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Staphylococcus Strains in Atopic Dermatitis in Children: Toxins Production and Resistance Properties

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Abstract: *Staphylococcus spp.* skin colonization is involved in the pathogenesis of atopic dermatitis (AD). While coagulase-positive *Staphylococcus (S.) aureus* strains are known to worsen symptoms, the role of coagulase-negative staphylococci (CoNS) remains controversial. Further research is needed to clarify the pathogenicity of CoNS in AD patients. A study involving 329 children with AD (mean age: 4.89 years) assessed the frequency of staphylococcal colonization on affected skin, along with the toxin-producing properties and antibiotic resistance of isolated strains. Mild AD: Predominantly colonized by CoNS (especially *S. epidermidis*). Moderate/Severe AD: Showed a significant increase in *S. aureus* colonization. CoNS (including *S. epidermidis*) could produce enterotoxins (A, B, C) and toxic shock syndrome toxin-1 (TSST-1), though less frequently than *S. aureus* strains. In severe AD, toxin-producing CoNS strains (particularly enterotoxin A producers) were more common than non-toxin-producing strains, suggesting a possible aggravation of inflammation via superantigen-mediated mechanism. CoNS exhibited higher resistance rates than *S. aureus*. Methicillin-resistance *S. epidermidis* (MRSE): 23.4%. Methicillin-resistance *S. aureus* (MRSA): 1.27%. This indicates that CoNS in AD may act as a reservoir for antibiotic resistance genes, complicating treatment. CoNS may contribute to AD pathogenesis through toxin production (exacerbating inflammation) and antibiotic resistance (limiting treatment options). Severe AD may involve a synergistic effect between *S. aureus* and toxin-producing CoNS.

Keywords: atopic dermatitis; *Staphylococcus epidermidis*; *Staphylococcus aureus*; enterotoxins; toxic shock syndrome toxin-1; antibiotic resistance; children

1. Introduction

Atopic dermatitis (AD) is a chronic inflammatory skin disorder with a complex, heterogeneous etiology, involving impaired skin barrier function, intradermal and systemic T-lymphocyte activation, and increased susceptibility to cutaneous infections [1]. Typically, skin of AD patients is colonized by *Staphylococcus (S.) aureus*, a coagulase-positive staphylococcus (CoPS) [2,3]. *S. aureus* exacerbates AD through expression of virulence factors that trigger disease flares [4], contribution to chronic relapsing course of the disease and potential resistance to anti-inflammatory corticosteroid treatment [5]. Factors promoting *S. aureus* colonization in AD include Th2/Th17 cytokines

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overexpression, dysregulation of antimicrobial peptides (e.g. HNP1 and β-defensins), microbial dysbiosis, and skin barrier defects [6]. Bacterial toxins further perpetuate inflammation by altering interleukin secretion, creating a self-sustaining inflammatory cycle [7]. CoNS (e.g. *S. epidermidis*) also colonize the affected skin. Their role remains poorly understood and debated [8].

S. epidermidis, a predominant member of CoNS, is a key component of the normal skin microbiota. During AD flares, *S. epidermidis* colonization increases compared to other commensals [9,10], suggesting a potential compensatory role in suppressing *S. aureus* overgrowth. *S. epidermidis* can modulate host immune responses and prevent pathogenic microorganism invasion by stimulating keratinocytes to produce endogenous antimicrobial peptides and by independently secreting bacteriocins with a potent antimicrobial activity [11]. In murine AD models *S. epidermidis*-derived vesicles downregulate pro-inflammatory genes (TNFα, IL1β, IL6, IL8 and iNOS), upregulate human β-defensins 2 and 3, and enhance resistance to *S. aureus* colonization [12]. Additionally, *S. epidermidis* produces specific compounds such as serine proteases and phenol-soluble modulins that inhibit biofilm formation and restrict *S. aureus* colony growth [13]. These observations support the potential protective role of CoNS in AD [14].

