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Differences in Antibiotic Resistance Patterns of *Enterococcus faecalis* and *Enterococcus faecium* Strains Isolated from Farm and Pet Animals

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The prevalence of acquired resistance in 146 *Enterococcus faecium* and 166 *Enterococcus faecalis* strains from farm and pet animals, isolated in 1998 and 1999 in Belgium, against antibiotics used for growth promotion and for therapy was determined. Acquired resistance against flavomycin and monensin, two antibiotics used solely for growth promotion, was not detected. Avoparcin (glycopeptide) resistance was found sporadically in *E. faecium* only. Avilamycin resistance was almost exclusively seen in strains from farm animals. Resistance rates were higher in *E. faecium* strains from broiler chickens than in strains from other animal groups with tylosin and virginiamycin and in *E. faecalis* as well as in *E. faecium* strains with narasin and bacitracin. Resistance against ampicillin was mainly found among *E. faecium* strains from pets and was absent in *E. faecalis*. Tetracycline resistance occurred most often in strains from farm animals, while enrofloxacin resistance, only found in *E. faecalis*, occurred equally among strains from all origins. Resistance against gentamicin was very rare in broiler strains, whereas resistance rates were high in strains from other origins. It can be concluded that resistance against antibiotics used solely for growth promotion was more prevalent in *E. faecium* strains than in *E. faecalis* strains. With few exceptions, resistance against the different categories of antibiotics was more prevalent in strains from farm animals than in those from pets.

Following the emergence of vancomycin (glycopeptide) resistance in enterococci, a discussion on the use of growth-promoting antibacterials has arisen. The fear of possible transfer of resistance genes from animal bacteria to human bacteria led to the ban on the use in animal feeds of most growth-promoting antibiotics in the European Community (6).

Reports of resistance in enterococci against growth-promoting agents are rare (1, 5, 15). Most data on resistance against growth promoters concern the prevalence of resistance against glycopeptides in enterococci from farm animals. Only one report has dealt with resistance rates in enterococci from pet animals against therapeutic antibiotics not used in animal feeds (21). However, only a limited number of strains and origins were investigated.

Since the prevalence of resistance against antibiotics in enterococci isolated from pet animals is largely unknown, we compared the resistance situation of enterococci from farm animals to that from pets. Most growth-promoting antibacterials are not used in the latter category of animals. The data obtained are discussed in respect to the major trends in antibiotic usage in the different animal groups. Resistance prevalences of enterococci from animals are compared to recent data on resistance prevalences among human enterococci.

MATERIALS AND METHODS

Bacterial strains. A total of 146 strains of *Enterococcus faecium* and 166 strains of *Enterococcus faecalis* were isolated in Belgium from 1998 to 1999. They originated from the feces of different pet animals (divided into strains originating from mammalian pets and those from avian pets) and farm animals (broiler chickens, bovines, and fattening pigs), as listed in Table 1. Among mammalian pet animals were cats, dogs, hamsters, squirrels, monkeys, rabbits, rats, and horses. Among the pet birds were pigeons, geese, ducks, parrots, parakeets, swans, canaries, and some other passeriformes. There was no connection between the strains from pets and the strains from farm animals. Strains from pet animals were derived from pets attending the different clinics at the Faculty of Veterinary Medicine, University of Ghent, or were derived from animals brought in for necropsy at the Department of Pathology of the Faculty of Veterinary Medicine, University of Ghent. Each strain was representative of a single origin: a single farm or owner originating from the Flemish provinces of Belgium. No data on previous antibiotic usage (for therapy or for growth promotion) were collected. In general, Belgian farm animals are fed a diet containing a growth promoter. This study was performed after the banning of avoparcin (banned in 1997), and some strains were collected shortly after the banning of bacitracin, tylosin, spiramycin, and virginiamycin (banned in January 1999). Fecal samples were inoculated on Columbia CNA blood agar (Gibco, Paisley, United Kingdom), a selective medium containing colistin and nalidixic acid, and incubated overnight in air supplemented with 5% CO_2 or on Slanetz and Bartley agar (Oxoid, Basingstoke, United Kingdom). Enterococcus-like colonies were purified and identified as described earlier (12, 13, 14). Two control strains, *E. faecalis* ATCC 29212 and *Staphylococcus aureus* ATCC 29213, were included in the tests, as recommended in the National Committee for Clinical Laboratory Standards (NCCLS) standard procedures for testing therapeutic antibiotics (NCCLS M31-A) (18). The *E. faecium* type strain, LMG 11423^T, was included as an additional control.

