Table 1. All Eye Exposure Incident Reports and Eye Protection Use Reported During that Exposure by Year; Exposure Prevention Information Network (EPINet) Healthcare Surveillance Research Group Network

Year	All Mucocutaneous Exposure Incidents/100 Average Daily Census (ADC)	% Eye Exposure Incidents	% Wearing Eye Protection	% Occurring in Patient/Exam Room
2017	10.1	48.1	3	61.3
2016	12.9	64.9	5.9	49.1
2015	11.4	66.9	6.9	51.9
2014	8.9	65.7	2.8	40.4
2013	5.9	64.5	12.8	28.1

for patients with suspected or proven respiratory viral infection. This protocol would err on the side of caution in an attempt to mitigate the risk of transmission to healthcare workers and others."

The Centers for Disease Control and Prevention (CDC), the Association of periOperative Registered Nurses (AORN), the Occupational Safety and Health Administration (OSHA), and others recommend similar protective measures: to use "(m)ask and goggles or a face shield . . . Use during patient care activities likely to generate splashes or sprays of blood, body fluids, secretions, or excretions." Incidence data demonstrate that guidance is neither protective nor prescriptive enough. Because most mucus membrane exposures occur to the eyes and because eye protection use is low (2.8%–12.8%), more specific guidance needs to include use not only "when splashes or sprays are likely" but also with elements of measure, control, and surveillance (occupational health, environmental health and safety, industrial hygiene, employee health, infection prevention, etc. rounds). Healthcare employers should improve availability and accessibility of protective eyewear in patient, exam, and procedure rooms, similar to including infection prevention and control caddies (gloves, gowns) for transmission- and contact-based or isolation precautions.

Given the increasing prevalence in patients with coinfection of human immunodeficiency virus (HIV) and hepatitis C virus (HCV), hepatitis B virus (HBV), tuberculosis (TB), and multidrug-resistant organisms (MDROs) such as MRSA, protecting healthcare personnel is more critical than ever.[8–10] A single eye exposure can result in transmission of 1 or more pathogenic organisms that can result in occupational illness or infection.

Author ORCIDs. Amber Hogan Mitchell, 0000-0001-6985-7262

Acknowledgments. Would like to thank Ginger Parker for her ongoing dedication to the EPINet Healthcare Surveillance Research Group Network.

Financial support. No financial support was provided relevant to this article.

Conflicts of interest. All authors report no conflicts of interest relevant to this article.

References

1. U.S. EPINet sharps injury and blood and body fluid exposure surveillance research group. blood and body fluid exposure report for 2017; 399 incident reports. International Safety Center website. https://internationalsafetycenter.org/wp-content/uploads/2018/10/Official-2017-BBFSummary.pdf. Published 2018. Accessed December 17, 2018.
2. US EPINet sharps injury and blood and body fluid exposure surveillance research group. blood and body fluid exposure report for 2016; 453 incident reports. International Safety Center website. https://internationalsafetycenter.org/wp-content/uploads/2018/06/Official-2016-BBFSummary.pdf. Published 2018. Accessed December 17, 2018.
3. US EPINet sharps injury and blood and body fluid exposure surveillance research group. blood and body fluid exposure report for 2015; 408 incident reports. International Safety Center website. https://internationalsafetycenter.org/wp-content/uploads/2017/06/Official-2015-BBFSummary.pdf. Published 2017. Accessed December 17, 2018.
4. US EPINet sharps injury and blood and body fluid exposure surveillance research group. blood and body fluid exposure report for 2014; 213 incident reports. International Safety Center website. https://internationalsafetycenter.org/wp-content/uploads/2016/08/Official-2014-BBFSummary.pdf. Published 2016. Accessed December 17, 2018.
5. US EPINet sharps injury and blood and body fluid exposure surveillance research group. blood and body fluid exposure report for 2013; 141 incident reports. International Safety Center website. https://internationalsafetycenter.org/wp-content/uploads/2015/08/Official-2013-BBFSummary.pdf. Published 2015. Accessed December 17, 2018.
6. Mermel, L. Eye protection for preventing transmission of respiratory viral infections to healthcare workers. *Infect Control Hosp Epidemiol* 2018;39:1387–1387.
7. Belser, JA, Rota, PA, Tumpey, TM. Ocular tropism of respiratory viruses. *Microbiol Mol Biol Rev* 2013;77:144–156.
8. Coinfection with HIV and viral hepatitis. Centers for Disease Control and Prevention website. https://www.cdc.gov/hepatitis/hiv-hepatitis-coinfection.htm. Updated February 7, 2018. Accessed December 17, 2018.
9. Bruchfeld J, Correia-Neves M, Källenius G. Tuberculosis and HIV coinfection. *Cold Spring Harb Perspect Med* 2015;5:a017871.
10. Centers for Disease Control and Prevention. Invasive methicillin-resistant Staphylococcus aureus infections among persons who inject drugs—six sites, 2005–2016. *Morbid Mortal Wkly* 2018;67:625–628.

