immune systems, and therefore are more susceptible to sickness more often. Therefore, the older population needs to be added and thus segmented into several different age groups that will help give clear indications as far as age and its influence on contracting the flu virus, and whether or not the vaccine is effective. For example, splitting people up into age groups such as ages 0-10 years, 10-20 years, 20-30 years, and 65 years and older, along with factoring in their occupations and daily activities, and then administering the same tests used in the experiment. This will be far more efficient in figuring out how the vaccine helps or does not help people of all ages rather than just a set age group such as children.

As someone who has doubts about the flu vaccine, I believe that more evidence must be found across particular age groups to prove the quality and the effectiveness of the vaccine on individuals at all stages of life. Field mentions that his study was limited to a private pediatric practice and the information was gathered over a 1-year period.[1] To expand upon this, a larger, more diverse and broad sample must be used to show the efficacy and validity of the flu vaccine. This study was a huge step in the right direction in bringing about awareness of the influenza vaccine and its capabilities. With the changes mentioned above, the vaccine's influence can be far more prevalent and potentially prevent a contagious and frequently diagnosed respiratory disease in America.

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Conflicts of interest: None to report.

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Reasons for influenza vaccination underuse: A case-control study

To the Editor:

I appreciate the interest in my influenza vaccine study.[1] In pediatric patients, vaccine efficacy varies from year to year, and can be influenced by the type of vaccine used. This is probably also true for older individuals. My study design may not be practical for older populations other than possibly in family practice settings, but my findings could have implications for many prospective vaccine efficacy studies. The same individuals who routinely shun influenza vaccines because of personal and family experiences of not getting sick with influenza in the absence of vaccination are also likely not to enroll in a prospective vaccine trial. Those who have experienced influenza morbidity during years when they were not immunized are more likely to enroll themselves or their children in such a trial. Thus, efficacy results in many open enrollment trials may not be applicable to the general population, but rather to the relatively high-risk population that signs up for the study. More needs to be determined about individual susceptibility to viral diseases like influenza to truly understand the efficacy of vaccines intended to prevent them.

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Klebsiella pneumoniae carbapenemase-producing *Serratia marcescens* outbreak in a university hospital

To the Editor:

Serratia marcescens is an important pathogen involved in hospital-acquired infections. Outbreaks have been reported and are difficult to eradicate. In neonates, the gastrointestinal tract represents an important reservoir for cross-contamination; however, in adult hospitalized patients, the respiratory tract is more important.[1]

To date, few reports of *Klebsiella pneumoniae* carbapenemase (KPC)-producing *S marcescens* have been described in Brazil. Recently, Da Silva et al[2] described an outbreak of carbapenem-resistant *S marcescens* with a focus on coproduction of KPC-2 and IMP-10 in a Brazilian university hospital. The outbreak occurred in an intensive care unit and all isolates were classified in the same clonal profile by pulsed-field gel electrophoresis (PFGE). We faced a simultaneous *S marcescens* outbreak in another Brazilian hospital, 1 of them by a KPC-producing clone.

The outbreak occurred at Hospital Universitario Evangelico de Curitiba, a tertiary-care trauma reference hospital in southern Brazil with a total of 660 beds. All 17 isolates of *S marcescens* from 12 patients admitted to Hospital Universitario Evangelico de Curitiba September 11-October 22, 2015, were evaluated. Epidemiologic and microbiologic data of bacterial isolates are presented in Table 1. Bacterial isolates were identified and tested for antimicrobial susceptibility using the Vitek2 Compact System (bioMérieux, Durham, NC). Isolates were submitted to polymerase chain reaction for bla_{KPC} using EasyQ KPC (bioMérieux, Marcy-lÉtoile, France)

Table 1
Clinical characteristics of patients associated with *Serratia marcescens* outbreaks

N	Sex	Age	Admission day	Outcome	Day of *Serratia*	KPC-2	Site of identification	Infection	Susceptibility	Comorbidities	Previous antibiotic	Clone
Adult intensive care unit												
1	M	70	August 26, 2015	Death	September 3	Yes	Blood	Yes	AK, CIP, GEN	SAH, DM, CRF, CHF	Yes	A
2	F	81	August 31, 2015	Survival	September 17	Yes	Urine	No	AK, CIP, GEN	SAH, Stroke	Yes	A
3	M	38	Septemer 8, 2015	Survival	September 17	Yes	Tracheal aspiration	no	AK, CIP, GEN	Recurrent Pyelonephritis	Yes	A
4	M	60	August 21, 2015	Survival	September 22	Yes	Perianal swab	no	AK, CIP, GEN	SAH, abdominal aneurism	Yes	–
5	F	79	September 3, 2015	Death	September 23	Yes	Perianal swab	no	AK, CIP, GEN	Gastric neoplasm, SAH	Yes	–
6	M	31	September 17, 2015	Survival	September 28	Yes	Tracheal aspiration	no	AK, CIP, GEN	None	Yes	A
7	M	51	September 22, 2015	Survival	October 09	Yes	Tracheal aspiration	no	AK, CIP, GEN	None	Yes	A
Neonatal intensive care unit												
9	F	59 days	July 27, 2015	Survival	September 25 and October02	No	Catheter tip and ocular swab	no	AK, GEN, CIP, MEM, PIP, CPM	Neonatal sepsis	Yes	B
10	F	15 days	August 27, 2015	Death	September 11, September 13, September 16	No	Blood	Yes	AK, GEN, CIP, MEM, PIP, CPM	Neonatal sepsis	Yes	B
11	F	5 days	September 18, 2015	Survival	Sepember 25	No	Ocular swab	no	AK, GEN, CIP, MEM, PIP, CPM	Congenital toxoplasmosis	Yes	B
12	M	39 days	August 21, 2015	Survival	September 29, October23	No	Blood	Yes	AK, GEN, CIP, MEM, PIP, CPM	Neonatal sepsis	Yes	–
13	F	47 days	September 12, 2015	Survival	Sep/29, Oct/07	No	Perianal and ocular swab	no	AK, GEN, CIP, MEM, PIP, CPM	Late neonatal sepsis	Yes	-