Antibacterials. The growth-promoting antibacterials were obtained as laboratory standard powders. The following antibiotic preparations were tested: avoparcin (Roche, Basel, Switzerland), virginiamycin (Pfizer, Rixensart, Belgium), bacitracin (67,000 IU/g; Sigma, St. Louis, Mo.), tylosin (Sigma), avilamycin (Eli Lilly, Indianapolis, Ind.), and narasin (Eli Lilly). The antibiotics were dissolved in appropriate solvents to make stock solutions containing 1,000 μg/ml, or 1,000 IU (USP)/ml in the case of bacitracin, and then further diluted in sterile distilled water according to the methods recommended by the NCCLS (18). The antibiotics not listed in M31-A were avoparcin and bacitracin dissolved in distilled water, virginiamycin dissolved in ethanol, and avilamycin and narasin dissolved in methanol. Further dilutions were performed as prescribed by the NCCLS.

The therapeutic antibiotics enrofloxacin (Bayer AG, Leverkusen, Germany),

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TABLE 1. Number of enterococcal strains tested

Origin	No. tested	
	E. faecium	*E. faecalis*
Broiler	31	35
Swine	33	36
Ruminants	10	25
Pet, avian	42	37
Pet, mammalian	30	33

oxytetracycline (Sigma), ampicillin (Sigma), gentamicin (Schering-Plough, Kenilworth, N.J.), and streptomycin (Sigma) were prepared as described above.

Susceptibility tests. MIC tests were carried out on Mueller-Hinton II agar (Becton Dickinson, Cockeysville, Md.) containing doubling dilutions of the antibiotics (7). Concentrations from 0.06 to 256 μg/ml were tested for all antibiotics except streptomycin and gentamicin, for which only the concentrations 500, 1,000, and 2,000 μg/ml were tested. Antibiotic-free agar plates were included as controls. Inocula were prepared by diluting overnight BHI (Oxoid) cultures in buffered saline to a density of 0.5 on the McFarland turbidity scale and were diluted 40-fold before inoculation. Plates were seeded with approximately 10^5 CFU. The plates were incubated in air at 37°C for 24 h. The strains showing resistance to avoparcin were tested for the presence of *van* genes by using multiplex PCR for the detection of the *vanA, vanB, vanC*$_1$, and *vanC*$_2$ genes as described before (14). Strains for which the ampicillin MICs were ≥16 μg/ml were tested for the production of β-lactamase by the nitrocefin test (Oxoid) (19). High-level resistance against streptomycin and gentamicin was defined as a MIC of >2,000 and ≥500 μg/ml, respectively, as indicated by the NCCLS guidelines (M31-A) (18). Breakpoints of other therapeutically used substances were derived from the same source.

Statistical analysis. Differences in prevalence of resistances among *E. faecium* and *E. faecalis* from different origins were analyzed by the Fischer exact test. A significance level of α 0.05 was used.

RESULTS

***E. faecium* and growth-promoting antibiotics.** All strains were naturally resistant to flavomycin (MIC at which 90% of isolates are inhibited [MIC_{90}], >256 μg/ml), although strains from broiler chickens and swine had a wider MIC distribution range (Table 2) than strains from other origins. No resistance was found against monensin (MIC range, between 0.12 and 4 μg/ml). Three strains were resistant to the glycopeptide antibiotic avoparcin. One strain originated from a broiler chicken, one from a pet rabbit, and one from a bovine. Their resistance was mediated by the *vanA* gene, as demonstrated by PCR. Lowered susceptibility to narasin was mainly evident in strains from broiler chickens and swine. High avilamycin MICs were only found among the farm animal strains.

