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Plasmid-Determined Resistance to Tellurium Compounds

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Transferable plasmids in gram-negative bacteria that confer resistance to potassium tellurite or tellurate were found. This resistance was distinct from resistance to mercury, silver, or arsenic compounds and was unrelated to antibiotic resistance. In *Escherichia coli*, plasmids determine a 100-fold increase in the minimal inhibitory concentration for tellurite and a 10-fold increase in tellurate resistance. Many, but not all, of the plasmids belong to incompatibility group S. In *Pseudomonas aeruginosa*, tellurium resistance is specifically associated with incompatibility group P-2 and involves a 5- to 10-fold increase in tellurite or tellurate resistance.

The oxyanions of tellurium, tellurite and tellurate, are toxic for most microorganisms (15). A notable exception is *Corynebacterium diphtheriae*, which is naturally resistant; therefore high concentrations of potassium tellurite have been used in selective media since 1912 to isolate this organism and distinguish its subtypes (8). Tellurite resistance is also characteristic of *Streptococcus faecalis* (47) and most *Staphylococcus aureus* (30).

The toxicity of tellurite may be related to its ability to act as a strong oxidant. Since tellurite is easily reduced by oxidoreductases under physiological conditions, it has been used in a manner similar to the tetrazolium dyes as an indicator stain for intracellular sites containing these enzymes (32, 33). Even susceptible bacteria growing on subinhibitory concentrations of tellurite produce jet-black colonies as a result of the reduction of tellurite and the formation of intracellular granules of metallic tellurium (52).

The biochemical basis for tellurite resistance has been studied by several groups. Terai et al. (51) showed that a cell-free extract of *Mycobacterium avium* was capable of reducing tellurite in the presence of reduced nicotinamide adenine dinucleotide or malate and malic dehydrogenase, and Thomas et al. (Bacteriol. Proc., P119, p. 124, 1963) described tellurite-reducing capacity in cell-free extracts of *S. faecalis*. However, the specific role of "tellurite reductase" in resistance was not established. As noted above, many bacteria contain oxidoreductases that would be capable of reducing tellurite given the appropriate electron donor in a cell-free system. One specific pathway that has been implicated in tellurite resistance is that involved in the reduction of sulfate. Scala and Williams (44) found that tellurite susceptibility was enhanced by growing *Escherichia coli* in the presence of L-methionine and that selenite toxicity was affected similarly. They proposed that tellurite and selenite, which are chemically similar to sulfate, could be reduced by the sulfate pathway providing that this pathway was not repressed by an exogenous reduced sulfur source.

The genetic basis for resistance to tellurite is not known. Although gram-negative bacteria are generally more susceptible to tellurite than gram-positive organisms, Alexander Fleming reported in 1932 and 1940 (16, 17) that some *E. coli* and *Proteus* isolates were tellurite resistant. Plasmids in gram-negative bacteria are known to mediate resistance to other metal ions, e.g., compounds of arsenic (22), mercury (37, 48, 50), and silver (40). We found that resistance to tellurite and tellurate is another plasmid-determined characteristic, and one that in *Pseudomonas aeruginosa* is specifically associated with plasmids belonging to the P-2 incompatibility (Inc) group.

MATERIALS AND METHODS

Bacterial strains and plasmids. Plasmids from Y. Chabbert, N. Datta, R. Hedges, S. Levy, E. Meynell, R. Olsen, and D. Smith were provided in or transferred to *E. coli* J53 (F^- *pro-22 metF63*) (7). *P. aeruginosa* PU21 (*ilvB112 leu-1 str^r-1 rif^r*) (35) and its R^+ derivatives were from our collection (G. A. Jacoby, *in* D. Schlessinger, ed., *Microbiology—1977*, in press).

Chemicals. Potassium tellurate, sodium selenite, and sodium selenate were obtained from Alfa Products, potassium tellurite was obtained from Fisher Scientific Co., and sodium arsenate was obtained from Merck and Co., Inc.

Culture media. *E. coli* strains were grown in

either L broth (38) or tryptone broth (16 g of tryptone [Difco Laboratories] and 5 g of NaCl per liter). *P. aeruginosa* strains were grown in nutrient broth (Difco) containing 4 mg of potassium nitrate per ml (35).

