{g}/\text{mL}$ ) harbour a plasmid-encoded *mupA* gene & are associated with decolonization failure
- Low-level mupirocin-R MRSA (MIC 8-256  $\mu\text{g}/\text{mL}$ ) have mutations in native tRNA synthase (V588F point mutation)
- Chlorhexidine R associated with plasmid-borne *qacA/B* genes that code for MDR efflux pumps, resulting in at least 2-4-fold increases in MBCs

Clinical relevance of low-level mupirocin R (as determined by Etest MICs & molecular assay for V588F point mutation) and genotypic chlorhexidine resistance (as determined by presence of *qacA/B* genes) remains unclear

# Methods:

- Nested case-control study of MRSA carriers who received decolonization therapy from 2001-2008
- Cases, patients who remained colonized, were matched by year to controls, those in whom MRSA was eradicated (follow-up, 2y)
- Baseline MRSA isolates tested for mupirocin R & chlorhexidine R
- [Also, SCC *mec* determination was performed on all pre- and available post-decolonization isolates as well as multilocus variable number of tandem repeats analysis (MLVA) & MLST]
- Effect of primary exposure of interest (low-level mupirocin & genotypic chlorhexidine R) was evaluated with multivariate conditional logistic regression analysis

# Results:

- The 75 case and 75 control patients were similar except that those persistently colonized were older ( $P=0.007$ ) with longer lengths of hospital stay ( $P=0.001$ )
- After multivariate analysis, carriage of combined low-level mupirocin & genotypic chlorhexidine R before decolonization independently predicted persistent MRSA carriage (OR, 3.4 [95%CI, 1.5-7.8])
- Other risk factors were older age (OR, 1.04 [95%CI, 1.02-1.1]), previous hospitalization (OR, 2.4 [95%CI, 1.1-5.7]), presence of a skin wound (OR, 5.7 [95%CI, 9.8-17.6]), recent antibiotic use (OR, 3.1 [95% CI, 1.3-7.2]), and CVC (OR, 5.7[95%CI, 1.4-23.9])

# Comments & conclusions 1:

- Genotypic analyses & resistance after decolonization showed that all low-level mupirocin R isolates contained SCC *mecl* & the V588F point mutation
- Interestingly, *mupA* usually associated with high-level mupirocin R was present in all low-level R isolates with mupirocin MICs  $\geq 64$   $\mu\text{g/mL}$
- MLVA typing of pre- and post-decolonization isolates were identical (i.e. MRSA persistence / relapse, not exogenous!)
- In 15% of cases: mupirocin R isolates at baseline developed R after decolonization; further 3% of cases had low-level mupirocin R MRSA at baseline & high-level R MRSA post decolonization – these MRSA isolates carried both the V588F point mutation and *mupA* gene at baseline

# Comments and conclusions 2:

- V588F mutation, not associated with fitness cost PLUS the fact that MRSA strains that carry the *qacA/B* genes have increased transmission potential when chlorhexidine-based surface disinfectants are used MAY explain why R strains predominate in certain institutions where targeted decolonization of MRSA carriers has been routine practice for decades
- Combined low-level mupirocin and genotypic chlorhexidine R (not latter alone) significantly increases the risk of MRSA carriage after decolonization therapy
- Institutions with widespread use of these agents should monitor for R and loss of clinical effectiveness

Nosocomial spread of hospital-  
adapted CC17 vancomycin resistant  
*Enterococcus faecium* in a tertiary-  
care hospital of  
Beijing, China

*Chin Med J* 2011;124(4):498-503

# Background:

- VREs were firstly isolated from patients in the UK, and shortly after, in the US
- Currently, they are a world-wide problem & of public health concern (fear of emergence of VRSA from plasmid transfer from e.g. VR *E faecium* (VREF))
- Incidence of VREs appears to be increasing in China, but few nosocomial outbreaks have been reported
- VRE isolates from a Beijing Academic Hospital (1100 beds) outbreak (2008-9) were characterized molecularly and the effect of IPC measures were studied

# Methods:

- 32 VRE isolates from 20 infected & 12 colonized patients were identified and phenotypically and molecularly characterized (PFGE, multilocus sequence typing, analysis of Tn *1546*-like elements, virulence gene detection)
- 21 strains found in the emergency care intensive unit, 9 from a Geriatric Ward, and 2 from other units
- IPC measures included rigorous environmental disinfection, screening for VRE colonization, contact precautions, education and strict antibiotic restriction

