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Full Length Research Paper

Survey of the efficacy and quality of some brands of the antibiotics sold in Calabar Metropolis, South-south region of Nigeria

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This study reports the survey of the level of efficacy of some brands of the antibiotics sold in Calabar, Nigeria. This survey was carried out using the agar diffusion technique. Ten brands of 5 different antibiotics were bought from different pharmacy shops in Calabar metropolis and tested against *Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Staphylococcus aureus and Streptococcus pyogenes* to determine their efficacies. The test antibiotics were 2 brands each of ampicillin, chloramphenicol, erythromycin, septrin and vancomycin. This survey measured the ability of these antibiotics to inhibit bacterial growth *in vitro*. The potency or activity per milligram of a chemotherapeutic agent is expressed on the basis of the lowest concentration of minimal inhibitory concentrations (MICs) or higher zones of inhibition. From the results, the overall mean zones of inhibition for the test organisms ranged from 20.2 – 33.3 mm, with 21.9 mm for *E. coli*, 26.4 mm for *K. pneumoniae*, 20.2 mm for *P. aeruginosa*, 33.3 mm for S. *aureus and* 21.7 mm for S. *pyogenes.* Our study showed that there were no significant differences (P > 0.05) between the mean zones of inhibition of the test antibiotics and standard controls tested against the test organisms except for *P. aeruginosa* and *K. pneumoniae*. The 2 brands of ampicillin and erythromycin were significantly (P = 0.027; P = 0.038) less effective against *P. aeruginosa* compared to the standard controls while for *K. pneumoniae*, the 2 brands of chlorampenicol and erythromycin were significantly (P = 0.049; P = 0.057) less effective compared to the standard controls. The results showed that the level of potency of these antibiotics vary according to the different manufacturers. Indeed, the findings of this survey confirm that some of the brands of antibiotics (ampicillin, chloramphenicol and erythromycin) sold in Nigeria may be fake or adulterated and do not contain the acclaimed quantity of active ingredients, which is a reflection of what goes on in many developing countries, in particular, in sub-Saharan Africa.

Key words: Active ingredients, antibiotics, efficacy, quality, selective toxicity, survey, zones of inhibition.

INTRODUCTION

Antibiotics are specific chemical compounds derived from or produced by microorganisms that even in small amounts can selectively inhibit the life processes or growth of other micro organisms. The concept of antibiotic (against life) substance was put forth by Vuillemin in 1889, but the formal definition as recognized today,

would not be introduced until 1942 by Waksman. Thus, antimicrobials such as antibiotics are chemical substances produced by microorganisms that can inhibit the growth of, or kill other microorganisms (Prescott et al., 2005; Okonko et al., 2008).

Today, some 5,000 antibiotics are known. Only about 1,000 of these have been carefully investigated and about 100 are currently used to treat infections (Dixon, 1994). Most are produced by actinomycetes, molds and bacteria. Throughout the last 50 years, a lot of scientific journals have been disseminating information on drug discovery and evolution. Hundreds of articles on infectious disease management have kept physicians abreast of both advancements and problems. A new vocabulary has emerged: plasmids, transposons, promiscuous DNA, mutator alleles, cairnsian mutation and many other esoteric terms. It is a whole new ball game (Hoel and Williams, 1997). Also, the discovery and development of the betalactam antibiotics are among the most powerful and successful achievements of modern science and technology (Okonko et al., 2008). The present day use of the term antibiotics was proposed by Naksman in 1945 as those chemical substances of microbial origin which in small amounts exert antimicrobial activity (Pelczar et al., 1993). Antibiotics are usually of microbial origin but some have come from higher forms of life and chemotherapeutic agents made synthetically. Their selective toxicity means a low toxicity for host cells and high toxicity for parasites (Melmon et al., 1989).

The sources of antibiotics include those obtained naturally from microorganisms such as antibiotics obtained from culture extracts and filtrates of fungi (examples, penicillins and cephalosporins), bacteria such as *Streptomyces spp*, *Bacillus spp*, etc (examples, rifampicin, aminoglycosides, chloramphenicol, erythromycin and tetracyclines) or those antibiotics obtained through synthetic and semisynthetic processes. Semisynthetic antibiotics involve the fermentation of certain parts of the microbial molecules using the appropriate microorganism and the derived products modified further by chemical processes, for example, penicillins and cephalosporins (Adebayo, 2000). However, Penicillin, Bacillus, Micromonospora, Cephalosporuim and Streptomyces species are the 5 genera of microorganisms that produce almost all the antibiotics sold in Nigeria (Adebayo, 2000). For an antibiotic to be effective, it must exhibit selective toxicity and high therapeutic index. High therapeutic index implies a high ratio of maximum dose at which the antibiotic can be tolerated to a minimum dose required to cure an infection.

Such antibiotic does not eliminate the normal microbial flora of the host in order to avoid an upset of the natural balance as well as prevent the readily development of resistant forms of the pathogens.

Drug resistance is a large and growing problem in infections that account for most of Africa's disease burden, including malaria, tuberculosis (TB), HIV infection, respiratory and diarrhea diseases (Okeke et al., 2007; Okonko et al., 2009 a,b). The proportion of malaria infections resulting in death has increased in Africa, largely due to resistance and the cost of effective anti-malarial agents is higher than the health budgets of malaria-endemic countries can accommodate (Arrow et al., 2004; Okonko et al., 2009 a, b). Similarly, a recent out-break of extensively drug-resistant TB in rural South Africa illustrated that resistant organisms pose an enormous and costly threat to HIV-infected persons and their HIV-negative contacts (Singh et al., 2007; Okonko et al., 2009a, b).