***E. faecium* and antibiotics used for both growth promotion and therapy.** Tylosin-resistant strains were isolated from all origins. Significantly more resistant strains were found in farm animals than in pet animals ($P < 0.001$). Virginiamycin-resistant strains (MIC, ≥16 μg/ml) were isolated from broilers, swine, and a pet. Strains for which the bacitracin MICs were 16 IU/ml or higher were found mainly in the collections from broilers and swine.

***E. faecium* and therapeutic antibiotics.** Ampicillin resistance was seen in strains from all origins except from pet birds. No β-lactamase production was detected in these strains. Resistant strains were significantly more frequent in the collection from mammalian pets than in those from farm animals and from avian pets ($P < 0.001$). Resistance against streptomycin (MIC, >2,000 μg/ml) was not detected in strains isolated from ruminants. No statistically significant differences were seen among the different origins for this type of resistance. Gentamicin-resistant *E. faecium* strains (MIC, ≥500 μg/ml) were isolated from all origins but mainly from swine. Almost all strains from broilers, pigs, and ruminants were resistant to oxytetracycline, while strains from pet animals were significantly less often resistant to this antibiotic ($P < 0.001$). A monomodal distribution of the enrofloxacin MICs was obtained, ranging from 0.5 to 8 μg/ml. The enrofloxacin MIC for the *E. faecium* type strain (LMG11423^T [equivalent to ATCC 19434^T]) was consistently in the higher range (8 μg/ml).

***E. faecalis* and growth-promoting antibiotics.** No resistance was found against the antibiotics flavomycin, avoparcin, and monensin. Resistance against narasin occurred in strains from all origins, but mainly in poultry strains. Avilamycin resistance was found in one strain each from a broiler chicken, a pig, and a duck.

***E. faecalis* and antibiotics used for both growth promotion and therapy.** Resistance against tylosin in *E. faecalis* strains from all origins was high. No significant differences were noted among the different animal origins. High virginiamycin MICs were seen for strains from swine and broilers only. High bacitracin MICs occurred for strains from all origins, but strains from broilers were more often affected than strains from other origins.

***E. faecalis* and therapeutic antibiotics.** No resistance was found against ampicillin. High-level streptomycin resistance (MIC, >2,000 μg/ml) occurred significantly more often among strains from swine than in strains from pets and ruminants ($P < 0.01$), and significantly more streptomycin-resistant strains were isolated from broiler chickens than from pet birds ($P < 0.01$). In contrast, high-level gentamicin resistance (MIC, ≥500 μg/ml) was not found among broiler strains. There were no significant differences in gentamicin resistance prevalence among strains from other origins. Tetracycline resistance was extremely frequent in strains from all origins. Not a single tetracycline-susceptible strain was present in the porcine collection. The enrofloxacin MIC_{90} was 2 μg/ml, while a few strains were inhibited only by 8 to 64 μg/ml (Table 2), indicating the presence of resistance.

The MICs of the growth promoters for quality control strains were within the ranges published earlier (4, 5, 7), and those of other antibiotics as published in the NCCLS standards (M31-A) for the *S. aureus* and *E. faecalis* strains (18).

DISCUSSION

Although antibiotics used solely for growth promotion are not included in national and international standards for susceptibility testing, and universally recognized breakpoints are not available, certain indications are useful. This is notably the case with virginiamycin, bacitracin, and avilamycin. For virginiamycin, MICs of ≥16 μg/ml can be considered as indicating resistance, because resistance genes have been demonstrated only in *E. faecium* strains not inhibited by concentrations of 16 μg/ml and higher (16). The bacitracin MICs for *E. faecium* strains show a rather extended range. This indicates that different resistance mechanisms might be present. However, nothing is known about the mechanisms or about the genes encoding this type of resistance in enterococci. Avilamycin-

TABLE 2. Susceptibility of *E. faecium* and *E. faecalis* strains from farm animals and pets

