# Nasal Carriage, Antimicrobial Susceptibility Profile, and Enterotoxin Genes of *Staphylococcus aureus* Isolated from Children with Asthma

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## Abstract

Asthma is a chronic respiratory disease that affects children worldwide. Increasing evidence suggests that *Staphylococcus aureus* contributes to the pathology of asthma. The aim of this study was to evaluate the nasal carriage, antimicrobial susceptibility profile, and presence of enterotoxin genes from *S. aureus* isolated from children with asthma. Nasal swab samples were collected from 158 children, including 98 children with asthma and 60 healthy controls. *S. aureus* isolates were identified using phenotypic methods and the presence of the *nuc* gene. Antimicrobial susceptibility testing was performed using the Kirby-Bauer disc diffusion method. Polymerase chain reaction (PCR) confirmed the presence of the *mec*A gene and enterotoxin genes. The *nuc* gene was confirmed in 83 isolates, resulting in a nasal carriage of 52.5% (83/158). The nasal carriage of *S. aureus* was higher among asthma cases (OR 0.201, 95% CI 0.063–0.645, *p* = 0.007). Methicillin-resistant *S. aureus* (MRSA) nasal carriage was 11.4%. The *S. aureus* isolates showed high resistance to cefoxitin (99%) and penicillin (92%) but were

sensitive to gentamicin (25%). Furthermore, 67.5% of the isolates were multi-drug resistant. The staphylococcal enterotoxin c gene (*sec*) was the most prevalent enterotoxin (19.7%) among cases and controls. These findings highlight the need for improved antibiotic stewardship in paediatric medicine and implementation of infection control policies.

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### Contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Oyewumi Oshamika, Oreoluwa Sonowo, Olatunde Odusote and Yeside Akinbolagbe. The first draft of the manuscript was written by Oyewumi Oshamika, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Below is the link to the electronic supplementary material.

### Supplementary file1 (DOCX 534 kb)

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Oshamika, O., Sonowo, O., Akinbolagbe, Y. *et al.* Nasal Carriage, Antimicrobial Susceptibility Profile, and Enterotoxin Genes of *Staphylococcus aureus* Isolated from Children with Asthma. *Indian J Microbiol* (2024). https://doi.org/10.1007/s12088-024-01272-z

#### **Download citation**

- Received17 October 2023
- Accepted24 March 2024
- Published18 April 2024
- DOIhttps://doi.org/10.1007/s12088-024-01272-z

#### Keywords

- Nasal carriage
- <u>Staphylococcus aureus</u>
- Asthma
- Antibiotic resistance
- <u>MRSA</u>

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