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BRIEF REPORT

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Similar environmental survival patterns of *Streptococcus pyogenes* strains of different epidemiologic backgrounds and clinical severity

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The spectrum of *Streptococcus pyogenes* (group A streptococci) infections and complications includes asymptomatic carriage, throat infection, and acute rheumatic fever, localized skin, soft tissue or bone infections, and invasive spread with positive blood cultures accompanied by toxic shock leading to rapid death [1–5]. The contagiousness of these *S. pyogenes* infections has been studied extensively [1–3] and the contribution of environmental sources has been considered [1, 5]. Following a nosocomial outbreak at our hospital due to an *S. pyogenes* strain [4] in which some findings paralleled those from earlier MRSA outbreaks [6], we decided to examine the survival of *S. pyogenes* strains in the environment to ascertain whether extended environmental survival contributes to the organism's spread, as noted for a number of MRSA outbreak strains at our hospital [7]. Thus, several *S. pyogenes* strains of different epidemiological backgrounds and clinical severity were selected, and the survival behavior of each was evaluated.

All of the *S. pyogenes* strains studied were diagnosed at the Atrium Medical Centre (AMC) and the German National Reference Laboratory for Streptococci at the Department of Medical Microbiology at the Rheinisch-Westfälische Technische Hochschule (RWTH) in Aachen, Germany. They were all obtained from clinical cases, and the cases reflected a wide spectrum of clinical severity or epidemiological behavior. The strains were divided into two groups and four patient subgroups: group A included strains from serious invasive infections (i.e., bacteremia, sepsis, including the manifestation of toxic shock syndrome), with subgroup 1 (strains 1 and 2) being nosocomial and subgroup 2 (strains 3 and 4) non-nosocomial; group B included strains from less serious non-invasive soft tissue or wound infections, with subgroup 3 (strains 5 and 6) being nosocomial and subgroup 4 (strains 7 and 8) non-nosocomial. *S. pyogenes* strains 2, 6 and 8 were isolated from different patients during a hospital outbreak reported previously by Davies et al. [4]. In Table 1 of that report the respective patients were assigned the codes G, P1 and M1.

The influence of desiccation on the survival of the different *S. pyogenes* strains was evaluated and compared as described previously in detail for MRSA [7]. Suspensions containing approximately 10^8 cfu/ml were prepared in sterile phosphate-buffered saline (PBS; pH7.2). Samples (1 ml) of each suspension were transferred to 50-ml flat-bottomed glass bottles and allowed to dry. All bottles were plugged with cotton wool to allow free communication with the hospital environment through indirect northern light, ambient temperature and relative humidity. The fluid component of the suspensions had completely evaporated after 10 days, and sampling was begun 4 days later. Remaining viable bacteria were recovered by adding 1 ml of PBS to the bottle. After vigorous vortexing in the closed bottle, the suspension was flooded onto a blood agar plate and incubated for 48 h at 37°C. For all strains, remaining colony forming units were measured at 1–2-day intervals until extinction. The average relative humidity of the ambient air and temperature during the study period were 31% and 23°C, respectively.

The survival rates of the different groups of *S. pyogenes* strains are shown in Table 1. It can be seen that from an initial measurement of approximately 10^8 cfu the strains died off rapidly, with the decline ranging from 4 to 7-log^{10} cfu during the 14-day dry-out period to counts between 20 and 9,000 cfu. After day 14, only 2 more weeks passed until the last viable *S. pyogenes* strain was extinct. A gradual die-off pattern was noted for all strains within a range of up to circa 2-log^{10} cfu at the same measurement points. The last day on which a viable count was measured for each strain was between day 24 and day 30. The nosocomial outbreak strains of subgroups 1 and 3 did not survive any longer than the non-outbreak strains in subgroups 2

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Table 1 Environmental survival (cfu) of *Streptococcus pyogenes* strains isolated from cases of varying clinical severity with a nosocomial (subgroups 1 and 3) or non-nosocomial (subgroups 2 and 4) epidemiology