Antibiotic and host origin	*E. faecium*				*E. faecalis*			
	MIC_{50}[a]	MIC_{90}[a]	Range[a]	% Susceptible	MIC_{50}[a]	MIC_{90}[a]	Range[a]	% Susceptible
Flavomycin								
Broilers	128	>256	8–>256	NA[b]	0.25	0.5	0.12–2	NA
Swine	>256	>256	32–>256	NA	0.25	0.5	0.12–1	NA
Ruminants	>256	>256	128–>256	NA	0.12	0.25	0.12–0.25	NA
Pet, avian	>256	>256	128–>256	NA	0.25	0.5	0.25–0.5	NA
Pet, mammalian	>256	>256	256–>256	NA	0.25	0.5	0.12–1	NA
Avoparcin								
Broilers	1	4	0.5–64	NA	1	2	0.5–2	NA
Swine	1	2	0.5–2	NA	1	2	0.5–2	NA
Ruminants	1	1	0.5–64	NA	2	4	0.5–4	NA
Pet, avian	1	2	0.5–2	NA	1	4	0.5–4	NA
Pet, mammalian	1	4	1–64	NA	1	2	0.5–4	NA
Monensin								
Broilers	4	8	0.12–4	NA	4	8	1–8	NA
Swine	4	8	0.12–4	NA	4	8	2–8	NA
Ruminants	2	8	0.12–4	NA	8	8	2–8	NA
Pet, avian	2	8	0.12–4	NA	4	8	2–8	NA
Pet, mammalian	8	8	0.12–4	NA	8	8	1–8	NA
Narasin								
Broilers	2	4	0.12–4	NA	0.25	2	0.06–4	NA
Swine	0.25	2	0.06–8	NA	0.25	0.25	0.06–4	NA
Ruminants	0.25	0.25	0.12–0.25	NA	0.25	0.25	0.06–2	NA
Pet, avian	0.25	0.25	0.06–0.5	NA	0.25	0.25	0.06–2	NA
Pet, mammalian	0.12	0.25	0.06–1	NA	0.25	0.25	0.06–2	NA
Avilamycin								
Broilers	0.5	32	0.12–>256	NA	0.5	1	0.12–32	NA
Swine	0.5	16	0.25–>256	NA	0.5	1	0.25–32	NA
Ruminants	0.25	0.25	0.25–1	NA	1	1	0.25–1	NA
Pet, avian	0.5	1	0.25–1	NA	0.5	1	0.25–>256	NA
Pet, mammalian	0.25	0.5	0.25–1	NA	1	1	0.25–1	NA
Virginiamycin								
Broilers	32	64	0.5–64	NA	4	16	1–32	NA
Swine	2	8	0.5–32	NA	4	16	2–32	NA
Ruminants	1	1	0.25–8	NA	2	4	0.5–4	NA
Pet, avian	0.5	1	0.25–4	NA	4	4	2–8	NA
Pet, mammalian	0.5	2	0.25–16	NA	4	4	1–8	NA
Bacitracin[a]								
Broilers	32	256	2–256	NA	64	>256	0.5–>256	NA
Swine	4	128	0.12–>256	NA	4	8	2–>256	NA
Ruminants	2	16	2–16	NA	4	256	1–>256	NA
Pet, avian	4	8	0.5–128	NA	4	256	1–>256	NA
Pet, mammalian	8	64	0.5–128	NA	4	8	1–256	NA
Tylosin								
Broilers	32	>256	1–>256	18	32	>256	0.5–>256	46
Swine	256	>256	0.5–>256	18	256	>256	2–>256	14
Ruminants	>256	>256	1–>256	20	>256	>256	1–>256	48
Pet, avian	2	128	0.5–>256	86	2	>256	1–>256	59
Pet, mammalian	2	>256	1–>256	57	5	>256	1–>256	58
Ampicillin								
Broilers	1	4	0.06–32	94	1	2	0.25–2	100
Swine	1	8	0.12–128	94	1	4	0.12–8	100
Ruminants	4	4	1–32	80	2	2	1–2	100
Pet, avian	1	4	0.12–4	100	2	4	1–8	100
Pet, mammalian	2	128	0.25–128	74	2	2	0.06–4	100
Oxytetracycline								
Broilers	64	>256	0.12–>256	8		>256	0.25–>256	21
Swine	128	>256	0.25–>256	3		>256	8–>256	0
Ruminants	>256	>256	0.5–>256	20		256	0.5–>256	29
Pet, avian	0.5	128	0.25–>128	69		256	0.5–>256	29
Pet, mammalian	16	128	0.25–>256	47		256	0.5–256	30