**Table 1.** The sources, antibiotic susceptibility pattern and resistance genes of VREF isolates

Isolates	Patients*	Unit†	Date of isolation	Source	Diagnosis‡	Minimum inhibitory concentration (mg/L)										Resistance genes	
						CI	GM	AM	VAN	TP	LVX	LNZ	RIFP	EM	DC		
E1	P1	E	March 2008	Urine	I	>32	>1024	>256	>256	32	>32	0.5	24	>256	32	vanA	aac(6′)-aph(2′′)
E2	P2	E	March 2008	Urine	I	>32	>1024	>256	64	48	>32	1.0	>32	>256	0.125	vanA	aac(6′)-aph(2′′)
E3	P3	Ger														A	aac(6′)-aph(2′′)
E4	P4	E														A	aac(6′)-aph(2′′)
E6	P5	E														A	aac(6′)-aph(2′′)
E8	P6	E														A	aac(6′)-aph(2′′)
E10	P7	E														A	aac(6′)-aph(2′′)
E11	P8	E														A	aac(6′)-aph(2′′)
E12	P9	E														A	aac(6′)-aph(2′′)
E13	P10	E														A	aac(6′)-aph(2′′)
E14	P11	E														A	aac(6′)-aph(2′′)
E19	P12	E														A	aac(6′)-aph(2′′)
E20	P13	E														A	aac(6′)-aph(2′′)
E23	P14	Ger														A	aac(6′)-aph(2′′)
E24	P15	Ger														A	aac(6′)-aph(2′′)
E25	P16	Ger														A	aac(6′)-aph(2′′)
E26	P17	E														A	aac(6′)-aph(2′′)
E27	P18	Ger														A	aac(6′)-aph(2′′)
E28	P19	Neu														A	aac(6′)-aph(2′′)
E29	P20	Ger														A	aac(6′)-aph(2′′)
E32	P21	Ger														A	aac(6′)-aph(2′′)
E55	P22	Ger														A	aac(6′)-aph(2′′)
E65	P23	E														A	aac(6′)-aph(2′′)
E84	P24	E														A	aac(6′)-aph(2′′)
E91	P25	E														A	aac(6′)-aph(2′′)
E97	P26	Ger	February 2009	Stool	C	>32	>1024	>256	>256	6	>32	1.0	4	>256	32	vanA	aac(6′)-aph(2′′)
E98	P27	E	February 2009	Stool	C	>32	>1024	>256	>256	64	>32	0.75	2	>256	32	vanA	aac(6′)-aph(2′′)
E100	P28	E	February 2009	Stool	C	>32	>1024	>256	>256	32	>32	1.0	4	>256	0.125	vanA	aac(6′)-aph(2′′)
E102	P29	E	February 2009	Stool	C	>32	>1024	>256	>256	32	>32	1.0	4	>256	32	vanA	aac(6′)-aph(2′′)
E104	P30	ICU	March 2009	Blood	I	>32	>1024	>256	128	64	>32	0.75	4	>256	32	vanA	aac(6′)-aph(2′′)
E105	P31	E	March 2009	Stool	C	>32	>1024	>256	>256	64	>32	0.38	2	>256	32	vanA	aac(6′)-aph(2′′)
E106	P32	E	March 2009	Urine	I	>32	>1024	>256	>256	64	>32	1.0	2	>256	32	vanA	aac(6′)-aph(2′′)

- 20/32 were clinically infected; rest, colonized  
 -VR genes detected using multiplex PCR  
 -PCR amplification was performed to detect aminoglycoside-modified enzymes  
 - virulence genes *esp* and *hyl* detected using multiplex PCR  
 -All isolates harboured the *vanA* gene, however 4 harboured the *vanB* phenotype (resistant to vancomycin [MICs 64- >256µg/mL] but susceptible to teicoplanin [MICs 2-6µg/mL])  
 -In addition to glycopeptide R all isolates were R to amp, erythromycin & FQs and all but one were high-level R to gentamicin  
 -Linezolid had excellent *in vitro* activity against MDR VREF isolates

\*Patients, indicated by P, were numbered in chronological order. †Unit: E: Emergency Intensive Care Unit (EICU); Ger: Geriatric Ward. ICU: Intensive Care Unit; Neu: Neurosurgery Unit. ‡Diagnosis: C: colonized; I: infected. CI: ciprofloxacin; GM: gentamicin; AM: ampicillin; VAN: vancomycin; TP: teicoplanin; LVX: levofloxacin; LNZ: linezolid; RIFP: rifampin; EM: erythromycin; DC: doxycycl.

