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Antibiotic resistance patterns of *Escherichia coli* isolated from HIV-sero positive adults at Mbagathi District Hospital, Nairobi, Kenya.

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ABSTRACT
Objectives: To determine changes in antibiotic resistance patterns in *E. coli* from persons living with HIV/AIDS (PLWHA). *Methodology and results:* A total of 264 strains of *E. coli* were isolated from PLWHA. The isolates were tested for antimicrobial susceptibility by Kirby Bauer disk diffusion technique. Out of the 216 (81.81%) *E.coli* from PLWHA and taking antibiotics (HIV^+A^+), 209-(96.76%), 198-(91.67%) and 188-(87.04%) isolates were resistant to trimethoprim-sulphamethazole, tetracycline and ampicillin respectively. All isolates from PLWHA not taking antibiotics(HIV^+A^-) were sensitive to fluoroquinolones and nalidixic acid but thirty nine (83.33%) of the them were resistant to trimethoprim-sulphamethazole.The most prevalent resistance was to trimethoprim-sulphamethazole and ampicillin.
Conclusion and application of findings: prolonged exposure to antibiotic makes PLWHA harbour strains of *E. coli* that are significantly more resistant to antibiotics with varying resistance patterns. The findings of this study should have major implications on public health policy guidelines on managing antimicrobial resistance in PLWHA, antimicrobial resistance detection modules and intervention schemes. It can also help in planning regular monitoring of antibiotic resistance patterns in order to provide the basis for developing rational prescription guidelines.
Key words: Acquired immunodeficiency syndrome, opportunistic infections, antibiotics, *Escherichia coli,* resistance

INTRODUCTION
Human immunodeficiency virus is the causative agent of Acquired Immunodeficiency Syndrome (AIDS), an acute and often fatal disease of human beings, which is pandemic and has reached almost all parts of the world (UNAIDS, 2007). Among its most devastating effects is compromising the immune system hence exposing the infected persons to opportunistic infections (OIs) which in turn makes HIV patients to require prolonged antibiotic use. Prolonged usage of antibiotics has been thought to affect resistance patterns in strains of *E. coli* in the gut of persons living with HIV/AIDS (PLWHA).

AIDS was first reported in the United States in 1981 (CDC, 2007) and since then it has become the most devastating disease facing humanity. By the end of the year 2007, it was estimated that 33.2 million people globally were infected with HIV, with one third of the infected persons being between 15-24 years old. Ninety

five percent of new infections occur in the developing world and almost 50% are women (UNAIDS, 2007). Death related to AIDS globally was reported to be 2.1 million in 2007, comprising of 1.7 million adults and 330,000 children less than 15 years (NASCOP, 2007).

The first HIV/AIDS case in Kenya occurred in 1978 but the Ministry of Health (Obel et al., 1984) officially reported it six years later. The first sentinel survey report in 1990 showed that the HIV infection prevalence in Kenya was 5.1% which increased steadily from 6.3% in 1991 to 13.4% in the year 2000 (MOH, 2005; NACC, 2007). The National HIV prevalence showed substantial decline to 10.4, 6.7 and 5.1% in 2002, 2003 and 2007, respectively before increasing to 7.4% in June 2008 (KAIS, 2008). Currently, the estimated prevalence of HIV infection in adults aged 15 to 49 years in Kenya is as shown in figure 1

