The work was funded by the Molecular Biology and Biotechnology Division of the Nigerian Institute of Medical Research, Yaba, Lagos, Nigeria. The secretarial services of Mrs. Onwudimegwu is also appreciated.

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Bacterial contaminants in cosmetic products from a patient with cystic fibrosis

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A small bacteriological study is performed on several personal cosmetic items, which had been in current and regular use by a young adult with cystic fibrosis (CF) who had a history of chronic *Pseudomonas aeruginosa* chest infection and a recent history of *Burkholderia cepacia* genomovar IIIa chest infection. The aim of this small study is to determine the bacteriological diversity of the cosmetics, with particular reference to these two Gram-negative pathogens, and to assess the significance of these in terms of infection control issues for the patient and other patients in the CF unit.

Personal cosmetic products (n=24) that had been in regular and current use were obtained from an 20-year-old lady with a history of CF who had a previous history of chronic pulmonary infection with *P. aeruginosa* and a more recent history of infection with *B. cepacia* genomovar IIIa. The cosmetic products included the following items: blusher, brushes (n=4), concealer, glitter, highlighter, illuminator, liquid latex, lip gloss, sponge, make-up (n=8), make-up pencil, mascara (n=2) and theatrical blood.

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Bacteriological analysis was performed on each item by sampling using one of three methods. For brushes, the item was brushed (x5) against the shaft of a sterile swab and above a standard plate of *B. cepacia*-selective agar (BCSA; Mast Diagnostics, Bootle, Wirral, Merseyside, UK). For solid make-up, a standardised swabbing technique was used, involving premoistening of the swab with tryptone soya broth (TSB; Oxoid CM0129, Oxoid, Basingstoke, England) before aggressively swabbing the surface of the cosmetic product. For cosmetic material, aseptic removal of approximately 0.5 g material into 9 mL TSB was performed.

All swabs were inoculated directly on the surface of BCSA medium and were then enriched non-selectively in TSB (9 mL) at 37°C for 48 h. BCSA medium was incubated at 37°C for 48 h and then at ambient temperature on the bench for up to five days. All TSB enrichment broths were incubated at 37°C for 24 h, and then 20 mL was streaked individually on both BCSA and Columbia blood agar (Oxoid CM331) containing 5% (v/v) defibrinated horse blood, and incubated at 37°C for 48 h. Resulting colonies were purified on CBA and then identified using molecular methods, as described previously by Xu *et al*.[1]

None of the cosmetic items were positive for *B. cepacia* or *P. aeruginosa* and no culturable bacteria were detected in 21/24 (87.5%) of the items. Four cosmetic items were positive for Gram-positive organisms, as detailed in Table 1.

P. aeruginosa is the most important bacterial pathogen in patients with CF,[2] as demonstrated by high-prevalence data in most of the national CF registries. Chronic *P. aeruginosa* colonisation of the major airways, which leads to debilitating exacerbations of pulmonary infection, is the major cause of morbidity and mortality in patients with CF; hence, it is important to be able to detect *P. aeruginosa* from patients' sputum.

Recently, Emerson *et al*.[3] published their findings of a US Cystic Fibrosis Foundation (CFF) registry-based study, which showed that infection related to *P. aeruginosa* is a major predictor of morbidity and mortality. The eight-year risk of death parameter is 2.6 times higher in patients who had positive sputum cultures for this organism, and they have a significantly lower percentage predicted forced expiratory volume (FEV1). These workers suggested that early interventions may help decrease associated morbidity and mortality of young patients with CF.

More recently, Rosenfeld e*t al*.[4] described the patho-physiology and risk factors for early *P. aeruginosa* infection in

Table 1. Molecular identification employing 16S rDNA PCR and automated sequencing of four bacterial isolates from a variety of cosmetic products used by a patient with cystic fibrosis.

Cosmetic item	Identification of contaminating bacterial organism
Highlighter	*Corynebacterium tuberculostearicum*
Eye pencil shavings	*Staphylococcus epidermidis*
Blusher	*Bacillus* sp.
Facial sponge	*Bacillus* sp.

CF. They suggest that chronic lower airway infection with *P. aeruginosa* is associated with significant morbidity and mortality among CF patients. However, they also suggest that first acquisition of *P. aeruginosa* does not appear to cause an immediate and rapid decline in lung function, as early isolates are generally non-mucoid, antibiotic-sensitive and present at low densities. This suggests a possible window of opportunity for early intervention.

