

were married, 36% were single, 18% were separated or divorced, 15% lived together with a partner (multiple answers possible), and 18% were single parents.

Interviewees were generally well-educated with 48% having a college or university qualification. Although the level of education was good, 32% were unemployed (women 29%, men 36%). Thirty percent had full-time or part-time jobs, and 58% had a monthly income under 1000 Euro, which is below the poverty line of 1100 Euro per month in Germany. Thirty-six percent of the participants left their native country to join family, 20% left because of political reasons and 17.5% for economic reasons.

## Results

### Basic knowledge of HIV/AIDS and infection risk

The majority of the immigrants asked felt that they were not well informed on this topic. Only half of those surveyed felt sure what HIV/AIDS was, about 25% were fairly sure, and a further 24% were 'unsure' or 'did not know'. In contrast with German citizens (almost 100% are certain what HIV/AIDS is, [3]), this level of knowledge is very low.

Only 81% knew for certain that infection can occur through sexual contact, 32% thought that transmission could occur by kissing, and 13% believed that sharing a cup or glass could pass infection on. Only 77% knew that sharing needles was a very risky. These results show uncertainty and worry, but also indifference towards possible risk of contracting HIV. More than half the immigrants from eastern Europe were very worried about acquiring HIV. Women from southeast Europe were the group with the least knowledge and least awareness of risky behaviour.

Seventy-three percent of interviewed immigrants had already received information about HIV/AIDS, 49% in a language that they understood well. The men interviewed were generally better informed about HIV/AIDS than the women, and immigrants from sub-Saharan Africa were better informed than southeast Europeans. As with the German population, mass media (television, billboards) was the most important way of getting information (although only 41.5% of immigrants surveyed were informed this way compared with 92% of the German population<sup>2</sup>. As a source of information about HIV/AIDS, personal contacts such as friends were mentioned (25%), and health services (28%) or teachers (20%).

### Knowledge and experience of HIV tests in Germany

Fifty-two percent of immigrants surveyed got information about health services from friends, 38% from family members, and 26% from other immigrants. The knowledge about HIV testing services in Germany was worryingly low. Only 24% knew that an HIV test is free and anonymous in Germany. Only 52% of those who had already undergone HIV testing in Germany could remember being counselled before the test, although this is required. The proportion of people tested who did not receive advice afterwards (or could not remember being advised) was similarly high at 57%.

## Conclusions for HIV/AIDS prevention

The results of this survey indicate that HIV/AIDS educational messages are not reaching immigrants as effectively as German citizens. Important basic knowledge was lacking in many cases, and information was not supplied about HIV testing or even during testing. The existence of free and anonymous testing by health services is too often not known. The fact that female immigrants are mostly informed about HIV by personal contacts makes specific tailored prevention activities required. As well as this, institutions which serve immigrants should be sensitised to this topic. Not least, it would help if HIV health information was advertised regionally, for example in buses, in the main languages of immigrants to that country.

*This article was translated from reference 1 by the Eurosurveillance editorial team.*

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## EMERGENCE AND DISSEMINATION OF A NEW MECHANISM OF RESISTANCE TO AMINOGLYCOSIDES IN GRAM-NEGATIVE BACTERIA: 16S rRNA METHYLATION

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Despite the development of new  $\beta$ -lactams and fluoroquinolones, aminoglycosides are still a very important class of antibacterials for the treatment of severe illness caused by a variety of pathogens, including Gram-negative bacteria, particularly if the pathogen has developed resistance to third-generation cephalosporins.

Aminoglycosides act by causing translational errors and by inhibiting translocation [1]. Their target sites include ribosomal domains in which the accuracy of codon-anticodon is assessed [2]. In particular, they bind to a highly conserved motif of 16S RNA which leads to alterations in ribosome function [3]. There are four known mechanisms of resistance to aminoglycosides in bacterial human pathogens:

1. decreased intracellular accumulation of the antibiotic by altering the outer membrane permeability, less inner membrane transport, or active efflux;
2. enzymatic modification of the drug, primarily through *N*-acetylation, *O*-nucleotidylation, or *O*-phosphorylation, which is the most common;
3. modification of the target by mutation in ribosomal proteins or in 16S RNA;
4. trapping of the drug.

Microorganisms that produce aminoglycosides have developed an additional pathway to avoid suicide. This self-defence mechanism involves post-transcriptional methylation of ribosomal RNA using *S*-adenosyl-methionine as a cofactor [4].

The *armA* (aminoglycoside resistance methyltransferase) gene, which confers resistance to 4,6-disubstituted deoxystreptamines (kanamycin, amikacin, isepamicin, gentamicin, netilmicin, sisomicin, and tobramycin) and to the structurally unrelated compound fortimicin, was initially characterised in *Klebsiella pneumoniae* BM4536. This was isolated from a urinary tract infection in 2000 in France. Possession of the *armA* gene did not confere resistance against the 4,5-disubstituted deoxystreptamines (lividomycin, neomycin, paromomycin, ribostamycin).

## TABLE

Minimum inhibitory concentrations (MICs) of various aminoglycosides against *E. coli* with and without plasmid pIP1204 carrying *armA*

Strain	AMI	MICs (mg/L) <sup>a</sup>						
		GEN	ISE	NET	TOB	APR	PAR	STR
BM694	2	0.5	0.5	0.5	0.5	2	4	4
BM694 (pIP1204)	1024	256	1024	256	256	2	4	8

a- Abbreviations: AMI, amikacin; APR, apramycin; GEN, gentamicin; ISE, isepamicin; NET, netilmicin; TOB, tobramycin; PAR, paromomycin; STR, streptomycin.