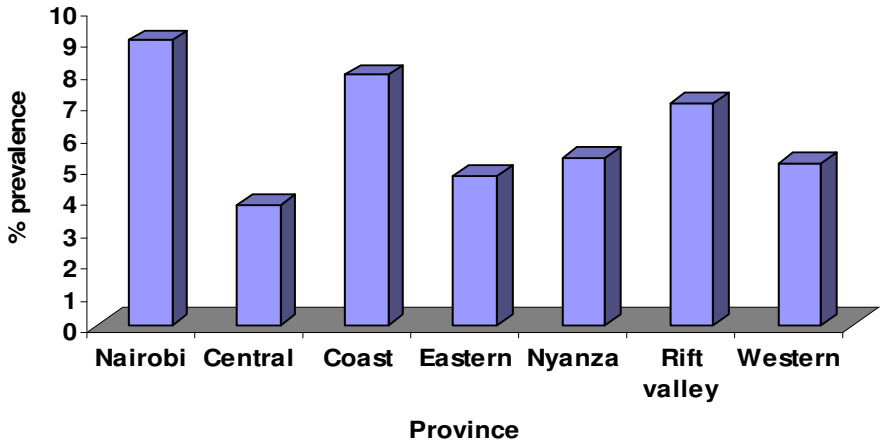

Figure 1: National estimates of HIV in Kenya per province

Kenya is therefore home to over 2 million persons living with HIV infections who may succumb to AIDS, which renders them susceptible to opportunistic infections. Several antibiotics are recommended for prophylaxis and treatment of these infections but their usage has been thought to cause widespread emergence of drug resistance among strains of *E. coli* and hence exacerbated the problem of controlling microbes in PLWHA and resurgence of bacterial diseases worldwide**.** Such resistance is of great concern as the organisms may become infectious and serve as an important reservoir for resistance genes, which can be transferred to potential pathogens. Available antibiotics would be rendered ineffective thus plunging the country into a possible post antibiotic resistance era. Therefore, there is need to isolate and identify strains of *E. coli* from PLWHA and determine the antibiotic resistance patterns developed to commonly used antibiotics.

MATERIALS AND METHODS

Study area: The study was carried out in Nairobi, Mbagathi District Hospital (MDH) that is an Infectious Disease Hospital (IDH) with a Comprehensive Care Center (CCC) for HIV infected people and at The Kenya Medical Research Institute (KEMRI). MDH serves as a district Hospital for Nairobi residents with the largest population served being residents of Kibera and Dagoreti divisions. The hospital attends to about 200 outpatients daily and has a bed capacity of 169. The average number of inpatients is 90-120 per day and over 50% of the inpatients have been reported to have HIV/AIDS (NASCOP, 2008).

Study population: The study population consisted of persons living with HIV/AIDS (PLWHA) and attending

MDH Comprehensive Care Center (CCC). Admission of PLWHA into the study was based on patients showing difficult in breathing with elevated body temperatures, and vomiting but without gastroenteritis or profuse diarrhoea. Other observations included persistent coughing, wasting and oral thrush. All cases had previously been confirmed to be serologically positive for HIV.

Ethical approval*:* The Health, Safety and Ethics committee of the Kenya Medical Research Institute (KEMRI), Nairobi, Kenya granted ethical approval for this study. Informed consent was obtained from the study subjects before collection of stool samples. Confidentiality and anonymity were strictly observed.

Stool sample collection*:* In this study, which was conducted from March to December 2007, 264 patients were recruited and their stool sampled randomly. A total of 48 samples were taken from HIV positive patients not taking antibiotics (HIV^+A^-) while 216 were taken from HIV positive patients taking antibiotics (HIV^+A^+). The samples were collected in sterile wide mouth containers. Demographic data on age, sex and antibiotics used in the preceding two years were obtained from each individual and from the individual's patient's files.

Isolation and identification of *E. coli*: The samples were inoculated on MacConkey agar and Selenite-F broth and the seeded plates incubated for 24h aerobically at 37^0C. Cultures in Selenite- F broth were subcultured on MacConkey agar and incubated for 24h aerobically. Colony morphology of the bacteria on the cultured plates (pink colored lactose fermenting colonies) was described and used to provide presumptive identification of *E. coli*. Gram staining of the colonies was done and the Gram reaction of the bacteria recorded. Further identification of the Gram-negative lactose fermenting bacilli was done using biochemical tests (Monica, 1984). The bacteria were inoculated in triple sugar iron agar (Oxoid Ltd, Basingstoke, UK), Sulphur indole motility agar (Oxoid Ltd, Basingstoke, UK), Simmons citrate agar (Oxoid Ltd, Basingstoke, UK), MR/VP broth and urea agar (Oxoid Ltd, Basingstoke, UK) and their reaction to oxidase reagent (1% dimethyl-p-phenelyne-diamine dihydrochloride) noted. Confirmatory tests were done by the use of API 20E (bioMěriux, Basingstoke, United Kingdom) system in accordance with the manufacturer's instruction. API Lab(R) software was used to interpret API-20E reactions.

Susceptibility testing: Bacterial susceptibility to antibiotics was determined on Muller Hinton agar (Oxoid, Basingstoke, UK) by standard disk diffusion procedures (Bauer *et al*., 1996). The following antibiotics were tested: amoxicillin/clavulanic acid (25 µg), sulphamethazole-trimethoprim (25 µg), nalidixic acid (30 µg), gentamicin (10 µg), nitrofuratoin (30 µg), norfloxacin (30 µg), tetracycline (30µg), cefuroxime (30 µg), ceftazidime (20 µg), chloramphenical (30 µg), ampicillin (10 µg) and ciprofloxacin (5.0 µg). Control samples *Escherichia coli* ATCC ® 25922 were run simultaneously with the test organisms. The results were interpreted according to the standard recommendations of the National Committee for Clinical Laboratory Standards (NCCLS, 2002).

