Identification of \textit{vat}(E) in \textit{Enterococcus faecalis} Isolates from Retail Poultry and Its Transferability to \textit{Enterococcus faecium}

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Sixteen isolates of \textit{Enterococcus faecalis} were recovered from retail poultry samples (seven chickens and nine turkeys) purchased from grocery stores in the greater Washington, D.C., area. PCR for known streptogramin resistance genes identified \textit{vat}(E) in five \textit{E. faecalis} isolates (three isolates from chickens and two isolates from turkeys). The \textit{vat}(E) gene was transmissible on a ca. 70-kb plasmid, along with resistance to erythromycin, tetracycline, and streptomycin, by conjugation to \textit{E. faecalis} and \textit{Enterococcus faecium} recipient strains. DNA sequencing showed little variation between \textit{E. faecalis} \textit{vat}(E) genes from the chicken samples; however, one \textit{E. faecalis} \textit{vat}(E) gene from a turkey sample possessed 5 nucleotide changes that resulted in four amino acid substitutions. None of these substitutions in the \textit{vat}(E) allele have previously been described. This is the first report of \textit{vat}(E) in \textit{E. faecalis} and its transferability to \textit{E. faecium}, which indicates that \textit{E. faecalis} can act as a reservoir for the dissemination of \textit{vat}(E)-mediated streptogramin resistance to \textit{E. faecium}.

Enterococci form part of the normal host flora of both the human and the animal gastrointestinal tract, where they seldom cause serious infections. However, it has been well documented that enterococci are etiological agents of endocarditis and urinary tract sepsis in humans (9). Of increasing concern is the rapid emergence of enterococci as the third leading cause of nosocomial infections in the hospital setting, surpassed only by \textit{Staphylococcus aureus} and coagulase-negative staphylococci (15, 16). As a result, enterococci have elonquently been referred to as the “pathogens of the 90s” (7). Approximately 85 to 90% of enterococcal infections are attributed to \textit{Enterococcus faecalis} and 5 to 10% are attributed to \textit{Enterococcus faecium}. Infections caused by other \textit{Enterococcus} species (\textit{E. durans}, \textit{E. avium}, \textit{E. raffinosus}, \textit{E. gallinarum}, and \textit{E. caseliflavus}) occasionally emerge and have warranted attention (16).

Infections caused by multidrug-resistant enterococci are usually treated with the glycopeptide vancomycin. However, as the use of vancomycin has steadily increased, so, consequently, has the incidence of vancomycin-resistant enterococci (21). Recent reports suggest that almost one-quarter of enterococci isolated from patients in intensive care units in the United States are vancomycin resistant (11, 12). Quinupristin-dalfopristin (Synercid), a semisynthetic mixture of the compounds streptogramins A and B, was recently approved for use for the treatment of vancomycin-resistant enterococci in both the United States and Europe (18, 19). Vancomycin-resistant, another mixture of the compounds streptogramins A and B, has been used as a growth promoter in animal production for over two decades. The use of antimicrobials in the animal production environment has the potential of selecting for resistant zoonotic bacterial pathogens, and it has been speculated that the extensive use of virginiamycin in animal husbandry may have contributed to the emergence of quinupristin-dalfopristin resistance among human gram-positive pathogens (5, 6; L. B. Jensen, L. B., A. M. Hammerum, F. M. Aerestrup, A. E. van den Bogaard, and E. E. Stobberingh, Letter, Antimicrob. Agents Chemother. 42:3330-3331, 1998). Consequently, the use of virginiamycin has been banned in the European Union since July 1999 (1). However, despite the 1998 ban of virginiamycin in Denmark, a recent report showed a 22.5% prevalence of virginiamycin-resistant \textit{E. faecium} isolates from pigs in 2000 (1). Although there is no clear explanation for this, it has been suggested that the presence of resistance to other antimicrobial agents such as erythromycin, tetracycline, and streptomycin may coselect for virginiamycin resistance (1). Resistance to streptogramins was first reported in staphylococci in 1980 (4). Only resistance to the A component is required; however, resistance to both the A component and the B component may result in higher MICs (4). To date a number of genes that confer streptogramin A resistance in both staphylococci and \textit{E. faecium} have been reported (2, 4, 18, 22, 23; K. V. Singh, B. M. Jonas, G. M. Weinstock, and B. M. Murray, Abstr. 41st Intersci. Conf. Antimicrob. Agents Chemother., p. 103, 2001). \textit{E. faecalis} is intrinsically resistant to streptogramin antibiotics, although the mechanism(s) underlying this resistance has yet to be fully described (13; Singh et al., 41st ICAAC). However, preliminary data suggest that an efflux pump, designated ABC23, may play a role in conferring quinupristin-dalfopristin resistance in \textit{E. faecalis} (Singh et al., 41st ICAAC). The amino acid sequence of the ABC23 efflux pump appears to show high degrees of similarity (41 to 64%) to those of the ABC proteins of \textit{Lactococcus lactis}; \textit{Bacillus subtilis}; \textit{Streptococcus pneumoniae}; \textit{Msr}(C) of \textit{E. faecium}; and \textit{Vga}(A), \textit{Vga}(B), and \textit{Msr}(A) of \textit{Staphylococcus aureus} (Singh et al., 41st ICAAC). To date, none of the streptogramin resistance genes found in staphylococci or \textit{E. faecium} have been identified in \textit{E. faecalis}.

