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 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; pH 7.2). 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
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) |
|-----------------------|----------------------------|--------------------------------|
|                       | 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        | 2000    |
| 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      |
| 29                    | 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*. 
References

1. Backhouse CI, Cartwright RY (1974) An outbreak of streptococcal skin sepsis in a closed community. Brit Med J 3:497–499
2. Efstratiou A (2000) Group A streptococci in the 1990s. J Antimicrob Chemother 45:3–12
3. Stevens DL (1992) Invasive group A streptococcus infection. Clin Infect Dis 14:2–13
4. Davies BI, Hirsch J, Werink TJ, Toenbreker H, Bainczijk F, Leeuwen van WJ (1999) A Streptococcus pyogenes outbreak caused by an unusual serotype of low virulence: the value of typing techniques in outbreak investigations. J Infect 38:185–190
5. Cruickshank JG, Lightfoot NF, Sugars KH et al (1982) A large outbreak of streptococcal pyoderma in a military training establishment. J Hygiene 89:9–21
6. Wagenvoort JHT (2000) Dutch measures to control MRSA and the expanding European Union. Eurosurv 5:26–28
7. Wagenvoort JHT, Sluijsmans W, Penders RJR (2000) Better environmental survival of outbreak vs. sporadic MRSA isolates. J Hosp Infect 45:231–234
8. Lidwell OM, Lowbury EJ (1950) The survival of bacteria in dust. J Hygiene 48:21–27
9. Talon D (1999) The role of the hospital environment in the epidemiology of multi-resistant bacteria. J Hosp Infect 43:13–17
10. Wenzel RP, Edmond MB (2003) Listening to SARS: lessons for infection control. Ann Int Med 139:592–593