Despite its beneficial roles, *S. epidermidis* possesses virulence factors that may exacerbate AD. It has the ability to produce enterotoxin that function as superantigens [15]. Pathogenicity islands, containing genes for staphylococcal enterotoxin B (SEB) have been identified in *S. epidermidis* phages [16]. Staphylococcal superantigens can stimulate Th2 lymphocytes to produce interleukin (IL)-31, which suppress filaggrin expression, increase pro-inflammatory cytokines production, activate basophils, and induce intense pruritus [17,18]. Staphylococcal enterotoxins may also act as allergens [19]. Clinical studies have shown positive correlations between *S. epidermidis* colonization density, serum anti-SEB IgE levels, and higher SCORAD indices [20]. Nevertheless, no studies have directly confirmed enterotoxin production by CoNS strains isolated from AD patients' skin. The potential adverse effects of *S. epidermidis* and other CoNS on the course of AD can be aggravated by their multi-drug-resistant properties [21,22]. Our study aims to provide a more detailed investigation of these potentially harmful characteristics of CoNS and compare them with those of *S. aureus* strains.

2. Materials and Methods

We conducted an observational cross-sectional study at the outpatient department of the University Children's Clinical Hospital, First Moscow State Medical University. Potential participants were initially identified by pediatricians, with subsequent evaluation by allergists and a dermatologists to confirm AD diagnosis using the Hanifin and Rajka criteria and assess eligibility.

The study included children aged 2 to 18 years with either recently or previously diagnosed AD, regardless of disease duration, severity or comorbid allergic conditions. Key inclusion criteria required visible AD lesions in both antecubital fossae, with disease severity classified using SCORAD index into: mild AD (SCORAD ≤ 25), moderate (25 – 50) and severe AD (≥50). Exclusion criteria comprised: age <2 years; active skin infection; immunodeficiency disorders, recent (within 1 month) use of immunosuppressants (including oral corticosteroids), systemic/topical antibiotics, topical anti-inflammatory medications or moisturizers.

Trained allergist/dermatologists obtained bilateral antecubital fossa swabs for *Staphylococcus spp.* identification. Samples were processed using MicroScan WalkAway plus System (Beckman-Coulter, Inc.) for microbial identification and antibiotic susceptibility testing.

Isolated staphylococcal strains were placed on a liquid nutrient medium with enzymatic casein hydrolysate and 1.0% of brain-heart infusion. Subsequent cultivation was performed on a rotary shaker at 210 rpm for 24 hours at 37°C. For cultivation, 50 ml tubes were used, into which 4.5 ml of culture medium were added. Bacterial cells were removed by centrifugation at 10.000 rpm for 15 minutes and obtained supernatant was heated for 30 minutes at 100°C. Detection of staphylococcal enterotoxin C (SEC) was conducted using a double diffusion method in a gel with monospecific serum to the SEC. Detection of staphylococcal enterotoxin A (SEA), and staphylococcal enterotoxin B (SEB) was determined with an enzyme-linked immunoassay test kit with a sensitivity of 2.0 ng/ml for SEA and 1.0 ng/ml for SEB. Toxic shock syndrome toxin 1 (TSST-1) was determined by using an enzyme immunoassay kit with a sensitivity of 10.0 ng/ml [23,24].

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We evaluated colonization patterns (CoNS vs. CoPS), toxin production profiles, antibiotic resistance rates with comparative analysis across AD severity groups.

3. Results

3.1. Microbial Isolation Patterns

Our study included 329 children with AD (median age 4.89 years, IQR 2–18). *Staphylococcus spp.* colonization was identified in 244 participants (74.4%), yielding 300 isolated staphylococcal strains.

- *S. aureus*: 160 strains (53.3%)
- CoNS: 140 strains (46.6%)
  - *S. epidermidis:* 87 (29.0%)
  - *S. haemolyticus*: 22 (7.3%)
  - *S. hominis:* 15 (5.0%)
  - *S. capitis:* 7 (2.3%)
  - *S. warneri:* 5 (1.7%)
  - *S. cohnii:* 2 (0.7%)
  - Single isolates of *S. simulans* and *S. saprophyticus*

Fifty-two patients (15.8%) demonstrated polymicrobial colonization, with the following associations:

- Most frequent: *S. aureus* + *S. epidermidis* (n=29)
- Moderate frequency: S. epidermidis + S. haemolyticus (n=5), *S. aureus* + *S. haemolyticus* (n=4), *S. epidermidis* + *S. hominis* (n=4)
- Rare associations (n=1 each): S. *aureus* + *S. saprophyticus/capitis*, *S. epidermidis* + *S. warneri/cohnii/capitis*, *S. haemolyticus* + *S. capitis*
- Triple colonization: *S. aureus* + *S. epidermidis* + *S. haemolyticus/hominis*

No staphylococcal growth was observed in 85 cases (25.8%).