Determination of metal ion resistance. (i) Rapid screening method. Resistance was determined qualitatively on a gradient plate by a modification of the method described by Fleming (16). A ditch (5 by 80 mm) was cut in the center of a square plate containing 50 ml of dilute (27 g/liter) heart infusion agar (Difco). Overnight cultures of the strains to be tested were streaked from the edge of the ditch to the edge of the plate. When these streaks were dry, 1 ml of the inhibitor solution (0.5% potassium tellurite or a saturated solution at 24°C of sodium arsenate, sodium selenate, sodium selenite, or potassium tellurate) was added to the ditch and the plates were incubated overnight at 37°C. After incubation, relative susceptibility was determined by measuring the zones of inhibition.

(ii) Minimal inhibitory concentrations (MICs). Quantitative susceptibility tests were performed by agar dilution on plates containing brain heart infusion agar (Difco) and graded concentrations of inhibitor that were spotted with 10^4 to 10^5 organisms from an overnight culture. The MIC was determined as the lowest concentration of inhibitor preventing growth after overnight incubation at 37°C.

Resistance to mercuric chloride, phenylmercuric acetate, and silver nitrate was determined as described previously (35, 40).

Determination of antibiotic resistance. Resistance to various antibiotics was determined either by the Kirby-Bauer technique (2) on Mueller-Hinton medium (Baltimore Biological Laboratory) or by spotting overnight cultures on agar plates containing suitable concentrations of antibiotic.

RESULTS

Resistance to tellurite. Figure 1 illustrates the resistance to tellurite conferred by certain plasmids in *E. coli* or *P. aeruginosa*. On the left side of the plate in Fig. 1 is a series of *E. coli* strains, and on the right side is a series of *P. aeruginosa* strains. Tellurite diffusing from the central ditch inhibited the growth of all but the resistant bacteria in the vicinity of the ditch. All the strains produced a black deposit that did not diffuse into the medium (Fig. 1). This black precipitate is known to be metallic tellurium (52). Both susceptible cells growing in low tellurite concentrations at the edge of the plate as well as resistant cells growing in the region of higher tellurite concentrations appeared black. Hence, both susceptible and resistant cells were capable of reducing tellurite to metallic tellurium, but they differed markedly in the concentration of tellurite allowing growth. Note also that R^- *E. coli* was more susceptible to tellurite than R^- *P. aeruginosa* by this test.

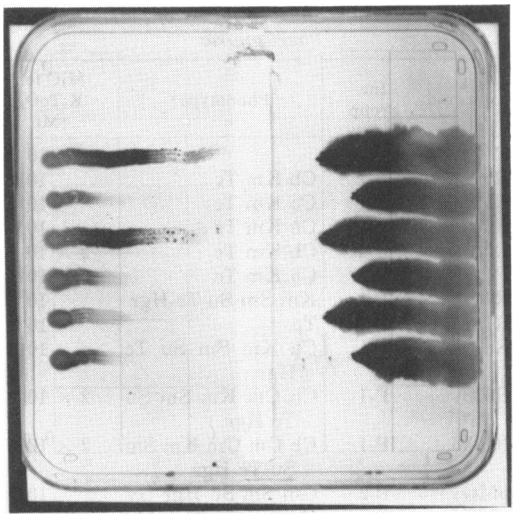

FIG. 1. *Qualitative demonstration of tellurite resistance. A gradient plate was prepared as described in the text. The strains on the left side of the plate are (from top to bottom): E. coli J53(R826), J53(R773), J53(pMG101), J53(JJ1), J53(R57b), and J53 R^-; and on the right side (from top to bottom): P. aeruginosa PU21(R3108), PU21(Sa), PU21(CAM), PU21(R46), PU21(pMG2), and PU21 R^-. One milliliter of a solution of 0.5% potassium tellurite was placed in the well and the plate was incubated at 37°C for 18 h.*

Quantitative aspects of tellurite resistance. These qualitative observations were confirmed by agar dilution susceptibility testing using graded concentrations of potassium tellurite. Table 1 lists the MICs for a variety of plasmid-containing strains of *P. aeruginosa*, and Table 2 provides similar data for a selected group of *E. coli* strains. Plasmids conferred a comparable level of tellurite resistance (MIC, 10^{-3} M) in both genera, but since R^- *P. aeruginosa* is intrinsically 10-fold more resistant than R^- *E. coli*, plasmids provided a 10-fold increase in tellurite resistance in *P. aeruginosa* and a 100-fold increase in *E. coli*.