**Table 2.** Molecular characteristics of isolates

Isolates	ST (allelic profile)	PFGE	esp <sup>†</sup>	hyl <sup>†</sup>	Conjugation frequency
E1	343 (15-1-1-39-1-20-1)	E	P	N	3.1×10 <sup>-5</sup>
E2	78 (15-1-1-1-1-1-1)	E	P	N	1.4×10 <sup>-5</sup>
E3	203 (15-1-1-1-1-20-1)	H	P	N	1.3×10 <sup>-6</sup>
E4	203 (15-1-1-1-1-20-1)	A1	P	P	5.5×10 <sup>-5</sup>
E6	203 (15-1-1-1-1-20-1)	A1	P	P	7.6×10 <sup>-5</sup>
E8	203 (15-1-1-1-1-20-1)	A1	P	P	1.9×10 <sup>-6</sup>
E10	203 (15-1-1-1-1-20-1)	C	P	N	1.8×10 <sup>-6</sup>
E11	203 (15-1-1-1-1-20-1)	A1	P	P	1.1×10 <sup>-5</sup>
E12	192 (15-1-1-1-1-7-1)	D	P	N	1.9×10 <sup>-5</sup>
E13	203 (15-1-1-1-1-20-1)	G	P	P	3.4×10 <sup>-6</sup>
E14	78 (15-1-1-1-1-1-1)	E	P	P	2.5×10 <sup>-5</sup>
E19	203 (15-1-1-1-1-20-1)	A1	P	P	1.3×10 <sup>-5</sup>
E20	203 (15-1-1-1-1-20-1)	A2	P	P	<10 <sup>-8</sup>
E23	203 (15-1-1-1-1-20-1)	A1	P	P	6.7×10 <sup>-6</sup>
E24	363 (15-1-1-39-1-20-1)	B	P	N	4.6×10 <sup>-5</sup>
E25	78 (15-1-1-1-1-1-1)	F1	P	N	1.8×10 <sup>-5</sup>
E26	363 (15-1-1-39-1-20-1)	A2	P	P	2.7×10 <sup>-5</sup>
E27	78 (15-1-1-1-1-1-1)	F1	P	P	1.9×10 <sup>-5</sup>
E28	414 (15-5-1-1-1-20-1)	F2	P	P	1.5×10 <sup>-6</sup>
E29	203 (15-1-1-1-1-20-1)	F1	P	N	1.8×10 <sup>-6</sup>
E32	203 (15-1-1-1-1-20-1)	F1	P	N	1.8×10 <sup>-5</sup>
E55	192 (15-1-1-1-1-7-1)	E	P	N	7.2×10 <sup>-7</sup>
E65	414 (15-5-1-1-1-20-1)	A1	P	N	<10 <sup>-8</sup>
E84	203 (15-1-1-1-1-20-1)	A1	P	P	2.7×10 <sup>-5</sup>
E91	203 (15-1-1-1-1-20-1)	A2	P	P	6.7×10 <sup>-6</sup>
E97	78 (15-1-1-1-1-1-1)	F1	P	P	1.9×10 <sup>-6</sup>
E98	203 (15-1-1-1-1-20-1)	A1	P	P	2.7×10 <sup>-5</sup>
E100	203 (15-1-1-1-1-20-1)	A1	P	N	1.5×10 <sup>-6</sup>
E102	78 (15-1-1-1-1-1-1)	A1	P	N	4.6×10 <sup>-6</sup>
E104	192 (15-1-1-1-1-7-1)	B	P	N	1.1×10 <sup>-5</sup>
E105	203 (15-1-1-1-1-20-1)	A1	P	P	4.6×10 <sup>-6</sup>
E106	203 (15-1-1-1-1-20-1)	A1	P	N	3.4×10 <sup>-5</sup>

<sup>†</sup>Designations at <http://efaecium.mlst.net>. <sup>†</sup>The *esp* gene and *hyl* gene were detected by PCR amplification, as described in the Methods. P: positive; N: negative.