Strain characteristic	Group A (virulent strains)				Group B (non-virulent strains)			
	Subgroup 1		Subgroup 2		Subgroup 3		Subgroup 4	
	Strain 1	Strain 2	Strain 3	Strain 4	Strain 5	Strain 6	Strain 7	Strain 8
Type	M1, T1, speA	M9, TB3264	M12, T12, speC	M3, T3, speA	M28, T28, speA, speC	MNT, T25	M22–60, T12, speA, speC	M28, T28
Anatomic origin	Blood	Blood	Blood	Blood	Soft tissue	Wound	Soft tissue	Wound
Nosocomial	Yes	Yes	No	No	Yes	Yes	No	No
Day of measurement								
1	10^8	10^8	10^8	10^8	10^8	10^8	10^8	10^8
14	350	4000	9000	4200	4000	700	20	4000
15	190	3500	3300	2980	3000	590	10	3100
16	180	2500	2200	2910	2100	120	10	2100
17	20	2200	180	490	2000	110	10	3000
18	0	330	0	710	320	20	10	280
19	20	380	0	650	170	60	20	140
20	0	410	20	460	530	320	0	860
21	40	210	60	860	920	40	10	240
22	140	620	80	800	660	40	0	750
23	90	0	50	780	980	130	0	80
24	20	140	60	590	280	120	0	430
25	0	0	10	120	10	40	10	170
26	0	0	0	140	10	20	0	180
27	80	0	90	190	20	10	0	110
28	70	0	0	0	10	0	10	70
30	0	0	20	0	0	0	0	30
33	0	0	0	0	0	0	0	0
35	0	0	0	0	0	0	0	0
37	0	0	0	0	0	0	0	0

and 4. There was also no difference in the survival patterns exhibited by the virulent (group A) strains causing serious invasive infections (subgroups 1 and 2) and those of the less serious non-invasive (group B) strains (subgroups 3 and 4). In our approach the outcome was simple: no *S. pyogenes* isolate survived on glass for longer than 1 month.

The rapid decline of all *S. pyogenes* strains tested—even our own outbreak strain that had demonstrated MRSA-like spread [4]—contrasts sharply with the prolonged survival of around a year reported previously for epidemic MRSA strains [7]. We did not find any survival characteristics that could clearly be correlated with a specific outbreak character. *S. pyogenes* strains thus seem to be disseminated in a fashion similar to *S. aureus*, with airborne spread playing a predominant role, supported by (intermediate) carriers via dispersal on skin scales from a carriage site or via direct transmission from hands or inanimate objects. Environmental contamination was noted particularly in the outbreak related to strain no. 5, and MRSA-like spread was noted in the outbreak related to strain no. 2. The severity of disease caused by the various infecting strains did not correlate with any alternative or specific survival pattern.

The potential danger of a contaminated environment has been recognized in earlier outbreaks [1, 5], and control measures aimed at removing dust and disinfecting surfaces were consequently implemented at our hospital during the outbreaks. Although the 4-week survival period found for our *S. pyogenes* strains in the hospital environment is shorter than the period of 3 months reported by Lidwell and Lowbury [8], it should be noted that their study measured survival in dust. Since the influence of various dust mixtures can be surprisingly variable [7], we chose not to include dust samples in our investigational approach.

Our finding that *S. pyogenes* strains survive in the inanimate environment for up to 1 month shows that contact transmission is facilitated in the short-term phase of an outbreak; however, long-term environmental survival cannot be considered an important factor in the dynamics of *S. pyogenes* transmission. The remarkable paucity of reports on the environmental survival of *S. pyogenes* strains could be related to the increasing interest in the behavior of other bacteria in the hospital environment, such as multiresistant pathogens, like MRSA [7, 9], vancomycin-resistant enterococci, *Clostridium difficile* or *Acinetobacter baumannii* [9], and the coronavirus causing severe acute respiratory syndrome. Investigation of the last syndrome has identified the survival of the pathogen in fomites as a factor possibly related to transmission [10]; thus, multiple pathways must be considered for transmission of all pathogens, including *S. pyogenes*.

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