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The aim of the present study was to determine if any of the known staphylococci or Enterococcus faecium streptogramin resistance genes are present in E. faecalis isolated from retail poultry samples.

MATERIALS AND METHODS

Sample collection and isolation of enterococci. Prepackaged retail chicken and turkey samples were collected from four supermarket chains in the Maryland suburbs of Washington, D.C., in June 1999. The packaging was aseptically opened, and the meat samples were rinsed in 50 ml of sterile buffered peptone water. One milliliter of the rinse suspension was plated in 10 ml of Enterococcus (BBL) broth, and the mixture was incubated at 45°C for up to 48 h. Esculin-positive cultures were streaked onto Enterococcus agar, and the plates were incubated at 35°C for 24 h. A single colony characteristic of Enterococcus spp. was picked from each plate and streaked onto Trypticase soy agar with 5% sheep blood to ensure purity and to check for hemolysis. Single colonies were picked from blood agar plates and streaked onto brain heart infusion agar. Suspected enterococci and Gram stains of the enterococci were used for catalase and pyrimidase production. Catalase-negative, gram-positive, pyrimidase-negative isolates were confirmed as Enterococcus spp. with the AccuProbe Enterococcus identification test (Gen-Probe, Inc.). AccuProbe-positive isolates were identified to the species level with the Automated Microbial Identification system (bioMerieux Vitek, Inc.).

Antimicrobial susceptibilities of enterococci. The MICs of antimicrobials for enterococci were determined with the Sensititre Automated Antimicrobial Susceptibility system (Trick Diagnostic Systems, Westlake, Ohio) and were interpreted according to National Committee for Clinical Laboratory Standards (NCCLS) guidelines for broth microdilution methods (10). All 16 isolates were resistant to tetracycline (NCCLS breakpoint, >8 μg/ml) and from 75% of the chicken sample isolates showed resistance to erythromycin (NCCLS breakpoint, >8 μg/ml). All 16 isolates were resistant to tetracycline (NCCLS breakpoint, >16 μg/ml). None of the isolates were resistant to penicillin (NCCLS breakpoint, >16 μg/ml). Twenty-two percent of the turkey sample E. faecalis isolates and 14% of the chicken sample E. faecalis isolates showed high-level resistance to gentamicin (NCCLS breakpoint, >500 μg/ml). All of the turkey sample E. faecalis isolates and 42% of the chicken sample E. faecalis isolates were resistant to streptomycin. The susceptibility profiles of the 16 E. faecalis isolates are shown in Table 1.

