perform antimicrobial susceptibility testing, and whole-genome sequencing to identify resistance mechanisms.

Results. Among the 24 patients with culture-confirmed *Shigella* infection, 4 were hospitalized and 2 died. All isolates were multidrug-resistant (Table 1) and harbored mechanisms for resistance to ampicillin, ceftriaxone, trimethoprim-sulfamethoxazole, azithromycin, and ciprofloxacin. Fifteen patients received one course of ciprofloxacin, 5 received multiple courses of antibiotics, and 4 received no antibiotics. Overall, 6 patients had treatment failure (Table 2); all 4 patients who received azithromycin had subsequent clinical failure and 2 also had microbiologic failure. Two patients had failure after ciprofloxacin (1 clinical, 1 microbiologic).

Conclusion. This outbreak of highly resistant shigellosis highlights the importance of comprehensive susceptibility testing and systematic outcome studies. Evidence of treatment failure after azithromycin suggests that an appropriate clinical breakpoint is needed to inform clinical decision-making. Ciprofloxacin treatment failures were observed in patients with a susceptible strain harboring a resistance mechanism, warranting further investigation.

Table 1. Antibiotic susceptibility of *Shigella sonnei* outbreak isolates (n=24).

Antibiotic	Minimum inhibitory concentration (MIC)	Interpretation[1]
Amoxicillin-clavulanic acid	4–8 µg/ml	S
Ampicillin	> 32 µg/ml	R
Azithromycin	> 32 µg/ml	NS
Cefoxitin	2–4 µg/ml	S
Ceftriaxone	32–64 µg/ml	R
Ciprofloxacin	0.12 µg/ml	S
Meropenem	≤0.06 µg/ml	S
Nalidixic acid	≥32 µg/ml	R
Streptomycin[2]	32–>64 µg/ml	R
Tetracycline	≥32 µg/ml	R
Trimethoprim-sulfamethoxazole	> 4 µg/ml	R

[1] Interpretation of MIC is characterized according to Clinical and Laboratory Standards Institute (CLSI) breakpoints (S = susceptible; R = resistant; NS = non-susceptible) when available. Non-susceptibility is defined based on the epidemiological cutoff value (ECV) for *Shigella sonnei* of ≥32 µg/ml established by CLSI in 2015. The ECV should not be used as a clinical breakpoint to predict clinical effectiveness.
[2] CLSI breakpoints are not established for streptomycin; interpretive standards used are NARMS-established breakpoints for resistance monitoring and should not be used to predict clinical efficacy.

Table 2. Patients with treatment failures during an outbreak of multidrug-resistant shigellosis (n=6).

Age (years)	Sex	Antibiotic course (listed in sequence given)[1]	Clinical failure following completion of antibiotics?	Days to diarrhea resolution[2]	Microbiologic failure following completion of antibiotics?	Days to negative culture or PCR[3]
90	F	Azithromycin 250–500mg PO QD x4d[4]	Yes	6	Unknown[5]	-
		Ceftriaxone 1000mg IV QD x5d[4]	Yes	5	Unknown[5]	-
		Ampicillin 500mg IV QD x5d[4]	Yes	3	No	8
88	M	Ceftriaxone 1000–2000mg IV QD x3d	Yes	1	Unknown[5]	-
		Ciprofloxacin 250mg PO BID x3d	No	-1	No	12
88	F	Ciprofloxacin 500mg PO BID x3d	Yes	4	No	7
82	F	Azithromycin 500mg PO TID x3d	Yes	2	No	11
79	F	Azithromycin 500mg IV QD x2d[4]	Yes	12	Yes	-
		Ceftriaxone 1000–2000mg IV QD x4d[4]	Yes	9	Yes	43
42	M	Azithromycin 500mg PO QD x3d	Yes	3	Yes	-
		Ciprofloxacin 500mg PO BID x3d	No	-14	Yes	-
		Ciprofloxacin 500mg PO BID x3d	No	-25	No	1

[1] Median days between courses was 1 day (range 0–15).
[2] Days from end of antibiotic course to end of diarrhea (defined as ≥3 loose stools per day).
[3] Days from end of antibiotic course to first negative culture obtained. Daily cultures following antibiotic completion were not obtained for any patients; therefore these values should not be interpreted as days to microbiologic cure.
[4] Administered concurrently.
[5] Patients who did not have cultures between antibiotic courses are listed as having unknown microbiologic outcomes for these courses.