**Table 2.** Molecular characteristics of isolates

-The *esp* gene was detected in all isolates, while the *hyl* gene was detected in only 17 strains

-Conjugation studies showed that, for 30 isolates vancomycin R was transferred at a frequency of 10<sup>-5</sup>/donor to 10<sup>-7</sup>/donor

-All transconjugants expressed R to vanco/teico with MICs equal to/lower than those of donor isolates except for 4 VanB phenotype-*vanA* genotype transconjugants were still susceptible to teicoplanin

>> This suggests that *vanA* gene with VanB phenotype can transfer between different strains

- 6 STs identified by MLST, most prevalent being ST203. Cluster analysis by eBURST showed all VRE strains belonged to CC17  
-PFGE: 8 patterns, commonest being A, F & E

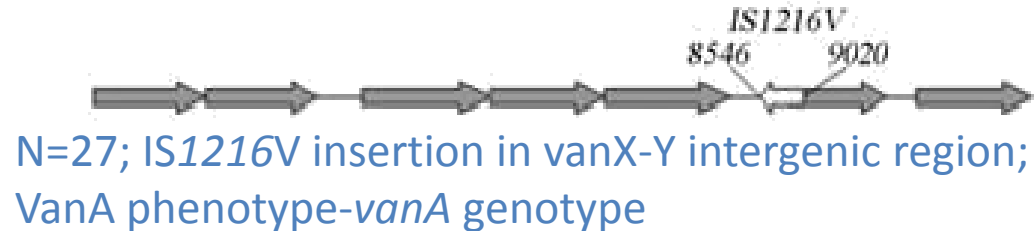
CMJ

BM4147



Type 1

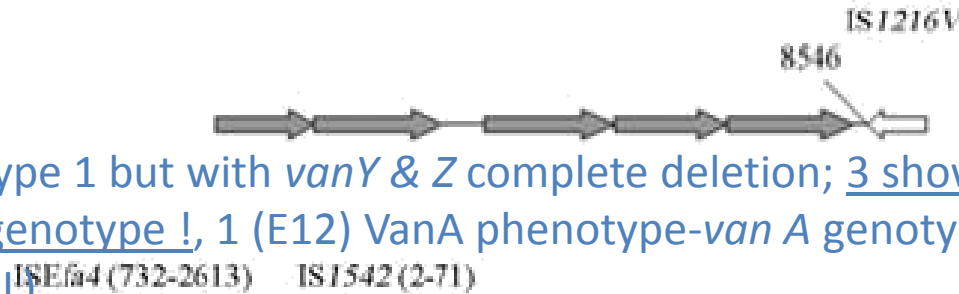
E1, E2, E3, E4, E6, E8, E10, E11, E13, E14, E19, E20, E23, E25, E26, E27, E29, E32, E65, E84, E91, E98, E100, E103, E104, E105, E105



Type 2

E12, E24, E55, E97

N=4; insertion as type 1 but with *vanY* & *Z* complete deletion; 3 showed *vanB* phenotype-*van A* genotype !, 1 (E12) VanA phenotype-*van A* genotype but IR to teico (MIC 16µg/mL)



Type 3

E28

N=1; large IS 1542-*van R* intergenic fragment, a ISEfa4 insertion, & similar insertion of IS1216V as types 1&2; *vanB* phenotype-*van A* genotype



**Figure 2. Structures of Tn1546-like elements of 32 VRE isolates typed by PCR. The location of genes and the direction of transcription of normal Tn1546-like element are shown at the top. The white arrows represent IS element. And the gray arrows are genes with interruption by IS elements. The positions of IS insertion sites are also shown.**

# Comments and conclusions:

- Recent studies have shown that majority of nosocomial VREF infections globally belong to CC17. Current Beijing study confirms this
- CC17 strains + *esp* putative virulence gene are associated with high-level R to most antibiotics
- *hyl* gene found with higher frequency in true infection vs. colonization
- Reports have shown that point mutations in sensor domain of vanS gene (not found in this study) & impairments of VanY&Z lead to teicoplanin susceptibility (found in 3 of 4 Type 2 strains)
- In current study same Tn1546 type was found in genetically diverse enterococci suggesting horizontal gene transfer of R
- Continuous IPC implementation resulted in outbreak being confined to EICU & the Geriatric Ward. No further cases occurred in a one-year period following the termination of the outbreak