Statistical analysis: Differences in prevalence of resistant isolates from PLWHA taking and not taking antibiotics were analyzed by the Z- test for unmatched samples using SPSS, version 16.0 software and a P value <0.001 was considered statistically significant. The null (H_0) hypothesis was that persons living with HIV/AIDS and on prolonged antibiotic exposure harbor strains of *E. coli* that are significantly more resistant.

RESULTS

Prevalence of resistant E. coli isolated from PLWHA: The overall mean difference in resistance levels of *E. coli* isolated from PLWHA is shown in figure 2. Isolates from HIV persons taking antibiotics (HIV^+A^+) and persons not taking antibiotics (HIV^+A^-) did not show any significant difference in resistance to ciprofloxacin, norfloxacin and trimethoprim-sulphamethazole while showed significant difference in resistance to nitrofurantoin (29.17%), nalidixic acid (19%), ceftazidime (39.35%) ,gentamicin (36.57%), cefuroxime (43.09%), tetracycline (45.84%), ampicillin (45.47%) chloramphenical (45.67%) and amoxil/clavulanic acid (50.0%).

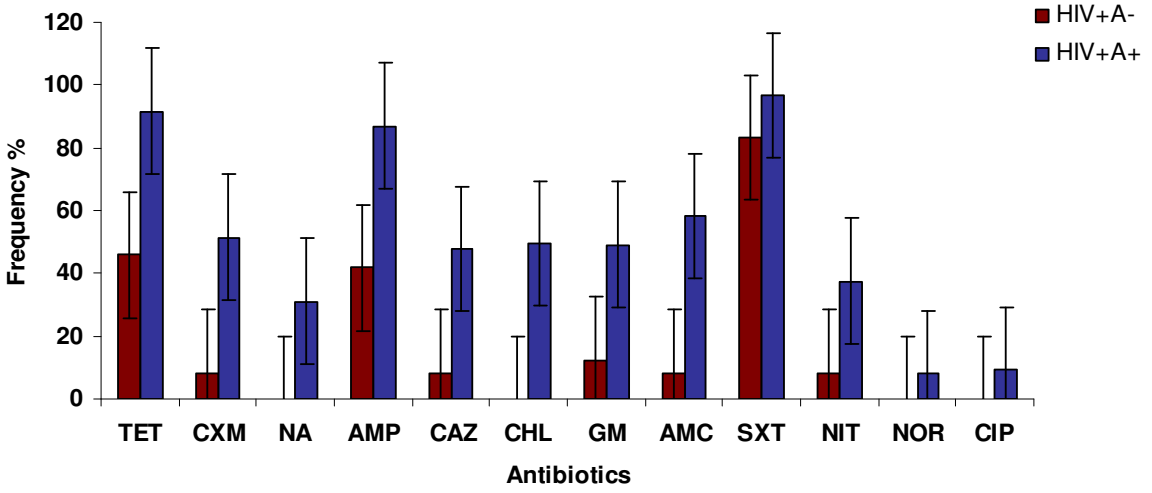

Figure 2: Comparison of the prevalence of antibiotic resistant *E. coli* strains isolated from HIV positive (HIV+) patients: taking (A+) and not taking antibiotic medication (A-). TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin, SXT-trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

During the study period, 264 patients were recruited and the details are as shown in the Table 1 .The isolates were clustered depending on the period of antibiotic use.

Table 1: Number of isolates per the period of antibiotic use

Period	HIV^+A	$HIV^+A^{+\ 0.5}$	$HIV^+A^{+\ 1.0}$	$HIV^+A^{+\ 1.5}$	$HIV^+A^{+\ 2.0}$
Number tested (*n*)	48(18.18%)	40(15.15%)	60(22.73%)	52(19.70%)	64(24.24%)
No. of isolates Resistant	42(16.27%)	40(15.50%)	60(23.25%)	52(20.15%)	64(24.80%)

HIV^+ - Human Immunodeficiency Virus positive, HIV^+A^- - and not taking antibiotic medication, $HIV^+A^{+\ 0.5}$ –and taking antibiotics for six months, $HIV^+A^{+\ 1.0}$ – and taking antibiotics for one yr, $HIV^+A^{+\ 1.5}$ – and taking antibiotics for one and a half years, $HIV^+A^{+\ 2.0}$ – and taking antibiotics for two years.