AK, amikacin; *CI*, ciprofloxacin; *F*, female; *GEN*, gentamycin; *M*, male; MEM, meropenem; *PIP*, piperacillin/tazobactam; *CPM*, cefepime; *SAH*, systemic arterial hypertension; *DM*, diabetes mellitus; *CRF*, chronic renal failure; *CHF*, chronic heart failure.

as previously described.[3] One isolate from each patient (12 isolates) was genotyped by PFGE according to Kaufman et al.[4]

The first case of *Serratia* infection was identified September 11, 2015, in a blood culture in the neonatal intensive care unit (NICU). All patients in the NICU (19 beds) were placed under contact precautions, and surveillance cultures were collected from all patients. Two days later, *S marcescens* was identified in blood culture from a patient in an adult ICU. The units (NICU and adult ICU) are separated by 3 levels and health care workers are exclusive to each unit. Furthermore, the susceptibility profile of *S marcescens* was different between the patient in the NICU (carbapenem resistant) and the adult ICU (carbapenem susceptible). After the identification of these first cases, another 7 occurred in different units, and 5 were resistant to carbapenems. Carbapenemase-producing *Enterobacteriaceae* phenotipically identified by modified Hodge test was positive. All isolates of *Serratia* spp resistant to carbapenem were sent to molecular carbapenemase detection and presented the bla_{KPC-2} gene. Cultures from beds, furniture, water tap, sink, alcohol gel dispenser, ventilation circuits, and the nebulization system were taken. All cultures from both ICUs were negative for *Serratia*. After the last case, no more KPC-producing *S marcescens* organisms were identified in the institution at least 1 year after.

According to PFGE, 8 isolates could be categorized into 2 profiles: type A (isolates 1, 2, 3, 6, 7) was the most prevalent and found in the adult ICU (all of them harboring bla_{KPC-2} gene) and the less prevalent type B (isolates 9, 10, 11) that showed a certain similarity with the isolates of the type A profile (79%) (Fig 1). All type B isolates were carbapenem-susceptible. This fact suggests that the isolates of both profiles may have the same common ancestor and bla_{KPC-2} gene acquired by horizontal transference. Two isolates from the adult ICU and 2 from the NICU were not recovered for PFGE.

This is the second report of a KPC-producing *Serratia* outbreak in Brazil. Clonal identification was an important step during investigation of the *Serratia* outbreak, as previously described.[5] The production of carbapenemases by *Serratia* is a matter of concern due to intrinsic resistance to polymyxins. Tigecycline, aminoglycosides, and quinolones are the last options for treatment. However, the pharmacokinetic characteristics of some of these drugs are not favorable in severe infections.

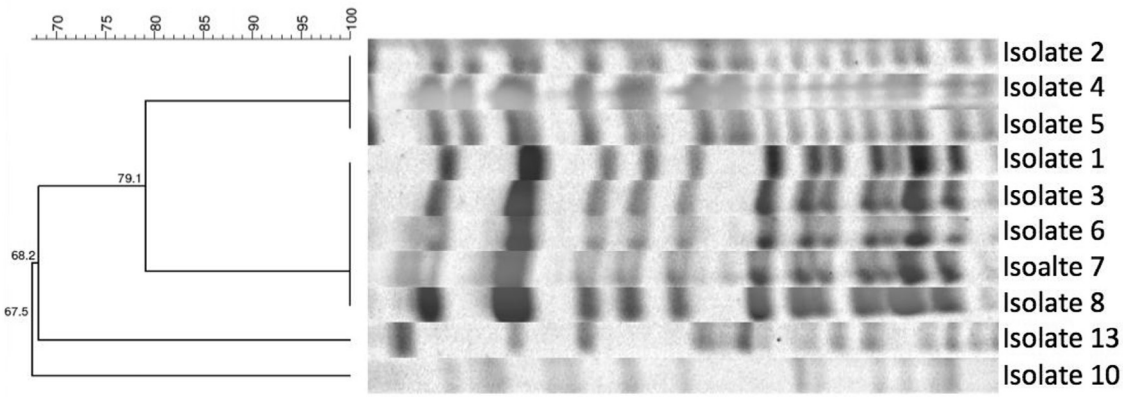

Fig 1. Pulsed field gel electrophoresis of isolates of *Serratia* spp during an outbreak. Isolates 1-7 are carbapenem-susceptible and 9-13 are carbapenem-resistant *Serratia marcescens.*

Strategies for the prevention and control of carbapenem-resistant Enterobacteriaceae are fundamental to avoid dissemination.

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