To date, there have been no reports on the bacterial composition of cosmetic products. Furthermore, the survival dynamics of problem Gram-negative pathogens in cosmetics are not known. Thus, further work is required to determine the survival and persistence of Gram-negative pathogens, including *P. aeruginosa* and *B. cepacia*, in these matrices. Until such studies report, it is recommended that patients with CF avoid sharing their cosmetic products among each other, as a precautionary measure, to help eliminate the potential for cross-infection with these pathogens.

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Potentially misleading Western blot results in Lyme disease diagnosis

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The laboratory diagnosis of Lyme disease is complex[1] and serology remains the technique of choice. Recommended practice is a two-step process involving a sensitive screening enzyme immunoassay (EIA) followed by a more specific confirmatory Western blot for all EIA-positive and equivocal samples and for EIA-negative samples with a high clinical suspicion of Lyme disease (e.g., tick bite and erythema migrans).[1,2] However, Western blot results require careful interpretation.

The National Lyme Disease Testing Service Laboratory, Raigmore Hospital, Inverness, tests over 3000 samples annually from across Scotland, many of which are from complex clinical cases. It was noted recently that serum from a patient with confirmed parvovirus B19 infection cross-reacted with the in-house *Borrelia burgdorferi* IgG Western blot. This could lead to the wrong interpretation of Western blot results. This study aims to discover if other viral infections produce similar results with the *B. burgdorferi* IgG Western blot.

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The study group consisted of six patients found to contain IgM antibodies to parvovirus B19 infection (parvovirus B19 IgM EIA, Biotrin International), six with cytomegalovirus (CMV) IgM (CMV IgM EIA, Microgen Bioproducts) and six with Epstein-Barr virus (EBV) IgM (EBV VCA IgM EIA, Diasorin). All patients had good clinical evidence of viral infection (Table 1).

The 18 serum samples were analysed by commercial *B. burgdorferi* IgG/IgM EIA (Zeus Scientific, New Jersey, USA) and an in-house *B. burgdorferi* IgG Western blot. The EIA was performed according to the manufacturer's instructions and the Western blot was performed as described previously.[2]

The blots used contained sodium dodecyl sulphate-extracted *B. burgdorferi* (i.e., antigen). A commercial positive control (Zeus Scientific) and in-house positive control, with defined band patterns, were included in each blot run. The number, intensity and molecular weight of bands observed with each sample were recorded, and a negative, equivocal, weak positive or positive result assigned according to predetermined criteria.[3] The Western blot was repeated for all samples.

Previous or subsequent serum samples from five patients (serum samples 5, 8. 9, 11 and 12 [Table 1]) who reacted with the *B. burgdorferi* IgG Western blot were also tested by the *B. burgdorferi* IgG Western blot to determine *B. burgdorferi* status.

All samples tested were EIA negative (Table 1). However, as the infections studied may produce clinical symptoms similar to Lyme disease (such as a rash), a Western blot may be performed on EIA-negative samples, based on clinical suspicion.[2,3] Western blot may also be performed on EIA-negative samples if they have been referred from another laboratory for confirmation.

Some of the polypeptides from a *B. burgdorferi* extract may react with patient antibodies that are not specific to *B. burgdorferi*.[4] These include the 41 kDa flagellin polypeptide, which, although it must be detected for a positive diagnosis, is known to cross-react with antibodies to proteins from other bacteria.[4,5] Other polypeptides in this category include those of 44, 56, 62, 64, 72 and 82 kDa. In contrast, bands with 18, 22, 32, 34, 39, 46, 58 and 92 kDa polypeptides are generally accepted as specific for *B. burgdorferi*.[1,6]

Three of six parvovirus B19 IgM-positive serum samples reacted with specific *B. burgdorferi* antigens and the 41-kDa antigen (Table 1). The antibody response to parvovirus B19 is directed mainly against the two structural proteins of the viral capsid (VP1 [83 kDa] and VP2 [58 kDa]).[7,8] Although the molecular weights of these polypeptides are different from the *B. burgdorferi*-specific bands detected here (Table 1), they may share sufficient homology at particular epitopes for parvovirus B19-specific antibody to bind with *B. burgdorferi*-specific polypeptides.

Five of six CMV-IgM positive sera produced equivocal or