Mean SCORAD index in patients with *S. aureus* skin colonization was 54.0±4.9, with CoNS skin colonization was 41.4±6.3, and with no staphylococcal growth was 43.2±6.4. In patients *S. aureus* + CoNS co-colonization, the mean SCORAD index was 51.7±4.6. Statistical comparisons revealed significantly higher SCORAD among patients with *S. aureus* skin colonization compared with CoNS skin colonization or no staphylococcal growth ($p=0.0066$ and $p=0.011$).

Among patients with mild AD, CoNS represented the predominant skin colonizers ($p<0.05$). In moderate AD cases, we observed comparable detection rates of CoNS and *S. aureus* ($p<0.05$). The colonization pattern shifted markedly in severe AD, where *S. aureus* became the predominant species ($p<0.05$). Statistical analysis revealed two significant trends in the severe AD subgroup: a progressive decline in CoNS detection alongside a concurrent increase in *S. aureus* colonization (both $p<0.05$). Patients showing no staphylococcal growth were predominantly diagnosed with mild AD (Table 1).

Table 1. Demographics of AD patients and *Staphylococcus spp.* skin colonization.

	Atopic dermatitis patients			
	Mild	**Moderate**	**Severe**	**Total**
Patients n(%)	68(20.6%)	124(37.7%)	137(41.6%)	329
Male n(%)	43(25.8%)	53(31.9%)	70(42.1%)	166
Female n(%)	25(15.3%)	71(43.5%)	67(41.1%)	163
Age (yr)	2.9±0.6	5.3±0.9	5.5±3.1	4.89±0.9
SCORAD	15.1±4.2	38.8±4.7	71.1±6.5	47.8±5.1
Staphylococci strais	61(20.4%)	109(36.3%)	130(43.3%)	300
S. aureus	22(13.7%)	55(34.3%)	83(51.8%)	160(53.3%)
CoNS total	39(27.8%)	54(38.5%)	47(33.5%)	140(46.6%)
S. epidermidis	24(27.5%)	39(44.8%)	24(27.5%)	87
S. haemolyticus	4(18.18%)	8(36.36%)	10(45.45%)	22
S. hominis	9(60.0%)	4(26.6%)	2(13.3%)	15

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S. warneri	1	1	3	5
S. saprophyticus	1			1
S. capitis			7	7
S. simulans		1		1
S. cohnii		1	1	2
No staphylococcal growth	20(23.5%)	33(38.8%)	32(37.6%)	85

3.2. Toxin-Producing Properties of CoNS vs CoPS

The toxin-producing properties of 83 staphylococcal strains were investigated, including 32 CoPS (*S. aureus*) and 51 CoNS. The CoNS group comprised 30 *S. epidermidis*, 8 *S. haemolyticus*, 7 *S. hominis*, 3 *S. warneri*, 2 *S. capitis*, and 1 *S. simulans* strain. All strains exhibited the ability to produce at least one toxin, with many strains producing multiple toxins simultaneously.

Among *S. aureus* strains, 78.1% (25/32) produced more than one toxin: 18.75% (6/32) synthesized two toxins, 46.9% (15/32) three, and 12.5% (4/32) expressed all four tested toxins. Similarly, 47.0% (24/51) of CoNS strains produced several toxins: 9.8% (5/51) produced two, 31.4% (16/51) three, and 5.8% (3/51) all four toxins. Non-toxigenic strains were observed in 9.0% (3/32) of *S. aureus* and 15.6% (8/51) of CoNS isolates, with no statistically significant difference ($p > 0.05$).