PCR studies and DNA sequencing. Significant resistance genes were amplified by PCR from 5 of the 16 (31%) E. faecalis isolates. Three (E)-positive isolates (isolates CVM 3478, CVM 3972, and CVM 3973) were recovered from chicken sample E. faecalis isolates, and two (E)-positive isolates (isolates CVM 3476 and CVM 3477) were recovered from turkey sample E. faecalis isolates. No other known streptogramin resistance determinants were detected in any of the chicken or turkey sample E. faecalis isolates. Two of the chicken sample E. faecalis isolates and 43% of the turkey sample E. faecalis isolates were resistant to streptomycin and two of the turkey sample E. faecalis isolates were also (E) positive by PCR (Table 1). In contrast, ermB was detected in 100% of the (E)-positive chicken sample E. faecalis isolates but was not detected in any of the (E)-positive turkey sample isolates. Among the E. faecalis isolates negative for (E) by PCR, ermB was detected in 75% of the chicken sample E. faecalis isolates and 85% of the turkey sample E. faecalis isolates. One chicken sample E. faecalis isolate was positive for both ermA and ermB; however, this isolate was negative for (E) by PCR. None of the isolates tested positive for ermC by PCR (Table 1).

Even though ermB and (E) reside in the same isolate in two chicken sample E. faecalis isolates that were PCR positive for (E) and ermB (isolates CVM 3972 and CVM 3973), we were unable to show (E)-ermB linkage on a transposon-like element, as described by Jensen et al. (Letter, Antimicrob.
TABLE 1. Antimicrobial susceptibility profiles, PCR data, and PFGE grouping of the 16 E. faecalis isolates recovered from retail meats*

<table>
<thead>
<tr>
<th>CVM strain no.</th>
<th>Source</th>
<th>PFGE group</th>
<th>Genotypes</th>
<th>MIC (μg/ml)</th>
<th>SYN</th>
<th>VIR</th>
<th>ERY</th>
<th>TET</th>
<th>PEN</th>
<th>GEN</th>
<th>STREP</th>
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<td>T</td>
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<td>−</td>
<td>−</td>
<td>16</td>
<td>&gt;4</td>
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<tr>
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<td>16</td>
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<td>1</td>
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<td>−</td>
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<td>&gt;4</td>
<td>&gt;8</td>
<td>&gt;16</td>
<td>2</td>
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<td>3476</td>
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<td>&gt;4</td>
<td>0.5</td>
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<td>3477</td>
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<td>E</td>
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<td>+</td>
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<td>66</td>
<td>100</td>
<td>0</td>
<td>22</td>
<td>100</td>
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</tbody>
</table>
% Resistant     |        |            |           |             | 16  | >4  | 0.5 | >16 | 2   | 64  | >1,000 |
| 3461           | C      | F          | −         | −           | 8   | >4  | >8  | >16 | 2   | 64  | >1,000 |
| 3467           | C      | G          | −         | −           | 16  | >4  | >8  | >16 | 2   | 2,048| 128   |
| 3468           | C      | H          | −         | −           | 16  | >4  | >8  | >16 | 2   | 64  | >1,000 |
| 3478           | C      | I          | +         | +           | 16  | >4  | >8  | >16 | 2   | 64  | 128   |
| 3971           | C      | J          | +         | +           | 16  | >4  | >8  | >16 | 2   | 64  | 128   |
| 3972           | C      | J          | +         | +           | 32  | >4  | >8  | >16 | 2   | 64  | 128   |
| 3973           | C      | J          | +         | +           | 16  | >4  | >8  | >16 | 2   | 64  | 128   |
% Resistant     |        |            |           |             | 100 | 100 | 85  | 100 | 0   | 14  | 42    |

* Abbreviations: SYN, quinupristin-dalfopristin; VIR, virginiamycin; ERY, erythromycin; TET, tetracycline; PEN, penicillin; GEN, gentamicin; STREP, streptomycin; C, chicken; T, turkey. All vat(E)-positive E. faecalis strains were able to transfer vat(E) to an E. faecalis or E. faecium recipient strain.