Antimicrobial resistance patterns (Antibiogram) of *E. coli* isolated from PLWHA stool samples: Thirty-three resistance patterns were assessed in the five categories of *E. coli* with Sxt^R Tet^R Amp^S Amc^S Gm^S Cxm^S Caz^S Na^S Chl^S Nit^S Nor^S Cip^S (isolates resistant to at least two antibiotics) being the most prevalent multiresistance pattern. The antibiotics were clustered into patterns depending on the resistance frequency (Table 2). Sxt^R Tet^S Amp^R Amc^S Gm^S Cxm^S Caz^R Na^S Chl^S Nit^S Nor^S Cip^S was the second most prevalent pattern in *E. coli* isolated from HIV + not on antibiotic use. While Sxt^R Tet^R Amp^R Amc^S Gm^S Cxm^S Caz^R Na^S Chl^S Nit^S Nor^S Cip^S and Sxt^R Tet^R Amp^R Amc^R Gm^R Cxm^R Caz^R Na^S Chl^S Nit^S Nor^S Cip^S were the second most prevalent resistant patterns in isolates from HIV + patients on antibiotic use with frequency of 30.5 and 6.3%. Multi drug resistance was detected in 75% of all HIV^+A^- *E. coli* strains, 90% of $HIV^+A^{+\ 0.5}$ strains and 100% of isolates from $HIV^+A^{+\ 1.0}$, $HIV^+A^{+\ 1.5}$ and $HIV^+A^{+\ 2.0}$.

Antibiotic resistance pattern of *E. coli* isolated from PLWHA on different periods of antibiotic use: Strains of *E. coli* isolated from HIV, not on antibiotic therapy did not show resistance to nalidixic acid, chloramphenical, norfloxacin and ciprofloxacin . But they demonstrated resistance of 8.33% to cefuroxime, ceftazidime and nitrofurantoin with a resistance outline of Sxt Tet Amp Amc Gm Cxm Caz Nit Na Chl Nor Cip.

Table 2: Antimicrobial resistance patterns of *E. coli* isolated from Persons Living with HIV and AIDS at Mbagathi District Hospital

Resistance pattern	Frequency of isolation	Percentage Frequency
SXT	11	4.26
SXT, TET	11	4.26
SXT, AMP	8	3.10
SXT, TET, AMP	14	5.43
SXT,AMP, CAZ	8	3.10
SXT, TET, CXM	6	2.33
SXT, TET, AMP, GM	12	4.65
SXT,TET,AMC, CXM	11	4.26
SXT,TET, AMP, AMC	7	2.71
SXT, TET, AMP,AMC, GM	12	4.65
SXT, AMP, GM,CXM, CAZ	9	3.49
SXT, TET, AMP, CAZ, NIT	8	3.10
SXT, TET, AMP, AMC, GM	12	4.65
SXT, TET, AMP, AMC, GM, CHL	11	4.26
SXT, TET, AMP, AMC, CAZ, CXM	6	2.33
SXT, TET, AMP, GM, CXM, NIT	4	1.55
SXT, TET, AMP, AMC, GM, CXM, CAZ	14	5.43
SXT, TET, AMP, CXM, CAZ, NIT, NOR	8	3.10
SXT, TET, AMP, AMC, CXM, CAZ, NIT	8	3.10
SXT, TET, AMC, GM, CXM, CAZ, NA	8	3.10
SXT, TET, AMP, AMC, CXM, CAZ, CHL	8	3.10
SXT, TET, AMP, AMC, GM, CAZ, NIT, CHL	11	4.26
SXT, TET, AMP, AMC, GM, CXM, CAZ, CHL	4	1.55
SXT, TET, AMP, GM, CXM, CAZ, NIT, NA	6	2.33
SXT, TET, AMP, AMC GM, CXM, CAZ, CHL, NA	9	3.49
SXT, TET, AMP, AMC GM, CXM, CAZ, NIT, NA	6	2.33
SXT, TET, AMP, AMC GM, CXM, CAZ, CHL, NOR	3	1.16
SXT, TET, AMP, AMC GM, CXM, CAZ, CHL, NIT NOR	8	3.10
SXT, TET, AMP, AMC GM, CXM, CAZ, NA CHL, NIT	4	1.55
SXT, TET, AMP, AMC, CXM, CAZ, NA, CHL, NOR, CIP	3	1.16
SXT, TET, AMP, AMC, GM, CXM, CAZ, NA, CHL, NIT NOR,	5	1.94
SXT, TET, AMP, GM, CXM, CAZ, NA, CHL, NIT NOR, CIP	2	0.78
SXT, TET, AMP, AMC, GM CXM, CAZ, NA, CHL, NIT NOR, CIP	1	0.39

TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin, SXT-Trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

The isolates showed a slightly increased resistance of 12.5% to augmentin and gentamicin while trimethoprime-sulphamethazole, tetracycline and ampicillin showed a resistance a bove 50%. (Fig 3).