# 3 Environmental Decontamination “Bundle”

# Terminal Decontamination Of Patient Rooms Using An Automated Mobile UV light Unit

*ICHE 2011;32(8):737-742*

# Background:

- For decades, environmental surfaces considered to play a minimal to no role in transmission of HAIs
- There is now ample evidence to suggest that environmental contamination plays an important role in transmission of HAI-pathogens such as MRSA, VRE, *Acinetobacter*, *C difficile*, and norovirus
  - *JHI 2009;73:378-385; JHI 2007;65:50-54; AJIC 2010;38:S25-S44; Infect Dis Clinic North Am 2011;25:45-76*
- Overall thoroughness of cleaning can range from 49% (*ICHE 2008;29:1-7*) to 57.1% (*JHI 2008;68:39-44*)
- Purpose of study was to determine the ability of a mobile UV light unit to reduce bacterial contamination of environmental surfaces in patient rooms

# Methods 1:

- An automated mobile UV light unit that emits UV-C light (dose set at 22000  $\mu\text{Ws}/\text{cm}^2$  to also eradicate bacterial spores) was placed in 25 patient rooms after patient discharge and terminal cleaning
- Unit operated using a 1- or 2-stage procedure
  - 1-stage: unit placed in the centre of the patient's room with door to bathroom open
  - 2-stage: (i) device placed and operated in the patient's bathroom with door closed [to deal with areas in bathroom that were not in direct line of sight of UV light) & (ii) device placed near centre of patient's room and operated with door closed

# Methods 2:

- For stage procedures 1 & 2:

Aerobic colony counts from 5 high-touch surfaces (e.g. bedside rail, overbed table, bathroom grab bar, etc.),

AND

Log reduction in *C difficile* spores (using uniformly spore-inoculated stainless steel carrier discs containing inoculum of  $10^5$ )

were determined before and after UV light decontamination (UVLD)

- To determine whether ozone (which can be generated by UV-C light in  $\lambda < 200$  nm) contributed to the antimicrobial effect of UVLD, in-room ozone concentrations were recorded before and at the end of UVLD cycles

# Results:

- For 1-stage procedure:
  - Mean aerobic colony counts for 5 high-touch surfaces ranged from 10.6-98.2 CFUs per Day/Engley (D/E) contact plate before UVLD to 0.3-24.0 CFUs after UVLD
  - Mean *C difficile spore* log reductions ranged 1.8 to 2.9. UVLD cycle times ranged from 34.2 to 100.1 minutes
- For 2-stage procedure:
  - Mean aerobic colony counts ranged from 10.0 to 89.2 CFUs per D/E plate before UVLD and were 0 CFUs after UVLD
  - Mean *C difficile spore* log reductions ranged from 1.4 to 3.2
- In-room ozone concentrations during UVLD ranged from undetectable to 0.012 ppm

# Conclusions:

- The mobile UV-C unit statistically and significantly reduced aerobic colony counts and *C difficile* spores on contaminated surfaces in patient's rooms
- This is the third study to demonstrate that an automated UVLD device significantly reduced contamination on high-touch surfaces in patient's rooms. (The 2 previous studies are reported in [BMC Infect Dis 2010;10:197](#) and [ICHE 2010;31:1025-1029](#))

# Comments:

- Commercially-available room no-touch disinfection units use 1 of 2 methods: UVLD or hydrogen peroxide (in dry mist/vapour)
- Advantages and disadvantages of these 2 technologies must be understood by IPCPs - [ICHE 2011;32\(8\):745 – Table 2](#).
- These technologies supplement but do not replace standard cleaning & disinfection. Dirt and debris must be removed!
- Cleaning ‘bundles’ include multiple activities: improved education, monitoring the thoroughness of cleaning, performance feedback, cleaning checklists, etc. Despite these, environmental cleaning is generally suboptimal. If additional studies continue to demonstrate effectiveness of ‘no touch’ technologies their use should be considered for terminal room disinfection
- Thus far, with the exception of the reduction in incidence of *C difficile* using HP vapour, there is no published evidence of clinical impact of these no-touch technologies

**THANK YOU FOR YOUR  
ATTENTION !**