SEB and TSST-1 were the most frequently detected toxins in staphylococcal filtrates. In undiluted samples, SEB was identified in 87.9% of *S. aureus*, 73.3% of *S. epidermidis*, and 66.7% of other CoNS strains, with no significant difference in detection rates. However, at a 1:10 dilution, SEB was significantly more prevalent in *S. aureus* (63.6%) compared to *S. epidermidis* (16.6%) and other CoNS (23.8%) ($p<0.001$). These results indirectly indicate the ability of *S. aureus* to produce SEB in greater amounts compared to other staphylococci strains.

For TSST-1, no significant difference was observed between *S. aureus* (87.9% undiluted, 69.7% at 1:10) and *S. epidermidis* (66.7% undiluted, 53.3% at 1:10), indicating comparable production levels. However, other CoNS strains exhibited significantly lower TSST-1 detection rates (47.61% undiluted, 28.57% at 1:10) compared to *S. aureus* ($p< 0.01$). Neither SEB nor TSST-1 was detectable in the filtrates of staphylococci strains at a 1:50 dilution.

SEA was the second most prevalent toxin. No significant differences were observed in undiluted filtrates among *S. aureus* (78.8%), *S. epidermidis* (73.3%), and other CoNS (76.2%). At a 1:10 dilution, SEA remained highly detectable in *S. aureus* (75.6%) and *S. epidermidis* (70.0%), but its prevalence dropped in other CoNS (47.6%, $p< 0.05$ vs. *S. aureus*). Notably, in S. aureus filtrates SEA retained detectable even at 1:50 dilution (24.2%), whereas in S. epidermidis filtrates SEA was not detected at this dilution. In a single *S. simulans* strain filtarate (4.7% of other CoNS) SEA was detected at 1:50, suggesting that some CoNS strains can generate SEA in quantities comparable to *S. aureus*.

SEC was the least frequently detected toxin. It was more prevalent in *S. aureus* (39.4%) than in *S. epidermidis* (10.0%, $p< 0.01$) or other CoNS (19.0%). SEC detection in diluted filtrates was not performed (Figure 1).

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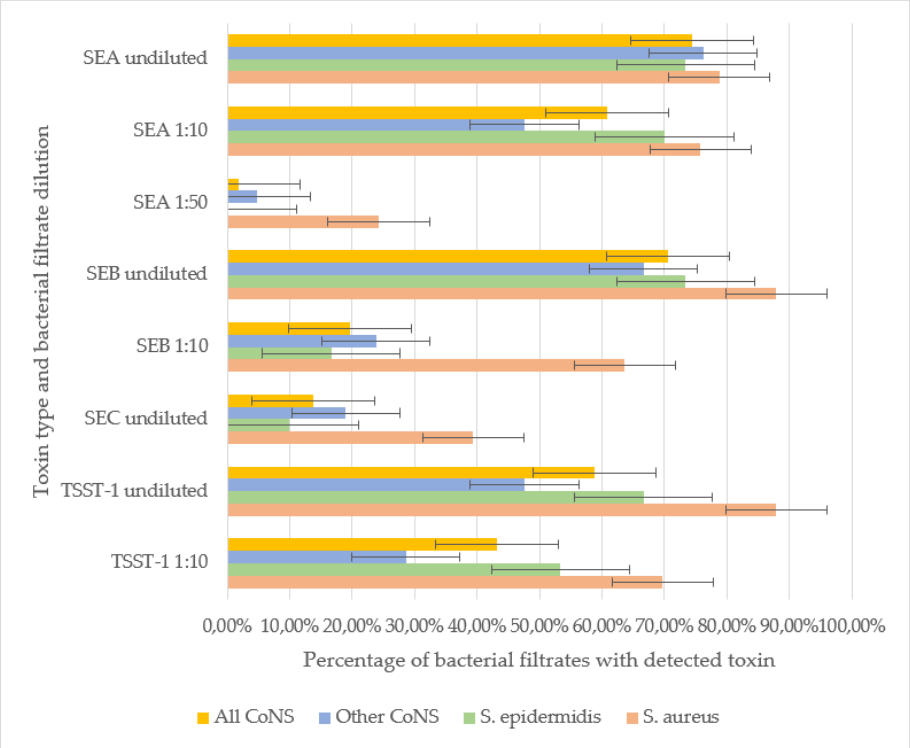
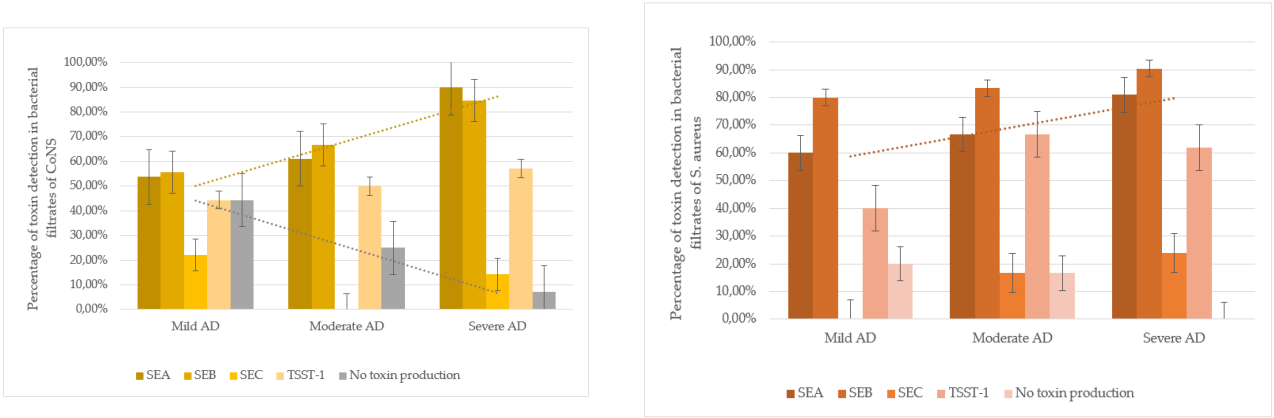