Agents Chemother. 44:2231-2232, 2000), in these two isolates. These findings indicate that vat(E) and ermB may not be close to each other on the plasmids found in these two isolates.

DNA sequencing confirmed the presence of vat(E) PCR products; however, interestingly, several variations between the vat(E) DNA sequences indicating allelic variation were detected. The vat(E) sequences of the two turkey sample E. faecalis isolates varied from the original vat(E)-1 sequence originally described (GenBank accession no. AF242872). Strain CVM 3476 had a single vat(E) mutation, A152→G (Ile15→Val). In contrast, turkey sample E. faecalis strain CVM 3477 possessed five point mutations resulting in four amino acid substitutions and one silent mutation. These changes were G40→T (Ala14→Ser), G16→C (Lys56→Gln), G51→T (Glu17→Asp), T56→A (Val19→Asp), and G273→T (Ser91→Ser). Less sequence divergence was observed between the vat(E) genes from chicken sample E. faecalis isolates. The vat(E) sequences of two of the chicken sample E. faecalis isolates were identical to the vat(E)-1 sequence originally described; however, the sequence of vat(E) from E. faecalis CVM 3478 varied by a single base, resulting in a single amino acid change, A13→G (Ile13→Val). This was the same base change seen with turkey sample E. faecalis isolate CVM 3476. All the amino acid changes are outlined in Fig. 1.

PFGE. PFGE of the 16 E. faecalis isolates identified a total of 10 different PFGE patterns, which were arbitrarily assigned to groups A through J (Table 1). The nine turkey sample E. faecalis isolates were distinguishable from the seven chicken sample E. faecalis isolates by PFGE. The two vat(E)-positive turkey sample isolates were indistinguishable from each other by PFGE (PFGE group E), even though they came from different meat samples from different stores. Of the three vat(E)-positive chicken sample isolates, two belonged to PFGE group I, while one belonged to PFGE group J.

Plasmid analysis. Analysis of the five E. faecalis isolates positive for vat(E) by PCR confirmed the presence of either three to four plasmids ranging in size from ca. 5 to ca. 70 kb. Turkey sample isolates CVM 3476 and CVM 3477 and chicken sample isolate CVM 3478 each had three plasmids that appeared to be of the same size, ca. 70, 15, and 5 kb, respectively. The remaining two chicken sample E. faecalis isolates (isolates CVM 3972 and CVM 3973) appeared to have the same plasmid profile outlined above; in addition, they also carried a ca. 7-kb plasmid.

Conjugation studies. Conjugation studies demonstrated that all five vat(E)-positive E. faecalis isolates were able to transfer the vat(E) streptogramin resistance determinant to both E. faecalis and E. faecium recipient strains. Conjugation frequencies for the transfer of vat(E) varied from 1×10⁻² to 8×10⁻² per recipient for both E. faecalis and E. faecium recipients.

Ten transconjugants per mating were selected and subjected to plasmid analysis and antimicrobial susceptibility testing. PCR analysis confirmed the presence of the vat(E) genes in the transconjugants. The plasmid profiles among the transconjugants varied, the ca. 70-kb plasmid was present in all transconjugants examined; however, the full complement of plasmids identified in donor strains was never observed in any of the transconjugants analyzed (data not shown). In each case, the MICs of quinupristin-dalfopristin, erythromycin, tetracycline, and streptomycin were the same for the transconjugants and the original parent strain.

DISCUSSION

Enterococci are an increasing problem in human medicine, as the array of antibiotics available for the treatment of enterococcal infections is diminishing. This is compounded by the fact that enterococci are intrinsically resistant to a large number of antibiotics and can rapidly acquire and disseminate resistance genes. Quinupristin-dalfopristin was approved for use in the United States in 1999 for the treatment of glyco-
peptide-resistant *E. faecium* infections. However, it is not efficacious against *E. faecalis*, as this species of *Enterococcus* is intrinsically resistant to the streptogramins.