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1604. Response to a Cluster of *Haemophilus influenzae* Serotype A Cases in a Small Alaska Community, 2018

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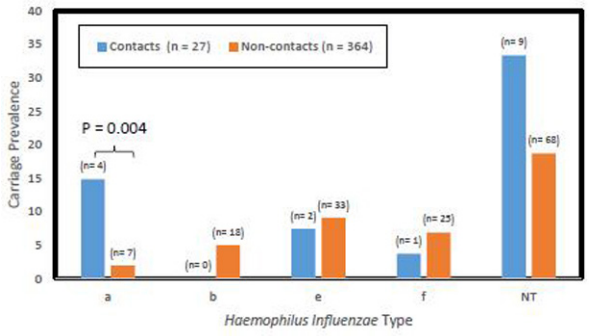
Background. Between May and July 2018, four invasive cases of *Haemophilus influenzae* type a (Hia) occurred in a remote Alaska community. A public health response was performed to prevent further illness and to understand local Hia transmission.

Methods. The team identified close contacts of the Hia patients, collected oropharyngeal (OP) swabs and provided prophylactic rifampin. Close contacts were persons who spent ≥4 hours with a Hia patient for ≥ 5 of the 7 days preceding hospitalization. Five days later, OP swabs were collected community-wide and prophylactic rifampin was offered to community members aged <10 years. Eight weeks later, OP swabs were collected from all willing community members. Samples were tested using PCR and culture to identify Hi carriage.

Results. No Hia cases occurred in this community after the response. The pretreatment carriage prevalence is shown in Figure 1. There was a significant difference in prevalence of Hia carriage between contacts (4/27, 14.8%) and non-contacts (7/364, 1.9%) ($P = 0.0043$). Contacts aged <10 years were significantly more likely to carry Hia compared with contacts aged ≥10 years (11/18 [61.1%] vs. 3/34 [8.8%], $P = 0.0001$). The case households had the highest proportion of individuals who carried Hia at any time, with 54%–60% of individuals in three case households carrying Hia at least once. Hia carriage was eliminated in two carriers who completed treatment and were tested immediately after rifampin prophylaxis. Testing 8 weeks later found that the prevalence of carriage did not significantly change in the contacts (5/42 [11.9%] to 6/25 [24%], $P = 0.18$) or the non-contacts (7/368 [1.9%] to 2/114 [1.8%], $P = 0.47$).

Conclusion. Children aged <10 years who had close contact with the Hia patients were the most likely to carry Hia. These findings suggest that people who do not have close contact do not benefit from prophylaxis as they have very low Hia carriage. While rifampin prophylaxis eliminated carriage of Hia in the short term, carriage prevalence did not change in the long term. Further research is needed to understand why contacts have such a high prevalence of carriage even after receiving appropriate prophylactic medication.

Figure 1: *Haemophilus influenzae* carriage prior to antimicrobial treatment, Alaska 2018.



Disclosures. **All authors:** No reported disclosures.

1605. Temperature Modulates the Rate of Increase of Antibiotic Resistance Across Europe

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Background. Widely recognized as a major public health threat globally, the rapid increase of antibiotic resistance in bacteria could soon render our most effective method to combat infections obsolete. Factors influencing the burden of resistance in human populations remain poorly described, though temperature is known to play an important role in mechanisms of bacterial growth and transmission.