Background We identified secondary infection of Buruli ulcer (BU) wounds as a cause of healing delay. In order to contribute to the improvement of wound management and reduction of healing delay, we initiated a study to gain understanding of the possible routes of infection and also characterised the resistant profiles of Gram negative bacteria isolated from the wounds of patients attending two health facilities in Ghana.

Methods Staphylococcus aureus isolates were characterised by the spa gene, mecA and the Pantone Valentine Leukocidin (PVL) toxin followed by spa sequencing and whole genome sequencing of a subset of isolates. Phenotypic antibiotic susceptibility testing of Gram negative clinical isolates was performed and multidrug-resistant Pseudomonas aeruginosa identified. The Enterobacteriaceae were further investigated for ESBL and carbapenem production, and some resistance conferring genes were analysed by PCR.

Results Twenty-four isolates were identified as methicillin-resistant S. aureus (MRSA), and lukFS genes encoding PVL were identified in 67 isolates. Typing and sequencing of the spa gene from 91 isolates identified 29 different spa types with t355 (ST152), t186 (ST88), and t346 dominating. While many distinct strains were isolated from both health centres, genotype clustering was identified within centres pointing to possible health care-associated transmission. Phylogenomic analysis confirmed these clusters. Among the GNB, phenotype screening showed widespread resistance to ampicillin, chloramphenicol, ticarcillin-clavulanic acid, cefuroxime and sulphonamethoxazole-trimethoprim. ESBL production was confirmed in 15 isolates phenotypically while 61.5% of screen-positive isolates harboured at least one ESBL-conferring gene. Carbapenem encoding genes were detected in 41% of the isolates.

Conclusions Our findings indicate that the health-care environment likely contributes to superinfection of BU wounds and calls for training in wound management and infection control techniques. The observed frequency of ESBL and carbapenem resistance indicates the need to set up surveillance networks and strictly enforce policies which guide the rational use of antibiotics.