The antibiotic resistance pattern developed by *E. coli* isolates from HIV persons taking antibiotics for six months ($HIV^{+}A^{-0.5}$) was Sxt Tet Cxm Amp Amc Caz Nit Gm Na Chl Nor Cip (Fig 4). Isolates did not show resistance to norfloxacin and ciprofloxacin but showed a slight resistance of 5% to chloramphenical and nalidixic acid. The isolates showed resistance of less than 50% to augmentin, ampicillin, cefuroxime, ceftazidime, amoxicillin-clavulanic acid and nitrofurantoin and 87 and 60% resistance to trimethoprim-sulphamethazole and tetracycline, respectively.

Resistance developed in isolates from HIV persons taking antibiotic for one year ($HIV^{+}A^{+1.0}$) varied from 3.33% for norfloxacin and ciprofloxacin to 90% in ampicillin. Isolates showed a resistance of 97.67% to both tetracycline and sulphamethazole-trimethoprim (Fig 5) but demonstrated a resistance of less than 50% to nalidixic acid, cefuroxime, chloramphenical, augmentin, ceftazidime, gentamicin and nitrofurantoin with a resistance pattern of Sxt Tet Amp Amc Cxm Chl Caz Nit Gm Na Nor Cip.

Resistance developed by bacteria isolated from patients taking antibiotics for eighteen months ($HIV^{+}A^{+1.5}$) was 3.33% for norfloxacin and ciprofloxacin (Fig 6). The resistance pattern for isolates in this group was Sxt Tet Amp Gm Cxm Amc Nit Chl Caz Na Nor Cip. *E. coli* showed a resistance of 32.69% to nalidixic acid, 40% to ceftazidime and 50% to both chloramphenical and nitrofurantoin. Isolates demonstrated a slightly higher resistance of between 55 and 70% to cefuroxime, gentamicin and augmentin, while all the isolates in this cluster were 100% resistant to tetracycline, ampicillin and trimethoprim-sulphamethazole

E. coli isolates from patients taking antibiotics for 24 months ($HIV^{+}A^{+2.0}$) showed a significant difference in resistance to all antibiotics (Fig 7). The antibiotic resistance pattern was Sxt Amp Tet Chl Na Caz Cxm Gm Amc Nit Cip Nor. The isolates showed the least resistance to nitrofurantoin, norfloxacin and ciprofloxacin (less than 50%) while to cefuroxime, nalidixic acid, gentamicin and amoxicillin-clavulanic acid, resistance was ranging from 50 to 69%. Resistance was 96.87% to both chloramphenical and tetracycline while all the isolates were resistant to sulphamethazole-trimethoprim and ampicillin.