Continued on following page

TABLE 2—*Continued*

Antibiotic and host origin	*E. faecium*				*E. faecalis*			
	MIC_{50}[a]	MIC_{90}[a]	Range[a]	% Susceptible	MIC_{50}[a]	MIC_{90}[a]	Range[a]	% Susceptible
Enrofloxacin								
Broilers	4	8	0.5–8	NA	1	2	0.25–8	NA
Swine	2	8	0.5–16	NA	0.5	1	0.25–2	NA
Ruminants	4	8	0.5–8	NA	0.5	16	0.25–64	NA
Pet, avian	1	8	0.5–16	NA	2	4	0.5–8	NA
Pet, mammalian	2	8	1–8	NA	1	1	0.12–32	NA
Streptomycin								
Broilers	<500	>2,000	<500–>2,000	88	<500	>2,000	<500–>2,000	74
Swine	1,000	>2,000	<500–>2,000	79	2,000	>2,000	<500–>2,000	53
Ruminants	<500	1,000	<500	100	1,000	>2,000	<500–>2,000	88
Pet, avian	<500	1,000	<500–>2,000	95	<500	1,000	<500–>2,000	97
Pet, mammalian	<500	>2,000	<500–>2,000	80	>500	>2,000	<500–>2,000	82
Gentamicin								
Broilers	<500	<500	<500–2,000	97	<500	<500	<500	100
Swine	<500	2,000	<500–2,000	79	<500	2,000	<500–>2,000	75
Ruminants	<500	<500	<500–2,000	90	<500	2,000	<500–>2,000	72
Pet, avian	<500	<500	<500–>2,000	93	<500	2,000	<500–2,000	70
Pet, mammalian	<500	<500	<500–>2,000	93	<500	2,000	<500–>2,000	88

[a] Micrograms per milliliter. Bacitracin MICs are in international units per milliliter.
[b] NA, not applicable (no NCCLS breakpoint available).

resistant strains seemingly have two types of resistance: a low-level type and a high-level type. This was also evident in a Danish study (1). Resistance against everninomycin, a related antibiotic, has been shown to be mediated by a mutation in the ribosomal L16 protein in *Streptococcus pneumoniae* (2). If resistance in enterococci is also mediated by point mutations, a double mutation or a mutation combined with a specific resistance gene might explain the two levels of resistance observed in the present study.

No acquired resistance was found against flavomycin and monensin, two antibiotics used solely for growth promotion. Monensin results obtained in this study were similar to the results obtained in a Danish investigation (1). In the latter study, a few *E. faecium* strains for which the flavomycin MICs were low were reported. However, it must be noted that identification errors may occur when phenotypic identification is used (3), and it has been shown that several enterococcal species can be classified as naturally flavomycin resistant while others are naturally susceptible (4). The flavomycin MIC for the *E. faecium* type strain used as an additional control in the present investigation was consistently ≥256 μg/ml.

Only three *E. faecium* strains were resistant to avoparcin, a glycopeptide antibiotic with full cross-resistance with vancomycin (6). This relatively low resistance rate is due to the fact that no selective enrichment and isolation in media containing vancomycin was applied. This factor strongly influences the isolation frequencies of resistant strains (9).

Ionophores are included routinely in broiler feed for the prevention of coccidiosis. In swine, salinomycin is allowed for growth promotion, and in ruminants, monensin is used for the same purpose. These products are not used for pet animals. This might explain the differences seen between the enterococci from different animal origins. Narasin resistance, fully cross-resistant with salinomycin (8), was found only in *E. faecium* from farm animals, mainly broilers. Since the resistance mechanism and resistance genes are unknown, no specific additional test is available for confirming resistance. In contrast, narasin resistance in *E. faecalis* was present in strains from all origins, but resistant strains were isolated mainly from broilers.