Figure 1. Toxin-producing properties of *Staphylococcus spp*.

An intriguing trend was observed in CoNS strains isolated from atopic dermatitis (AD) patients: SEA production correlated with disease severity. In mild AD, 53.8% (7/13) of CoNS strains produced SEA; in moderate AD, 61.1% (11/18); and in severe AD, 90.0% (18/20). The difference between mild and severe AD cases was statistically significant ($p < 0.05$). Additionally, non-toxigenic CoNS strains were more frequent in mild AD (44.4%, 4/9) than in moderate (25.0%, 3/12) or severe AD (7.1%, 1/14), with a significant difference between mild and severe cases ($p< 0.05$). These findings suggest that severe AD is associated with higher colonization by SEA-producing CoNS and lower colonization by non-toxigenic strains (Figure 2).



(**a**) (**b**)

Figure 2. *Staphylococcus spp.* toxins production and AD severity: (**a**) CoNS strains (including *S. epidermidis*): toxin-producing properties were studied in 51 strain (mild AD – 13, moderate AD – 18, severe AD - 20). In mild AD CoNS produced SEA less often compared to severe AD group ($p<0.05$). The number of non-toxin-producing strains was significantly higher among mild AD compared to severe AD ($p<0.05$). (**b**) *S. aureus* strains: toxin-producing properties were studied in 32 strains (mild AD – 5, moderate AD – 6, severe AD - 21). The strains had pronounced toxic properties, regardless of the severity of the disease.

3.3. Antibiotic Susceptibility of CoNS vs CoPS

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The antibacterial susceptibility of 160 *S. aureus* strains, 87 *S. epidermidis* strains, and 53 other CoNS strains (excluding *S. epidermidis*) was evaluated. The study assessed sensitivity to the following antibacterial agents: β-lactam antibiotics including penicillins (benzylpenicillin, ampicillin, amoxicillin-clavulanate, oxacillin), cephalosporins (cefazolin, cefepime), and carbapenems (imipenem); macrolides (azithromycin, clarithromycin, erythromycin); fluoroquinolones (ciprofloxacin, ofloxacin, levofloxacin, moxifloxacin); glycopeptides (vancomycin); lincosamides (clindamycin); rifamycins (rifampicin); tetracyclines (tetracycline); amphenicols (chloramphenicol); sulfonamides with trimethoprim (co-trimoxazole); aminoglycosides (gentamicin); and oxazolidinones (linezolid) (Figure 3).