Relationship of tellurite resistance to resistance to other metal ions and to antibiotics. The plasmids chosen for testing included many that conferred resistance to mercury ions and some that provided resistance to arsenate, silver, and phenylmercuric acetate. Of the plasmids that conferred resistance to tellurite in *E. coli*, R477 and R478 conferred resistance to arsenate and mercuric salts, R826 and R828 provided resistance to arsenate, mercuric ions, and phenylmercuric acetate, and pMG101 was unusual in providing resistance to silver nitrate as well as to mercuric salts. All the tellurite-re-

TABLE 1. *Tellurite susceptibility of plasmids in P. aeruginosa*

Plasmid	Inc group	Phenotype[a]	MIC for K_2TeO_3 (M)
R⁻			10^{-4}
RP1	P-1	Cb Km Tc	10^{-4}
RP4	P-1	Cb Km Tc	10^{-4}
R30	P-1	Cb Km Tc	10^{-4}
R68	P-1	Cb Km Tc	2×10^{-4}
RP-638	P-1	Cb Km Tc	2×10^{-4}
R702	P-1	Km Sm Su Tc Hgr	10^{-4}
R751	P-1	Tp	10^{-4}
R839	P-1	Cb Km Sm Su Tc Hgr	10^{-4}
R938	P-1	Cb Cm Km Sm Su Tc Hgr	2×10^{-4}
R1033	P-1	Cb Cm Gm Km Sm Su Tc Hgr	2×10^{-4}
pMG1	P-2	Gm Sm Su Hgr Uv	10^{-3}
pMG2	P-2	Gm Sm Su Hgr Uv	10^{-3}
pMG5	P-2	Ak Bt Km Su Tm Hgr Pmr	10^{-3}
RPL11	P-2	Cb Cm Gm Sm Su Tc Hgr Pmr	10^{-3}
R38	P-2	Sm Su Tc Hgr Pmr	10^{-3}
R39	P-2	Sm Su Tc Hgr Pmr	10^{-3}
Rms159	P-2	Cm Sm Tc Hgr Pmr	10^{-3}
R931	P-2	Sm Tc Hgr Uv	10^{-3}
R3108	P-2	Sm Su Tc Hgr Pmr	10^{-3}
CAM	P-2	Cam Uv	10^{-3}
CAM-OCT	P-2	Cam Oct Uv	10^{-3}
Rip55	P-3	Cb Cm Km Gm Su Tm Hgr	5×10^{-5}
Rip64	P-3	Cb Cm Gm Su Tm Hgr	5×10^{-5}
R679	P-4	Sm Su	10^{-4}
R1162	P-4	Sm Su	10^{-4}
R5265	P-4	Sm Su	10^{-4}
Rms163	P-5	Cm Su Tc	10^{-4}
Rms149	P-6	Cb Gm Sm Su	10^{-4}
Rms148	P-7	Sm	10^{-4}
FP2	P-8	Hgr Pmr	10^{-4}
R46	N	Cb Sm Su Tc Asr	10^{-4}
R7K	W	Cb Sm	10^{-4}
R388	W	Su Tp	5×10^{-5}
Sa	W	Cm Gm Km Sm Su Tm	5×10^{-5}
RPl-1	?	Cb	5×10^{-5}
R2	?	Cb Sm Su Uv	10^{-4}
RP8	?	Cb	5×10^{-5}
R91	?	Cb	10^{-4}
FP5	?	Hg	2×10^{-4}

[a] Phenotypic abbreviations (resistance): Ak, amikacin; Asr, sodium arsenate; Bt, butirosin; Cb, carbenicillin; Cm, chloramphenicol; Gm, gentamicin; Hgr, mercuric chloride; Km, kanamycin; Pmr, phenylmercuric acetate; Sm, streptomycin; Su, sulfonamide; Tc, tetracycline; Tm, tobramycin; Tp, trimethoprim; Uv, ultraviolet irradation. Phenotypic abbreviations (biodegradation): Cam, camphor; Oct, octane.