The *armA* gene was located on the self-transferable IncL/M plasmid pIP1204 of about 90 kb which also encodes extended spectrum  $\beta$ -lactamase CTX-M-3 [5].

The *armA* gene was detected in 12 isolates among 34 enterobacteria from 3 hospitals in Paris, France, 2 hospitals in Sofia, Bulgaria, and from our laboratory collection. The bacteria collected by the laboratory were likely to produce a CTX-M enzyme since they were more resistant to cefotaxime than to ceftazidime. The isolates containing *armA* were *C. freundii* (3 strains out of 3), *Enterobacter cloacae* (1/1), *E. coli* (2/19), *K. pneumoniae* (4/5), *Salmonella enterica* serotype Enteritidis (1/1), and *Shigella flexneri* (1/1) [6]. Transfer of high-level aminoglycoside resistance from the 12 *armA* containing strains to *E. coli* was obtained by conjugation. Using disc diffusion susceptibility tests and polymerase chain reaction with specific primers and sequence analysis, the *E. coli* transconjugants were found to express resistance to 4,6-disubstituted deoxystreptamines mediated by *armA* after acquiring an IncL/M plasmid. More recently, presence of *armA* on a self-transferable IncN plasmid in an *E. coli* pig isolate from Spain has been reported [7].

Conjugation, analysis of DNA sequences, PCR mapping, and plasmid conjugation experiments, indicated that the *armA* gene was part of the functional transposon Tn1548. The 16.6-kb transposon is a typical composite element flanked by two copies of IS6 in direct orientation [8]. Functionality of Tn1548 under natural conditions was confirmed by its presence on plasmids of different incompatibility groups [5,7].

Taken together, these data support the notion that spread of *armA* results from both conjugation and transposition. This combination accounts for the worldwide documented dissemination of aminoglycoside resistance by 16S rRNA methylation in enterobacteria from human or animal origin and in *Acinetobacter baumannii* in Europe and in Asia [6,7,9,10]. Two other closely related 16S rRNA methylases, RmtA in *Pseudomonas aeruginosa* isolates and RmtB in enterobacteria, have been recently reported in Japan [11, 12]. It therefore appears that post-transcriptional modification of 16S rRNA can confer high-level resistance to all the clinically available aminoglycosides, except streptomycin, in Gram-negative human pathogens.

The findings discussed here indicate that global dissemination of *armA* in Gram-negative pathogens has occurred and demonstrate the importance of maintaining ongoing surveillance of aminoglycoside resistance of human and animal isolates.

Strains suspected to harbour the *armA* gene can be sent for analysis to Marc Galimand, Unité des Agents Antibactériens, Institut Pasteur, 25 rue du Docteur Roux, 75724 Paris Cedex 15, France.

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## STREP-EURO: PROGRESS IN ANALYSIS AND RESEARCH INTO SEVERE STREPTOCOCCAL DISEASE IN EUROPE, 2003-2004

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The Strep-EURO project (<http://www.strep-euro.lu.se/>) collects data on severe group A streptococcal (GAS) disease in Europe. It was launched in September 2002 to improve understanding of severe GAS disease in Europe so that an integrated picture of these infections can be achieved [1], and consists of over 50 members from 11 European countries. We briefly summarise below progress on the project to date, including some preliminary results from the participating countries: Germany, Cyprus, Czech Republic, Denmark, Finland, France, Greece, Italy, Romania, Sweden, and the United Kingdom.

During the first phase of the project, a European case definition was agreed on, along with items to be included in a clinical and epidemiological questionnaire. The inclusion criteria were the isolation of GAS from blood or another normally sterile site of the body, or from a non-sterile site in the presence of a clinical diagnosis of streptococcal toxic shock syndrome (STSS) or necrotising fasciitis (NF), as defined previously [2].

Enhanced surveillance of GAS invasive disease began on 1 January 2003 for a two year period. Collected isolates were characterised by both serology (T, OF and M typing), and molecular tools (e.g., *emm*-sequencing, superantigen and antimicrobial drug resistance gene identification, and strain comparison by pulsed field gel electrophoresis (PFGE) and multilocus sequence typing (MLST)). Strains were screened for antibiotic susceptibility and minimum inhibitory concentrations determined by E test. To achieve a standardization of methods and assessment of capabilities in the different laboratories, two sets of external quality assessment (EQA) strains for antibiotic susceptibility testing and one set for typing were sent to participating laboratories. Data file specifications for collection of patient and microbiological data were developed by consensus, and each partner submitted their results to a central database in Finland.

Over 5000 cases were identified in the first 18 months, considerably more than had been anticipated. Around 3000 cases were from the United Kingdom, yielding an incidence for 2003 of 3.8/100 000 for that country. Similar incidences were documented in Sweden, Denmark and Finland, but the incidence was considerably lower in the other participating countries. This may be related to the fact that surveillance in northern Europe approached total coverage, whereas in the remaining countries, geographical coverage was limited and not always definable.

The type distribution of GAS also varied markedly. In a few countries, types 1, 3 and 28 were predominant; however, an overall increase of new invasive types (*emm* 77, 81, 82, 89) was noticeable. Relatively high rates of MLS antibiotic (macrolide, lincosamide, streptogramin B) resistance in some countries (France, Italy) and very frequent tetracycline resistance was found in almost all countries. In the UK, intravenous drug use (IDU) was found to be a major risk factor, which is consistent with a previously reported trend [3]. In France, the spread of a clone of GAS, type 28, resistant to MLS drugs and bacitracin, associated with puerperal sepsis, was reported [4]. Early results from the pathogenesis work package have identified that low antibody levels against some newly described cell wall-attached