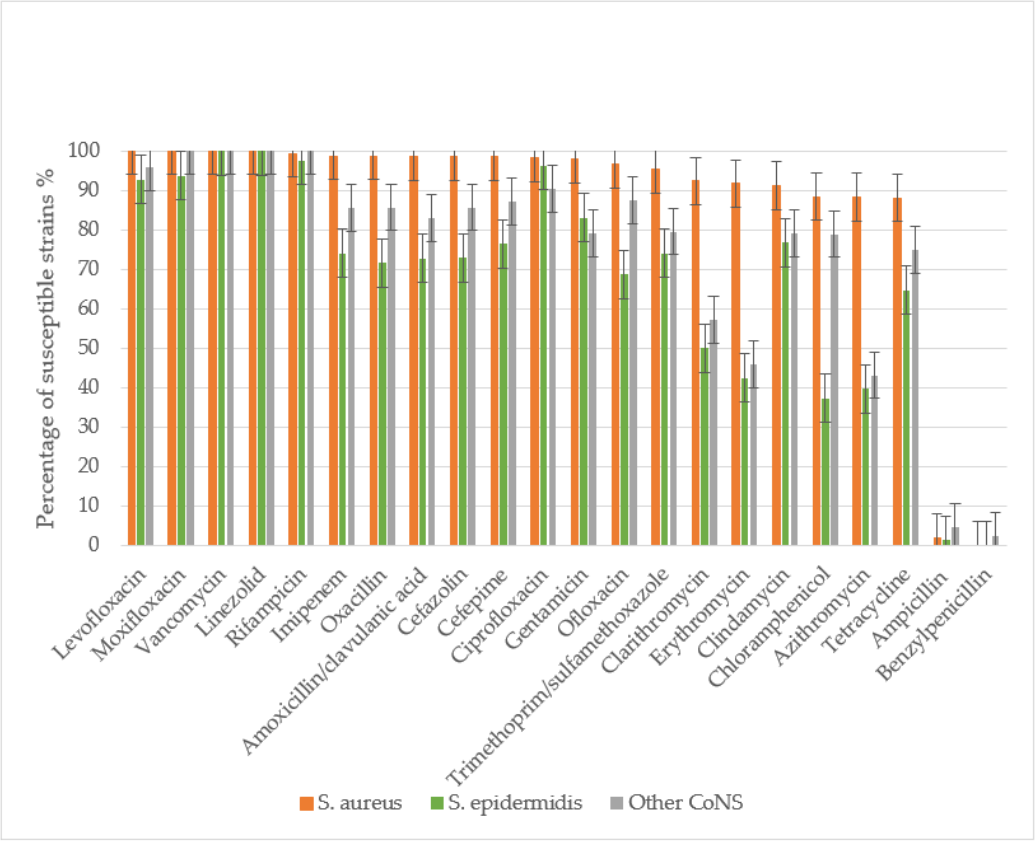

Figure 3. *Staphylococcus spp.* antibiotic susceptibility in descending order for *S. aureus*. *S. aureus* demonstrated absolute sensitivity to vancomycin, linezolid, and later-generation fluoroquinolones (levofloxacin and moxifloxacin), with a low MRSA prevalence of 1.27% as determined by oxacillin resistance. CoNS, especially *S. epidermidis*, showed significantly more resistant properties to most antibiotics. The incidence of MRSE was 28.4%.

All *S. aureus* and *S. epidermidis* strains demonstrated complete resistance to benzylpenicillin, with only minimal sensitivity retained to ampicillin. Resistance rates to ampicillin were 97.9% for *S. aureus*, 98.9% for *S. epidermidis*, and 95.9% for other CoNS. *S. aureus* maintained high sensitivity to amoxicillin/clavulanate, with only 1.38% of strains showing resistance. The prevalence MRSA was low at 1.27%. In contrast, *S. epidermidis* and other CoNS exhibited significantly higher resistance to amoxicillin/clavulanate (27.2% and 17.03%, respectively), with methicillin-resistant strains reaching 28.4% (MRSE) and 14.2% ($p<0.01$ compared to *S. aureus*).