The prevalences of resistances to antibiotics used both therapeutically and for growth promotion were generally much higher than those for resistances to the antibiotics used solely for growth enhancement. However, it should be noted that for the antibiotics virginiamycin and bacitracin, only topical preparations are available. These are only sporadically used in mammalian pet animals and not at all in pet birds. Bacitracin-resistant enterococci were found to occur in several animal species, including pet birds. The reason for this remains unclear. Tylosin and related macrolides are the only antibiotics in this group that are commonly applied in the therapy of pet and farm animals. This is reflected by the high resistance prevalence in pet animals as well as in farm animals. Nevertheless, significantly more tylosin-resistant *E. faecium* strains were isolated from farm animals than from pets, possibly reflecting the more extensive use of this antibiotic in farm animals.

For the therapeutically most important antibiotics in human enterococcal infections, certain differences were noted between the resistance situation in animal and in human clinical strains. The resistance rates among animal strains were lower than those found among human *E. faecium* strains isolated from hospital patients in a recent European study (20), while the rates among ruminant and mammalian pet strains were similar to those found in human strains in 1993 in Belgium (22). High-level streptomycin resistance, defined as a MIC of >2,000 μg/ml, has been reported to be generally more frequent than high-level gentamicin resistance, defined as a MIC of ≥500 μg/ml (17, 20). This was not evident in our study except for the poultry strains, in which gentamicin resistance was rare. The prevalence of gentamicin resistance in *E. faecium* strains from swine, ruminants, and avian pets was higher than that recently reported in human strains (20), while the opposite was true for *E. faecalis*.

Enterococcal infections are rare in animals and are not treated as such (10, 11). However, as intestinal inhabitants, enterocci are under selective pressure from every antibiotic administered that is active against them. Tetracyclines are still used frequently in animals, and resistance rates were very high in both the *E. faecium* and *E. faecalis* collections, especially in strains originating from swine. Only one porcine *E. faecium* strain was found to be susceptible, and all porcine *E. faecalis* strains were resistant. Enrofloxacin is only marginally active against *E. faecium*. Although all strains were in the intermediate or resistant category of the NCCLS susceptibility criteria, they cannot be considered as having acquired resistance, since not only the enrofloxacin MICs for the field strains were monomodally distributed. The breakpoint of this antibiotic is the median of the monomodal distribution of the MICs for the *E. faecium* strains tested here. Moreover, the MIC for the *E. faecium* type strain used was 8 μg/ml, which is above the breakpoint.

In conclusion, resistance against growth-promoting antibiotics and antibiotics used for both growth promotion and therapy, as well as resistance against therapeutic antibiotics, was present mainly in strains from food animals. β-Lactam resistance, more frequently found in strains from mammalian pets, was the only exception in the last category of agents.