Why do susceptible bacteria become resistant to infection control measures? A *Pseudomonas* biofilm example

Leandro Reus Rodrigues Perez PhD

Hospital Mãe de Deus, Porto Alegre, Brazil

Author for correspondence: Leandro Reus Rodrigues Perez, E-mail: leandro.reus@gmail.com

Cite this article: Perez LRR. (2019). Why do susceptible bacteria become resistant to infection control measures? A *Pseudomonas* biofilm example. *Infection Control & Hospital Epidemiology*, 40: 386–388, https://doi.org/10.1017/ice.2018.348

To the Editor—Pseudomonas aeruginosa is an opportunistic pathogen involved in a wide variety of infections among hospitalized patients; it is one of the main agents that cause pneumonia in mechanically ventilated patients.[1] After colonizing the respiratory tract, *P. aeruginosa* may lead to extensive damage to the host tissues

Table 1. Quorum-Sensing (QS) Genes, Ability to Produce Biofilm and Minimal Inhibitory Concentration (MIC) and Minimal Biofilm Eradication Concentration (MBEC) Among 199 Meropenem-Susceptible *P. aeruginosa* Isolates

	QS Genes					Meropenem, Range in μg/mL	
No. of Isolates (%)	*las*I	*las*R	*rhl*I	*rhl*R	Biofilm Production	MIC	MBEC
62 (31.1)	Pos	Pos	Pos	Pos	Moderate	0.125–2.0	0.5–64.0
61 (30.6)	Pos	Pos	Pos	Pos	Strong	0.064–2.0	0.5–64.0
38 (19.1)	Pos	Pos	Pos	Pos	Weak	0.064–1.0	0.125–4.0
27 (13.6)	Neg	Pos	Pos	Pos	Moderate	0.032–2.0	0.064–16.0
8 (4.0)	Neg	Neg	Neg	Neg	Nonproducing	0.032–0.5	0.032–0.5
3 (1.5)	Neg	Neg	Pos	Pos	Nonproducing	0.5	0.5

Note. Pos, positive; Neg, negative.

via the production of virulence factors, which are controlled by the quorum-sensing (QS) mechanism, an important cell-to-cell communication system for biofilm formation and maintenance.

As previously reported,[2,3] at least 2 major pathways, the *las* and *rhl* systems, are involved in a biofilm regulation process through the production of signaling molecules (ie, acyl-homoserine lactones; AHLs). In many cases, *P. aeruginosa* is an antibiotic-sensitive strain, but clinical concern arises when an infection involving biofilm needs to be treated. In addition, widely used anti-*Pseudomonas* antibiotics, such as meropenem, can be affected from the standpoint of resistance development when a biofilm is involved. By comparing the minimal inhibitory concentration (MIC) and minimal biofilm eradication concentration (MBEC), we aimed to evaluate (1) the influence of presence of the *las* and *rhl* genes on ability to produce biofilm and (2) the increase in meropenem resistance.

In total, 199 *P. aeruginosa* isolates recovered from endotracheal secretions of hospitalized patients, collected between January 2015 and July 2016, were included in this study. The isolates were identified based on their inability to ferment glucose, on their ability to production a blue–green pigment, and on biochemical tests such as oxidase production, nitrate reduction, and growth on cetrimide agar (bioMérieux, Marcy l'Etoile, France). As inclusion criteria, we selected only isolates (1 per patient) shown to be meropenem susceptible because we aimed to compare the impact of biofilm production on the development of resistance to meropenem.