TABLE 2. *Tellurite susceptibility of selected plasmids in E. coli J53*

Plasmid	Inc group	Phenotype[a]	MIC for K_2TeO_3 (M)	Plasmid reference
R⁻			10^{-5}	
JJ1	FII	Cm Sm Sp Su Hgr	10^{-5}	45
R57b	C (A-C)	Ap Cm Gm Km Su Hgr	10^{-5}	45
pMG101	?	Ap Cm Sm Su Tc Agr Hgr	10^{-3}	40
JR211	C (A-C)	Ap Cm Gm Km Sm Su Hgr Pmr	10^{-5}	45
R476b	S	Sm Su Tc Asr Hgr	10^{-5}	29
R477	S	Cm Km Sm Su Tc Asr Hgr	10^{-3}	29
R478	S	Cm Km Tc Asr Hgr	10^{-3}	29
R773	FI	Sm Tc Asr	10^{-5}	22
R826	S	Ap Cm Gm Km Sm Tc Asr Hgr Pmr	10^{-3}	29
R828	S	Ap Cm Gm Km Sm Tc Asr Hgr Pmr	10^{-3}	29

[a] Phenotypic abbreviations as for Table 1 and, in addition (resistance): Ap, ampicillin; Agr, silver nitrate; Sp, spectinomycin.

sistant plasmids in *P. aeruginosa*, except CAM and CAM-OCT, gave mercuric ion resistance and many give phenylmercuric acetate resistance as well.

However, other plasmids that gave resistance to arsenic compounds (R45, R46, R48, R476b, and R773), mercuric ion (JJ1, R15, R16a, R40a, Rip55, R57b, Rip135, R222, R391, R476b, R692, R699a, R702, R715b, R742, R769, R839, R902, R906, and R938), or mercuric and phenylmercuric resistance (JR211, R471a, and R830a) did not give tellurite resistance. There was also no association between antibiotic resistance and tellurite resistance in either *E. coli* or *P. aeruginosa*. In particular, the camphor and camphor-octane plasmids of *Pseudomonas* lacked antibiotic resistance but conferred resistance to tellurite. Thus, there was no correlation between resistance to tellurite and resistance to any other previously described metal cation, oxyanion, or antibiotic.

Resistance to selenite, selenate, and tellurate. Since selenite and tellurite are chemically related and since, as noted above, susceptibility to both is affected by growth on reduced sulfur sources (44), strains containing plasmids conferring tellurite resistance were tested for resistance to selenite and selenate and also for

resistance to tellurate, the more highly oxidized form of tellurium. All of the plasmids of *E. coli* or *P. aeruginosa* that provided tellurite resistance also conferred a 10-fold increase in resistance to tellurate. None of the plasmids of *E. coli* or *P. aeruginosa* provided any significant enhancement of selenite or selenate resistance.

Relationship of tellurite resistance to plasmid incompatibility group. The 39 plasmids in *P. aeruginosa* listed in Table 1 belong to at least 11 different incompatibility groups (Jacoby, in press). Only plasmids of group P-2 conferred resistance to tellurite, and no IncP-2 plasmid lacked this property. Thus, in *P. aeruginosa* there was a close association between tellurite resistance and the P-2 incompatibility group.

Fifty-seven plasmids belonging to 23 incompatibility groups were tested in *E. coli* (Table 3). Five conferred resistance to tellurite. Four of these plasmids (R477, R478, R826, and R828) belonged to IncS, but one IncS plasmid (R476b) did not give tellurite resistance. pMG101 also provided tellurite resistance. Its Inc specificity is still under investigation, but pMG101 is not a member of IncS (M. N. Swartz, personal communication). Thus, among the plasmids found in enterobacteria that we have tested, tellurite resistance appears to be an uncommon property and is not associated with a single incompatibility group.

TABLE 3. *Tellurite susceptibility of plasmids in E. coli*

Inc group	Plasmids tested[a]	Plasmid reference
A	RA1	12
C (A-C)	R16a, R40a, Rip55, R57b, JR211, R692, R699a, R715b, R742	4, 20, 45
FI	R386, R773	14, 22
FII	JJ1, R222	45
FIV	R124	24
G	R811	20
H	R27, R726	13
Iα	R64, R483	10, 25
Iγ	R621a	25
Iζ	R805a	13
J	R391	7
K	R387	20
L	R471a, R830a	29
M	R446b, Rip135	26, 45
N	R15, R45, R46, R48, Rip113, R269N-1, R447b	4, 19, 26
O	R[16]	13
P	R702, R751, R839, R906, R938, R995	21, 28, 29, 36
S	R476b, R477, R478, R826, R828	29
T	R402	7
V	R769, R902	21
W	R7K, R388, Sa	7, 11, 23
X	R6K, R485	26
Y	ΦAmp	27
unclassified	pMG101	40

[a] Plasmids conferring tellurite resistance are underlined.