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REFERENCES

1. **Aarestrup, F. M., F. Bager, N. E. Jensen, M. Madsen, A. Meyling, and H. C. Wegener.** 1998. Surveillance of antimicrobial resistance in bacteria isolated from food animals to antimicrobial growth promoters and related therapeutic agents in Denmark. APMIS **106:**606–622.
2. **Adrian, P. V., W. Zhao, T. A. Black, K. J. Shaw, A. S. Hare, and K. Klugman.** 2000. Mutations in ribosomal protein L16 conferring reduced susceptibility to everninomycin (SCH27899): implications for mechanism of action. Antimicrob. Agents Chemother. **44:**732–738.
3. **Bascomb, S., and M. Manfini.** 1998. Use of enzyme tests in characterization and identification of aerobic and facultatively aerobic Gram-positive cocci. Clin. Microbiol. Rev. **11:**318–340.
4. **Butaye, P., L. A. Devriese, and F. Haesebrouck.** 2000. Influence of different medium components on the in vitro activity of the growth-promoting antibiotic flavomycin against enterococci. J. Antimicrob. Chemother. **46:**713–716.
5. **Butaye, P., L. A. Devriese, and F. Haesebrouck.** 1999. Phenotypic distinction in *Enterococcus faecium* and *Enterococcus faecalis* strains between susceptibility and resistance to growth-promoting antibiotics. Antimicrob. Agents Chemother. **43:**2569–2570.
6. **Butaye, P., L. A. Devriese, and F. Haesebrouck.** 1999. Glycopeptide resistance in *Enterococcus faecium* strains from animals and humans. Rev. Med. Microbiol. **10:**235–243.
7. **Butaye, P., L. A. Devriese, and F. Haesebrouck.** 1998. Effects of different test conditions on MICs of food animal growth-promoting antibacterial agents for enterococci. J. Clin. Microbiol. **36:**1907–1911.
8. **Butaye, P., L. A. Devriese, and F. Haesebrouck.** 2000. Incomplete cross resistance against ionophores in *Enterococcus faecium* and *Enterococcus faecalis* strains from pigs and poultry. Microb. Drug Resist. **6:**59–61.
9. **Butaye, P., L. A. Devriese, and F. Haesebrouck.** 1999. Comparison of direct and enrichment methods for the selective isolation of vancomycin-resistant enterococci from feces of pigs and poultry. Microb. Drug Resist. **5:**131–134.
10. **Butaye, P., P. Verleyen, L. A. Devriese, H. Van Bree, and F. Haesebrouck.** 2000. *Enterococcus faecalis* infection after orthopedic surgery in a dog. Vlaams Diergen. Tijdschr. **69:**42–43.
11. **Devriese, L. A., P. De Herdt, E. Uyttebroek, C. Lepoudre, R. Ducatelle, P. Dom, and F. Haesebrouck.** 1994. Streptococcen-en enterococcen-infecties bij vogels. Vlaams Diergen. Tijdschr. **63:**109–111.
12. **Devriese, L. A., B. Pot, L. Van Damme, K. Kersters, and F. Haesebrouck.** 1995. Identification of *Enterococcus* species isolated from foods of animal origin. Int. J. Food Microbiol. **26:**187–197.
13. **Devriese, L. A., B. Pot, K. Kersters, S. Lauwers, and F. Haesebrouck.** 1996. Acidification of methyl-α-D-glucopyranoside: a useful test to differentiate *Enterococcus casseliflavus* and *Enterococcus gallinarum* from *Enterococcus faecium* species group and from *Enterococcus faecalis*. J. Clin. Microbiol. **34:**2607–2608.
14. **Dutka-Malen, S., S. Evers, and P. Courvalin.** 1995. Detection of glycopeptide resistance genotypes and identification to the species level of clinically relevant enterococci by PCR. J. Clin. Microbiol. **33:**24–27.
15. **Dutta, G. N., and L. A. Devriese.** 1982. Susceptibility of fecal streptococci of poultry origin to nine growth-promoting agents. Appl. Environ. Microbiol. **44:**832–837.
16. **Jensen, L. B., A. M. Hammerum, F. M. Aarestrup, A. E. van den Boogaard, and E. E. Stobberingh.** 1998. Occurrence of *satA* and *vgb* genes in streptogramin-resistant *Enterococcus faecium* isolates of animal and human origin. Antimicrob. Agents Chemother. **42:**3330–3331.
17. **Murray, B. E.** 1990. The life and times of the enterococcus. Clin. Microbiol. Rev. **3:**46–65.
18. **National Committee for Clinical Laboratory Standards.** 1999. Approved standard M31-A. Performance standards for antimicrobial disk and dilution susceptibility tests for bacteria isolated from animals. National Committee for Clinical Laboratory Standards, Wayne, Pa.
19. **O'Callaghan, C. H., A. Morris, S. M. Kirby, and A. H. Shingler.** 1972. Novel method for detection of β-lactamase by using a chromogenic cephalosporin substrate. Antimicrob. Agents Chemother. **1:**283–288.
20. **Schouten, M. A., A. Voss, J. A. A. Hoogkamp-Korstanje, and The European VRE Study Group.** 1999. Antimicrobial susceptibility patterns of enterococci causing infections in Europe. Antimicrob. Agents Chemother. **43:**2542–2546.
21. **Thal, L. A., J. W. Chow, R. Mahayni, H. Bonilla, M. B. Perri, S. A. Donabedian, J. Silverman, S. Taber, and M. J. Zervos.** 1995. Characterization of antimicrobial resistance in enterococci of animal origin. Antimicrob. Agents Chemother. **39:**2112–2118.
22. **Vandamme, P., E. Vercauteren, C. Lammens, N. Pensart, M. Ieven, B. Pot, R. Leclercq, and H. Goossens.** 1996. Survey of enterococcal susceptibility patterns in Belgium. J. Clin. Microbiol. **34:**2572–2576.