Biofilm production was performed according to a microtiter plate assay, and polymerase chain reaction (PCR) assays were carried out to determine the presence of *las*I, *las*R, *rhl*I, and *rhl*R genes using specific primers, according to parameters previously described.[3] The minimal inhibitory concentration (MIC) was determined using microtiter plate assays as recommended by the Clinical and Laboratory Standards Institute (CLSI) guidelines (CLSI 2018). The minimal biofilm eradication concentration (MBEC) assay was performed as described by Moskowitz *et al.*[4]

The presence of genes related to the QS system was evaluated by PCR for 199 *P. aeruginosa* isolates. In 161 of these 199 isolates (80.9%), all 4 QS genes evaluated were detected (Table 1). In 8 of the non–biofilm-producing isolates, no QS genes were detected. In 3 other isolates, only genes from the *rhL* system were detected. Thus, we hypothesized a central role of the *las* system, mainly, on the biofilm development and/or structure maintenance. Regarding biofilm producers, 188 of 199 isolates (94.5%) produced any degree of biofilm (Table 1). Importantly, most isolates were moderate or strong biofilm producers, and this finding probably reflects a high meropenem resistance level because high values of MBEC (ie, high levels of resistance) were observed among these isolates (Table 1). In fact, high MBEC/MIC ratios of 1,000× (0.064/64) were found in 12 strong-biofilm–producing isolates. The differences between the values of the MBEC and MIC were more evident for isolates with a moderate or strong ability to produce biofilms, whereas non–biofilm-producing isolates showed the same MBEC and MIC values.

Biofilm formation was largely associated with infections due to colonization of medical devices (eg, catheters and tracheostomy tubes). In these cases, eradication of the infection is difficult because the antimicrobial agents may not penetrate the biofilm, and the decreased metabolic activity of bacteria within biofilms is also due to the increase in gene transfer.[5] In a prior study, we demonstrated that *P. aeruginosa* isolates harboring metallo-β-lactamases had the ability (most strong or moderate) to produce biofilm *in vitro*, which represents an "overlapping of mechanisms" that challenges pulmonary infection treatment.[6] On the other hand, in another study, *Acinetobacter baumannii* complex showed an inverse relationship between meropenem resistance and biofilm formation.[7] Thus, it is important to evaluate the behavioral relation of each bacterial species to better establish targeted prevention efforts and control measures. Although attention has been focused on the widespread resistance to carbapenems, little is known about biofilm production and its important repercussions. Biofilm may represent an important bacterial barrier for control and prevention measures, even for susceptible strains.

To the best of our knowledge, this is the first study evaluating the presence of QS genes among *P. aeruginosa* clinical isolates and its influence on biofilm development and meropenem resistance. Our results have demonstrated that isolates planktonically susceptible to meropenem (under laboratory conditions) may show high levels of resistance to this drug if they occur in biofilm. This finding may reflect a nonresponse to infection control measures based only on standard susceptibility profiles, and greater caution should be taken when biofilm-related infections are suspected.

Acknowledgments. The author would like to thank Sophia L. Perez for technical support.

Financial support. No financial support was provided relevant to this article.

Conflicts of interest. All authors report no conflicts of interest relevant to this article.

https://doi.org/10.1017/ice.2018.348 Published online by Cambridge University Press

References

1. Aaron SD, Kottachchi D, Ferris WJ, *et al.* Sputum versus bronchoscopy for diagnosis of *Pseudomonas aeruginosa* biofilms in cystic fibrosis. *Eur Respir J* 2004;24:631–637.
2. Latifi A, Foglino M, Tanaka K, Williams P, Lazdunski A. A hierarchical quorum-sensing cascade in *Pseudomonas aeruginosa* links the transcriptional activators LasR and RhIR (VsmR) to expression of the stationary-phase sigma factor RpoS. *Mol Microbiol* 1996;21:1137–1146.
3. Perez LR, Machado ABMP, Barth AL. The presence of quorum-sensing genes in *Pseudomonas isolates* infecting cystic fibrosis and non-cystic fibrosis patients. *Curr Microbiol* 2013;66:418–420.
4. Moskowitz SM, Foster JM, Emerson J, Burns JL. Clinically feasible biofilm susceptibility assay for isolates of *Pseudomonas aeruginosa* from patients with cystic fibrosis. *J Clin Microbiol* 2004;42:1915–1922.
5. Stewart PS. 2002. Mechanisms of antibiotic resistance in bacterial biofilms. *Int J Med Microbiol* 2002;292:107–113.
6. Perez LR, Antunes AL, Freitas AL, Barth AL. When the resistance gets clingy: *Pseudomonas aeruginosa* harboring metallo-β-lactamase gene shows high ability to produce biofilm. *Eur J Clin Microbiol Infect Dis* 2012;31:711–714.
7. Perez LR. *Acinetobacter baumannii* displays inverse relationship between meropenem resistance and biofilm production. *J Chemother* 2015;27:13–16.