Differential reaction to tellurite between *E. coli* and *P. aeruginosa*. We observed another difference between *E. coli* and *Pseudomonas* with regard to tellurite that was not apparently plasmid related. All of the strains of *P. aeruginosa*, whether tellurite resistant or not, produced a strong odor of garlic when growing on a permissive concentration of tellurite. This odor is characteristic of the methylated forms of metals of groups V and VI of the periodic table (3, 39, 46) and suggests that *Pseudomonas* strains not only reduce but also methylate tellurium. The garlic odor was not found with susceptible or resistance strains of *E. coli* growing on tellurite.

DISCUSSION

Resistance to tellurite and tellurate is not related to any previously described resistance to metal compounds or to antibiotics, and it appears to constitute a new plasmid-determined resistance. Following the suggestions of Novick et al. (41), we propose the phenotypic designation Ter for telluri(a)te resistance.

The biochemical mechanism for the Ter phenotype in both *E. coli* and *P. aeruginosa* remains to be determined. The mechanism appears to be functionally different from alterations in tellurite susceptibility produced by growth of *E. coli* on sulfur compounds, since in this case susceptibility to selenite is affected as well (44), unlike the plasmid-determined resistance. Although both susceptible and resistant cells are capable of reducing tellurite, plasmid-carrying strains may have a more effective reducing system that is tellurite specific. Another possibility is a plasmid-determined alteration in permeability to tellurium oxyanions. These mechanisms are being investigated.

The garlic odor produced by strains of *P. aeruginosa* growing on tellurite is apparently not plasmid mediated. We have not yet identified this product but think that it is probably dimethyl telluride, which is known to have a strong garlic odor (46). Fungi (18) and certain anaerobic bacteria (39) can methylate compounds of selenium, tellurium, and arsenic.

More recently, *Pseudomonas* species have been isolated that can methylate cadmium (31), selenium (5), or tin (C. Huey, F. E. Brinckman, S. Grim, and W. P. Iverson. 1975. *In* Proceedings of the International Conference on Transport of Persistent Chemicals in Aquatic Ecosystems II 73, Ottawa, Canada).

The possibility that tellurite resistance is plasmid mediated in other genera deserves investigation, particularly among enterococci where *S. faecalis*, but not *S. faecium*, is tellurite resistant (47), where tetracycline and tellurite resistance are correlated (1), and where tetracycline resistance can be plasmid mediated (6, 9, 34).

Tellurite resistance appeared to be an uncommon property among the enterobacterial plasmids we tested and was not uniquely associated with a single incompatibility group, although four of the five Ter plasmids belonged to IncS. Among plasmids found in *Pseudomonas*, however, the Ter phenotype seems to be specifically associated with plasmids of the IncP-2 group, and it serves as a rapid screening test for plasmids of this type. It has not been possible to compare the same Ter plasmids in both genera, since IncP-2 plasmids have not been transmitted to *E. coli* by conjugation nor have IncS plasmids been transmitted to *P. aeruginosa* (Jacoby, in press). A correlation between incompatibility specificity and susceptibility to another metal, bismuth ions, has previously been found for plasmids of *S. aureus* (43).

Although tellurite resistance is not uniquely associated with resistance to any other metal compound, it is often found together with resistance to arsenic, mercury, or silver compounds. Clustering of metal ion resistances is also a feature of plasmids in *S. aureus* (42, 43, 49). Since these resistances are not directed against any agent in widespread therapeutic use, the selective pressure promoting their maintenance is not as obvious as that preserving antibiotic resistance. However, the frequency with which metal ion resistance occurs on plasmids in both gram-positive and gram-negative organisms suggests that they may play an important role in the maintenance of plasmids in some natural settings. We recently found that 35% of gram-negative organisms isolated from hospital sewage are tellurite resistant. In addition, 20% of bacteria isolated from city sewage are resistant to tellurite, and of these 80% are gram-negative organisms. Many of these isolates are multiply resistant to antibiotics and other metal ions. We are currently assessing the frequency with which these resistances are transferable.

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