To our knowledge the present study is the first to report the presence of the streptogramin resistance gene *vat*(E) in *E. faecalis*. This is surprising since one might expect that a species already intrinsically resistant to an antibiotic class would not acquire additional resistance determinants against that class of antibiotics. In the present study, besides *vat*(E), no other streptogramin resistance determinants were identified in *E. faecalis*. The fact that no other streptogramin resistance determinants were identified is an important observation, as reports from Europe have identified *vat*(D) and *vgb*, in addition to *vat*(E), in *E. faecium* (19; Jensen et al., Letter, Antimicrob. Agents Chemother. 42:3330-3331, 1998). As the present study evaluated only 16 *E. faecalis* isolates, the ability to detect other possible resistance determinants was limited.

In addition, the three *vat*(E)-positive chicken sample *E. faecalis* isolates also harbored the *ermB* gene; however, the turkey sample *vat*(E)-positive *E. faecalis* isolate tested negative for *ermB* by PCR. Of the *vat*(E)-negative *E. faecalis* turkey isolates, 85% were positive for *ermB* by PCR. Jensen et al. (Letter, Antimicrob. Agents Chemother. 44:2231-2232, 2000) previously demonstrated that *vat*(E) and *ermB* are linked on the same plasmid in 70% of *E. faecium* poultry isolates from Denmark. However, in the present study we did not find linkage of *vat*(E)-*ermB* as part of a possible transposon-like element. Despite this, the fact that both *vat*(E) and *ermB* are present in the same strain, and in some cases on the same plasmid, may help to explain the acquisition of *vat*(E) by *E. faecalis* as a result of selective pressure from macrolide use in poultry.

Examination of the *vat*(E) DNA sequences obtained from *E. faecalis* isolates of chicken origin revealed that two of the *vat*(E) sequences were identical to that of *vat*(E-1); however, the sequence of strain CVM 3478 showed a single base change from that of the *vat*(E-1) allele originally described. In contrast, much more variation was observed between the *vat*(E) sequences of the two turkey sample *E. faecalis* isolates, which exhibited 3% sequence divergence, resulting in 93% amino acid identity. However, in one chicken sample isolate (CVM 3478) and one turkey sample isolate (CVM 3476), *vat*(E) showed the same base change, resulting in the same amino acid substitution (Ile15→Val). The MICs for these two isolates were almost identical (only differences in the MICs of erythromycin were seen), and the plasmid profiles of the two isolates were identical. We could therefore predict either that these two isolates are clones or that they share common plasmids. However, these two isolates were distinguishable by PFGE, indicating that perhaps they may have only acquired *vat*(E) from a common reservoir. The DNA sequence data obtained from this study suggest that the *vat*(E) genes in the chicken and turkey sample isolates may not, in all cases, have originated from the same source. There appears to be greater homology among the chicken *E. faecalis* *vat*(E) sequences, whereas heterogeneity may exist between the turkey *E. faecalis* *vat*(E) sequences. To confirm this hypothesis, a larger number of isolates would need to be studied.

Eight allelic variations of *vat*(E) from *E. faecium*, designated

![Vat(E) Variations](image.png)
vat(E-1) through vat(E-8), have been deposited in GenBank (17, 19). However, the vat(E) allelic variations observed from *E. faecalis* isolates in this study have not previously been observed in *E. faecium*. It is of interest that the substitutions found in the vat(E) alleles, including those described in the present study, are all focused around a very small region of the vat(E) allele, namely, between base positions 34 and 57 (amino acids 11 to 19). How these amino acid substitutions affect the activity of the Vat(E) protein is not known at present, as the three-dimensional crystal structure of the Vat(E) protein has not been determined. However, we suspect that this small region (amino acids 11 to 19) does not play an important role in streptogramin binding either directly or indirectly, as amino acid variations in this region do not seem to have any effect on the level of resistance to quinupristin-dalfopristin.