S. aureus showed high susceptibility to cephalosporins, with resistance to both cefazolin and cefepime observed in only 1.27% of strains. Significantly higher resistance rates were noted in CoNS: 27.0% (*S. epidermidis*) and 14.3% (other CoNS) to cefazolin, and 23.5% and 12.8% to cefepime, respectively.

CoNS demonstrated substantially greater resistance to macrolides than *S. aureus* ($p<0.01$). Resistance rates among *S. epidermidis* ranged from 50% (clarithromycin) to 60.3% (azithromycin), while other CoNS showed 42.8-56.8% resistance across macrolides. In contrast, *S. aureus* resistance

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remained low (8.16-11.62%), though still higher than its resistance to β-lactam/β-lactamase inhibitor combinations and cephalosporins.

For fluoroquinolones, *S. aureus* maintained high susceptibility, with only 1.86-3% resistance to second-generation agents (ofloxacin, ciprofloxacin) and complete sensitivity to third- and fourth-generation fluoroquinolones (levofloxacin, moxifloxacin). CoNS showed significantly higher resistance, particularly *S. epidermidis* (31.25% to ofloxacin). Resistance to newer fluoroquinolones was lower (3.71-7.32% in *S. epidermidis*, 4.17-12.5% in other CoNS), with all CoNS remaining susceptible to moxifloxacin.

All staphylococcal strains retained sensitivity to reserve antibiotics vancomycin and linezolid. Resistance to rifampicin and imipenem was rare in *S. aureus* (0.63-1.26%) but more prevalent in CoNS (25.97% of S. epidermidis and 14.59% of other CoNS to imipenem). While 2.46% of *S. epidermidis* showed rifampicin resistance, other CoNS maintained complete susceptibility.

CoNS exhibited significantly greater resistance to most antibiotic classes ($p<0.05$), including β-lactams, macrolides, cephalosporins, tetracyclines, aminoglycosides, lincosamides, and fluoroquinolones. No association was found between disease severity (mild, moderate, severe) and antibiotic resistance patterns for any staphylococcal species when tested against all studied antimicrobial agents.

4. Discussion

Our study revealed distinct patterns of staphylococcal colonization in atopic dermatitis (AD) patients. *S. aureus* colonization was significantly more prevalent in severe AD cases, while CoNS strains, particularly *S. epidermidis*, dominated in mild AD. Interestingly, the presence of mixed staphylococcal associations did not correlate with disease severity. These colonization patterns likely reflect specific phases of the local inflammatory process, where compromised skin barrier function shifts the microbial balance from CoNS predominance to *S. aureus* dominance. Under normal conditions, innate immune mechanisms effectively control pathogenic bacterial growth. However, reduced antimicrobial peptide production by keratinocytes in AD permits uncontrolled staphylococcal proliferation, potentially driving disease progression.

S. epidermidis is more likely to be isolated from the affected skin in mild AD. During exacerbation of AD, either because of the ability of staphylococci to form biofilms or because *S. epidermidis* is displaced by *S. aureus*, the latter become more numerous. The association of bacteria is detected in a certain phase of AD, perhaps at a transient point when colonization by *S. epidermidis* or other CoNS shifts toward massive growth of *S. aureus*. In severe AD, *S. aureus* colonization predominates over CoNS strains. The latter produces toxins more intensively in patients with severe AD compared to CoNS strains detected on the skin of patients with mild AD. Notably, in severe AD, we observed not only increased S. aureus colonization but also enhanced toxin production by residual CoNS strains, likely due to reduced microbial competition and impaired immune regulation.