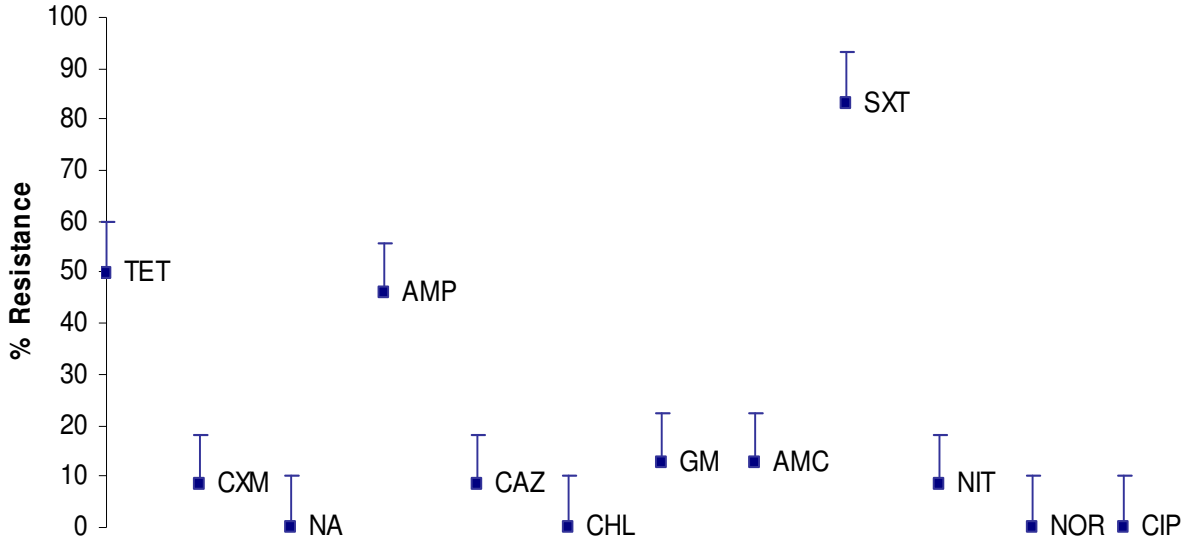

Figure 3: Frequency of resistance developed by *E. coli* isolated from HIV positive patients not taking antibiotics ($HIV^{+}A^{-}$). TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin, SXT-Trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

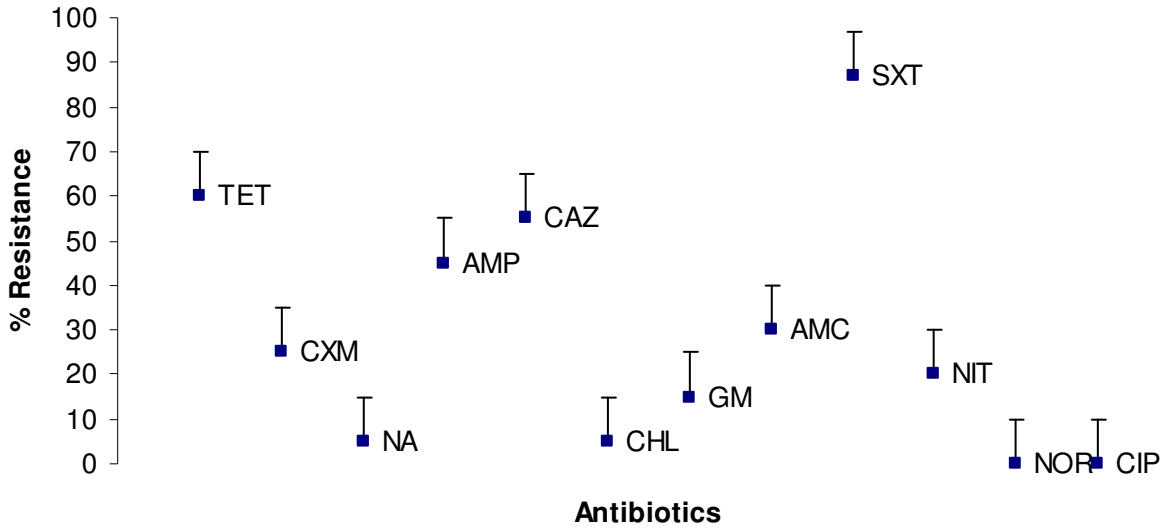

Figure 4: Frequency of resistance developed by *E. coli* to antibiotics after six months of use. ($HIV^{+}A^{+0.5}$). TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin, SXT-Trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

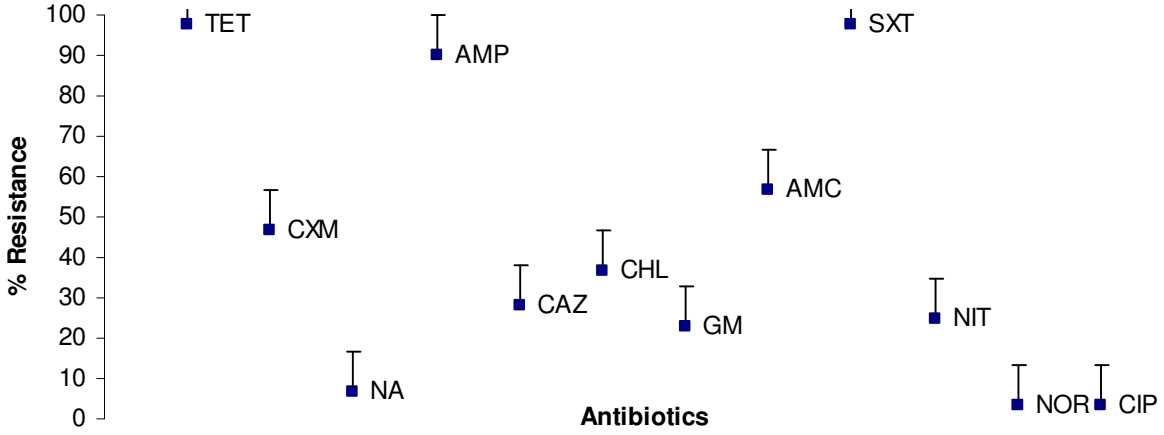

Figure 5: Frequency of resistance development by *E. coli* to antibiotics after twelve months of use ($HIV^{+}A^{+1.0}$). TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin, SXT-Trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