A comparison of extended-spectrum beta-lactamase (ESBL)–producing *Escherichia coli* and *Klebsiella pneumoniae* bloodstream infections in Alberta using a provincial surveillance system

Kathryn R. Bush MSc[1], Jennifer Ellison MSc[1], Kaitlin Hearn BEH(AD)[1], Ted Pfister MSc[1] and Geoffrey Taylor MD[1,2]

[1]Infection Prevention and Control, Alberta Health Services, Alberta and [2]Department of Medicine, University of Alberta, Edmonton, Alberta

To the Editor—The recent study by Scheuerman *et al*[1] investigating risk factors associated with extended-spectrum β-lactamase (ESBL)–producing *Escherichia coli* and ESBL-producing *Klebsiella pneumoniae* bloodstream infections was of interest to our group and spurred further investigation into local infection prevention and control surveillance data. Data were collected prospectively from acute care facilities within Alberta. The data were retrospectively analyzed for factors comparable to Scheuerman *et al*[1] including gender, age, case classification, time from admission to positive culture, and source of secondary infection. The data reflect all ESBL bloodstream infection cases in Alberta from April 2013 to March 2018, and our results are similar to the findings of Scheuerman *et al.*[1] (Table 1) Of 593 ESBL isolates, 551 (93%) were *E. coli*. Of the cases that were extracted from our database, a statistically significant higher proportion of ESBL-producing *K. pneumoniae* bloodstream infections were classified as hospital acquired or healthcare associated with a longer average time from admission to culture than bloodstream infections with ESBL-producing *E. coli*. Conversely, a statistically significant higher proportion of ESBL *E. coli* bloodstream infection cases were noted to be community-acquired; only 19% of *Klebsiella* isolates were considered community-acquired.

The results obtained within Alberta are similar to the findings of Scheuerman *et al.*[1] Going forward, future investigations may provide additional clarity on the differences between ESBL-producing isolates based on further study of clinical and nonclinical parameters, including the proportion of nonurine ESBL-producing *E. coli* isolates compared to ESBL-producing *K. pneumoniae* isolates, the appropriateness of initial antimicrobial therapy, and the travel history of patients with ESBL infections.

Author ORCIDs. Kaitlin Hearn, 0000-0002-8201-7986

Author for correspondence: Kathryn Bush, Email: kathryn.bush@ahs.ca

Cite this article: Bush KR, *et al.* (2019). A comparison of extended-spectrum beta-lactamase (ESBL)–producing *Escherichia coli* and *Klebsiella pneumoniae* bloodstream infections in Alberta using a provincial surveillance system. *Infection Control & Hospital Epidemiology*, 40: 388, https://doi.org/10.1017/ice.2018.353

Table 1. Comparison of ESBL-EC and ESBL-KP BSI Case Characteristics, 2013–2018

Covariate	ESBL-EC (n = 551), No. (%)	ESBL-KP (n = 42), No. (%)	P Value[a]
Male	309 (56)	28 (67)	NS
Median age (IQR)	72 (22)	67 (12)	<.05
BSI Classification			
Hospital-acquired	149 (27)	22 (52)	<.05
Healthcare-associated	197 (36)	12 (29)	NS
Community-acquired	203 (37)	8 (19)	<.05
Epidemiological Parameter			
Urinary tract	312 (76)	17 (61)	NS
Other	99 (24)	11 (39)	
Time from admission to culture, average d	9	19	<.05

Note. ESBL, extended-spectrum β-lactamase; EC, *Escherichia coli;* KP, *Klebsiella pneumoniae;* BSI, bloodstream infection; NS, not significant ($P > .05$).
[a]The Mann-Whitney U test was used to calculate *P* values for continuous variables. A test of proportions or the χ^2 test was used for categorical variables.

Acknowledgments. We acknowledge the infection control professionals and physicians of Alberta Health Services and Covenant Health for these surveillance data.

Financial support. No financial support was provided relevant to this article.

Conflicts of interest. All authors report no conflicts of interest relevant to this article.

Reference

1. Scheuerman O, Schechner V, Carmeli Y, *et al.* Comparison of predictors and mortality between bloodstream infections caused by ESBL producing *Escherichia coli* and ESBL-producing *Klebsiella pneumoniae*. *Infect Control Hospital Epidemiol* 2018;39:660–667.