Both CoPS and CoNS demonstrated capacity for simultaneous production of toxins-superantigens (SEA, SEB, SEC, TSST-1), with our study documenting higher enterotoxigenic activity among CoNS than previously reported [25]. This finding raises important questions about the role of CoNS in AD pathogenesis, particularly *S. epidermidis*, whose impact remains controversial [26]. Our data from patients across the AD severity spectrum suggest that specific S. epidermidis strains may actively influence disease progression. While traditionally viewed as a natural antagonist of *S. aureus*, certain *S. epidermidis* strains appear less capable of inhibiting *S. aureus* virulence in AD patients [28]. Genomic analyses reveal strain-specific differences in histopathological potential, antibiotic resistance profiles (including methicillin resistance), and immunomodulatory capacity [29].

Phylogenetic studies show striking differences between staphylococcal species in AD: while *S. aureus* exhibits clonal expansion, *S. epidermidis* demonstrates remarkable phylogenetic diversity across all disease stages. Mild AD cases predominantly harbor clades A29 and A30 strains, contrasting with the A20 dominance in healthy adults [25]. Notably, AD skin often lacks protective CoNS strains (*S. epidermidis* and *S. hominis*) that produce anti-*S. aureus* antimicrobial peptides [29]. Instead, AD-derived *S. epidermidis* strains exhibit pathogenic potential through multiple mechanisms:

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S. epidermidis strains isolated from lesional AD skin exhibit distinct pathogenic properties that significantly alter epidermal structure and function. Unlike commensal strains from healthy skin, AD-associated preferentially activates STAT6 while suppressing the protective AhR/OVOL1 pathway, accompanied by significantly reduced indole production ($p<0.01$) [30]. These changes lead to marked downregulation of key differentiation markers, including filaggrin (FLG) and desmoglein-1, compromising epidermal barrier function. AD-derived *S. epidermidis* strains exhibit other proinflammatory properties. Production of cytotoxic phenol-soluble modulins (PSMs) that show a strong positive correlation with disease severity ($r=0.78$, $p<0.001$) [31]. Secretion of the cysteine protease EcpA, which effectively degrades both desmoglein-1 (by 62±8%) and the antimicrobial peptide LL-37 in vitro, thereby impairing physical barrier function and promoting skin inflammation [32]. Generation of extracellular serine protease (Esp) that triggers IL-13 activation (3.5-fold increase) and drives a Th2-polarized immune response, characteristic of AD pathogenesis [33]. These findings collectively demonstrate that specific S. epidermidis strains possess multiple virulence factors capable of exacerbating AD through both direct barrier disruption and immune modulation. Not all *S. epidermidis* strains can inhibit *S. aureus* biofilm formation. In some patients, these species coexist, forming mixed-species biofilms that enhance their survival and pathogenicity [26].

Our study revealed a high prevalence of antibiotic resistance among CoNS. A 2017 species-level analysis of AD flares demonstrated that MRSA was more common in severe cases, whereas MRSE predominated in milder forms [25]. Supporting this, a 2023 genome-wide association (GWA) study reported MRSA in 13.79% and MRSE in 39% of cases, revealing shared plasmids between *S. epidermidis* and *S. aureus* that confer antibacterial resistance [16]. These findings underscore the possible involvement of CoNS in AD pathogenesis.

5. Conclusions

This study highlights that CoNS, particularly *S. epidermidis*, may play a more significant role in AD than previously thought, especially in severe cases. Certain strains can impair the skin barrier and exacerbate inflammation. Our results emphasize the need for further research on staphylococcal dynamics in AD and the potential for targeted microbial interventions. Specifically, alongside conventional anti-inflammatory therapy, future treatments should consider agents that modulate bacterial colonization—suppressing pathogenic strains while restoring beneficial flora during both active disease and remission. This study advances our understanding of AD etiology and may guide novel therapeutic strategies.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available on request from the corresponding author due to privacy reasons.

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Abbreviations

The following abbreviations are used in this manuscript:

AD	Atopic dermatitis
CoNS	Coagulase-negative staphylococci
MRSA	Methicillin-resistant *Staphylococcus aureus*
MRSE	Methicillin-resistant *Staphylococcus epidermidis*
CoPS	Coagulase-positive staphylococci
SEA	Staphylococcal enterotoxin A
SEB	Staphylococcal enterotoxin B
SEC	Staphylococcal enterotoxin C
TSST-1	Toxic shock syndrome toxin 1

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