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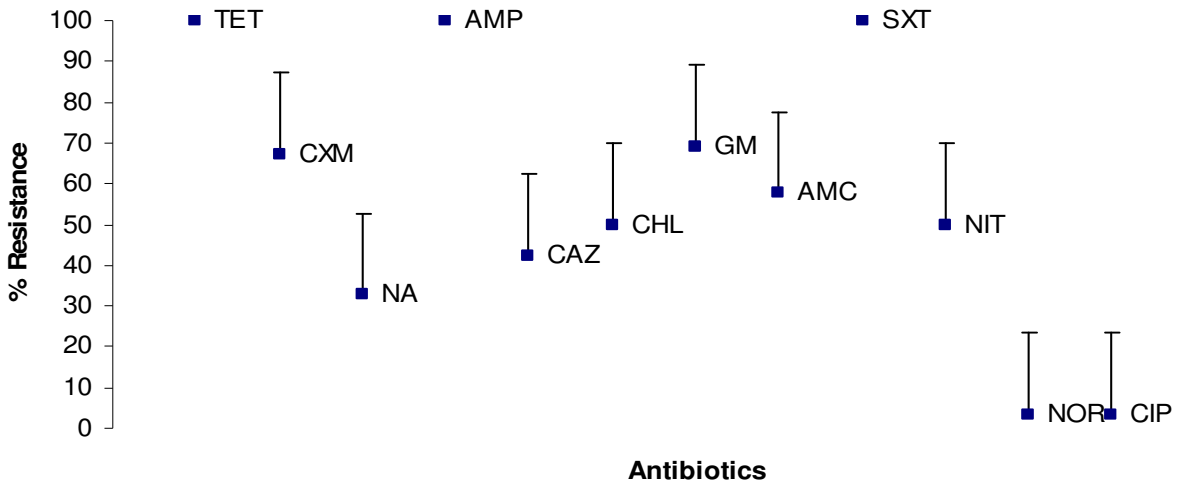

Figure 6: Frequency of resistance developed by *E. coli* to antibiotics after eighteen months of use ($HIV^{+}A^{+1.5}$). TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin, SXT-Trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

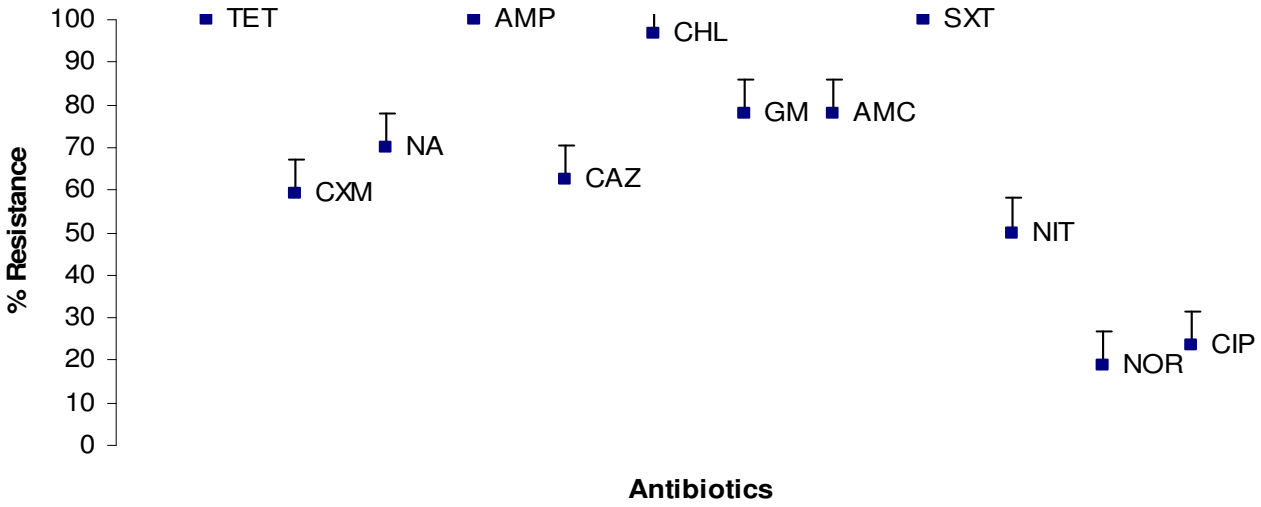

Figure 7: Frequency of resistance development by *E. coli* to antibiotics after twenty four months of use ($HIV^{+}A^{+2.0}$).TET-Tetracycline, CXM-Cefuroxime, NA-Nalidixic acid, CHL-Chloramphenical, GM-Gentamicin, AMC-Augmentin,SXT-Trimethoprim-sulphamethazole, NIT-Nitrofurantoin, NOR-Norfloxacin, CIP-Ciprofloxacin