PFGE analysis showed that the two vat(E)-positive turkey sample isolates were indistinguishable from each other. Similarly, two vat(E)-positive chicken sample *E. faecalis* isolates were assigned to PFGE group J and one vat(E)-positive chicken sample isolate was assigned to PFGE group I. These results suggest that the vat(E) genes are not confined to a single clone in *E. faecalis*, as determined by PFGE.

It was interesting that the two vat(E)-positive turkey sample *E. faecalis* isolates belonged to the same PFGE group; however, there was 3% DNA sequence divergence in the vat(E)-coding region, suggesting that a clone of *E. faecalis* may have acquired vat(E) from different sources.

Conjugation studies demonstrated that the vat(E) gene could be transferred not only from an *E. faecalis* donor to an *E. faecalis* recipient but also from an *E. faecalis* donor to a streptogramin-susceptible *E. faecium* recipient. This implicates *E. faecalis* as a reservoir for vat(E) genes that can be transferred to *E. faecalis* or *E. faecium*. This is not surprising, as previous studies have shown that plasmids and transposons readily move between enterococcal species (15, 16). Susceptibility to quinupristin-dalfopristin, erythromycin, tetracycline, and streptomycin was detected following the transfer of the 70-kb plasmid. This is based on the observation that not all transconjugants received the full complement of plasmids that were contained within the donor strain; however, each transconjugant received the 70-kb plasmid. Although the linkage of vat(E) and emrB as part of a possible transposon-like element has recently been reported (Jensen et al., Letter, Antimicrob. Agents Chemother. 44:2231-2232, 2000), we could not establish a vat(E)-emrB linkage. It is possible that vat(E) may have appeared in *E. faecalis* by virtue of gene linkage on the same plasmid as the resistance determinants for either erythromycin, tetracycline, or streptomycin resistance. Thus, the vat(E) gene may have been coselected by the use of either macrolides, tetracyclines, or the aminoglycosides in the poultry production environment. It is therefore feasible that an organism such as *E. faecalis* that is intrinsically resistant to a given antimicrobial may, under certain conditions, harbor resistance genes as a consequence of coselection. This notion is supported by reports from Denmark, which showed that in 1998, 55.6% of *E. faecium* isolates recovered from pigs were resistant to virginiamycin. Following the 1998 ban on the use of virginiamycin in feeds, this prevalence decreased to 8.0% in 1999 and then increased again to 22.5% in 2000. Although it was unclear why resistance to virginiamycin increased in 2000, the investigators suggested that the increase was due to the emergence of isolates that were simultaneously resistant to erythromycin, kanamycin, penicillin, streptomycin, and tetracycline (1). This may explain to some degree the appearance of vat(E) in our isolates of *E. faecalis*. All five *E. faecalis* isolates that were vat(E) positive by PCR showed reduced susceptibilities to erythromycin, streptomycin, and tetracycline. It is plausible that the use of these antibiotics, either singly or in combination, may have coselected for the appearance and maintenance of vat(E) in a population of *E. faecalis* isolates.

In conclusion, we have detected vat(E) in *E. faecalis*, a species intrinsically resistant to quinupristin-dalfopristin, probably by virtue of efflux mechanisms, and found that this gene could readily be transferred to other *E. faecalis* and *E. faecium* isolates. The recovery of vat(E)-containing *E. faecalis* isolates from retail chickens and turkey samples (42 and 22% of samples, respectively) indicates a reservoir of streptogramin resistance genes that can potentially be transferred to human gram-positive pathogens via consumption of contaminated food. Even though the study cohort comprised a small number of randomly selected isolates, the presence of vat(E) in approximately 30% of the *E. faecalis* isolates suggests that the prevalence of vat(E) in the larger environment is significant. Until we fully understand the mechanisms of streptogramin resistance in enterococci and quantify the risk of gene transfer from nonhuman isolates to human isolates, the occurrence of streptogramin resistance genes in enterococci of animal origin remains a potential public health hazard.

REFERENCES