DISCUSSION

Comparison in E. coli isolates from HIV-sero positive persons taking and not taking antibiotics did not show any significant difference to ciprofloxacin, norfloxacin and trimethoprim-sulphamethazole. The reduced resistance to ciprofloxacin and norfloxacin may be due to decreased use of the antibiotics because of their toxicity, being expensive and used only on prescription. Trimethoprim-sulphamethazole (SXT) is a first line treatment antibiotic for upper respiratory infections. The antibiotic is relatively cheap and can illegally be acquired over the counter even without prescription. These factors can therefore contribute to a buse of SXT and hence it is decreased significant difference in the two populations. SXT is the most commonly used

antibiotic since the advent of HIV/AIDS (Maartens, 2002) and is used as a prophylactic drug and for treatment of most bacterial infections. UNAIDS recommended the use of SXT on a wide scale in Africa in 2000 among PLWHA (UNAIDS, 2007).

This has already been started in Kenya by doctors (MSF Belgium), who are working from Mbagathi District Hospital and by the Center for Disease Control (CDC) in western Kenya hence it's least significant difference in resistance Thirty three different resistance patterns were demonstrated from all the isolates with Sxt(R) Tet (R) Amp(s) Amc(s) Gm(s) Cxm(s) Caz(s) Na(s) Chl(s) Nit(s) Nor(s) Cip(s) being the most prevalent. Antibiotic resistance patterns differed in E. coli isolated from the five categories of patients depending on the exposure time. But isolates from HIV patients who were not taking antibiotics (HIV+A-) and those taking antibiotics for six months (HIV+A+0.5) has almost the same prevalent resistance patterns in that the organisms were highly resistant to trimethoprim-sulphamethazole and least resistant to ciprofloxacin. This could probably have been because of E. coli having not been exposed to the antibiotics for a long time.

The antibiotic resistant pattern for isolates from HIV persons taking antibiotics for one year remained almost the same as for isolates from HIV persons taking antibiotics for six months (Sxt Tet Amp Amc Cxm Chl Caz Nit Gm Na Nor Cip) except for ampicillin which turned out to be highly resistant than Cefuroxime. Ampicillin being an inexpensive and easily available antibiotic, its resistance rate could have increased as a result of increased use. The antibiotic resistance pattern of E. coli isolated from HIV persons taking antibiotics for one and a half years was Sxt Tet Amp Gm Cxm Amc Nit Chl Caz Na Nor Cip. More isolates were resistant to gentamicin, nitrofurantoin and chloramphenical than for the HIV persons taking antibiotics for one year. The antibiotic resistance pattern for isolates from HIV+A+2.0 was Sxt Amp Tet Chl Na Caz Cxm Gm Amc Nit Cip Nor. E. coli isolates in this group of PLWHA were significantly more resistant to all the antibiotics compared to isolates from HIV persons not taking antibiotics (HIV+A-) . Generally resistance developed to first line antibiotics was high compared to quinolones and fluoroquinolones that are expensive and used only on prescription. Prophylactic and chemoprophylactic use of antibiotics by PLWHA improves persons' lives but the impact of these antibiotics on development of antibiotic resistant strains in E. coli and circulating bacterial pathogens needs to be regularly monitored in order to curb increased mullti-drug resistant (MDR) strains and reduce their spread. This concern should receive equal attention if not more than what is given to other components of HIV/AIDS awareness campaign. Susceptibility testing should also be given more attention like other components of HIV in all hospitals and research institutes. Massive use of antibiotics should be augmented with relevant programs on knowledge, attitude, hygiene and practice to raise awareness on drug resistance. The option of food supplements to the current antibiotic therapy, which requires prolonged consumption with the possible risk of antibiotic resistance can be considered as this could help boost the immune system thus reducing the problem of opportunistic infections (OIs). Other routes of inoculation or mode of administration of the antibiotics could also be considered like intrtramuscular or intradermal as this could reduce the possibility of normal intestinal flora getting into direct contact with the antibiotics.

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