Much of the current discourse on infectious disease and drug resistance as it affects sub-Saharan Africa is limited to the pressing problems associated with HIV, TB, malaria and other emerging- and re-emerging resistant organisms. Resistance, however, equally compromises the management of acute respiratory infections, sexually transmitted diseases and diseases spread by the fecal–oral route, such as typhoid fever, cholera, dysentery and other diarrhea diseases (Okeke et al., 2007). Moreover, young children are especially likely to acquire resistant enteric infections, from which they can experience less obvious, but long-term adverse effects (Okeke et al., 2007; Okonko et al., 2009a, b).

This study reports a survey of the level of efficacy and quality of some brands of antibiotics sold in Calabar, Nigeria. It determines the level of potency of the antibiotics based on their different brands or manufacturers and the diameters of zones of inhibition (in mm) of the antibiotics against the test pathogens.

MATERIALS AND METHODS

Study area

The study area was Calabar, Cross River State, South-south region of Nigeria. Calabar is one of the most ancient, colonial and cosmopolitan cities in Nigeria.

Media, chemicals and reagents

All the chemicals and reagents used were of analytical grade, obtained from Sigma chemical co. Ltd, England. Media used in this study included: Nutrient Agar (NA), Mac Conkey agar (MCA), Blood Agar, Mueller-Hinton Agar and Mannitol Salt Agar (MSA). All media were prepared according to the manufacturer's specification and sterilized at 121°C for 15 min at 15lb pressure.

Test organisms

Clinical isolates of the test microorganisms used in this study were obtained from Microbiology Section of the Sufat Medical Laboratories, Ishie, Calabar, the Microbiology laboratory of the

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University of Calabar Teaching Hospital, Calabar and the Department of Microbiology, University of Calabar, (UCTH), Calabar respectively.

Isolation and identification of isolates

Isolations were also made from the clinical samples such as blood (for blood culture), urine, pus swab, wound swab and sputum collected from the above laboratories. All the samples and the test organisms were replicate on different media and the plates were then incubated at 37°C for 24 - 48 h. Discrete colonies were sub-cultured into fresh agar plates aseptically to obtain pure cultures of the isolates. Colonies identifiable as discrete on the Mueller Hinton Agar were carefully examined macroscopically for cultural characteristics. All isolates were Gram stained to determine their gram reaction. Sugar utilization tests were carried out. Other tests carried out were Coagulase, Catalase, Citrate utilization, Urease activity, Oxidase, Methyl Red (MR)- Voges-Proscauer (VP), motility, Indole production, Kligler's Iron Agar (KIA) and Carbohydrate fermentation as described by Jolt et al. (1994). The isolates were identified by comparing their characteristics with those of known taxa, as described by Cheesbrough (2003, 2006) and Oyeleke and Manga (2008).

Antibiotic susceptibility testing

Assayed antibiotics

The test antibiotics used in this study were 2 brands each of ampicillin, cholramphenicol, erythromycin, septrin (cotrimoxazoles) and vancomycin (Tables 2 -7). These antibiotics were bought from reputable patents and pharmaceutical shops/stores located within Calabar metropolis. Standard antibiotic sensitivity disks were also purchased from scientific supply stores in the Calabar metropolis.

Preparation of the antibiotic sensitivity disks

Whatman No.1 filter papers were obtained and disks of about 5.25 mm were cut out from the filter papers. These were wrapped in foil paper and sterilized in the oven at 160°C for 1 h. The sensitivity disks were prepared to the National Committee for Clinical Laboratory Standards (NCCLS) subcommittee standards and guidelines (2002) to contain the concentrations 25 and 10 mcg equivalent to the standards. The standard commercial disks were 2 brands each of ampicillin (25 mcg), gentamicin (10 mcg), ciprofloxacin (10 mcg), erythromycin (10 mcg), septrin (25 mcg) and vancomycin (10 mcg). The different brands of the antibiotics were diluted to obtain the concentrations of the commercial standard disks using sterile distilled water. In order to get 25 mcg from 250 mg of the antibiotic 250 mg of the antibiotic was converted to 250000 mcg. This was dissolved in 10ml of sterile distilled water. This gave 25000 mcg and a 1: 10 dilution was prepared which gave 2500 mcg concentration. The 100 sensitivity disks already sterilized were put into the above solution. Each disk will absorb 25 mcg of the drug. In order to get 10 mcg from 500 mg of the antibiotic, 500 mg was converted to 500,000 mcg. This was also dissolved in 5 ml of sterile distilled water which gave 100,000mcg concentration and a 1: 100 dilution was prepared to give 1000 mcg. One hundred disks were each soaked with 1 ml containing 10 mcg of the antibiotic.

Activities of the antibiotics against the test organisms

The antibiotic susceptibility patterns of the isolates to common antibiotics sold in Calabar were evaluated using the agar-disk diffusion method on Sheep blood agar and Mueller-Hinton agar according to the National Committee for Clinical Laboratory Standards (NCCLS) and Manual of Antimicrobial Susceptibility Testing guidelines (Baur et al., 1996; Ebie et al., 2001; NCCLS, 2002; Cheesbrough, 2000, 2002, 2003; Coyle, 2005; Okonko et al., 2009 a, b). Mueller-Hinton agar (Difco Laboratories, Michigan, USA) is the NCCLS recommended medium for sensitivity analysis. It is an ideal medium for routine antimicrobial susceptibility tests since it shows good batch-to-batch uniformity and is low in tetracycline and sulfonamide inhibitors (Cheesbrough, 2006).

Inoculum preparation

The disk diffusion test was adopted (Finegold et al., 1978). Trypticase soy broth and nutrient broth were prepared. Five discrete colonies of the different identified isolates were inoculated into 5 ml of the broths and incubated at 35°C. A turbidometer was used to monitor the turbidity of the broth cultures. Immediately the turbidity exceeded the barium sulfate standard, the incubation was stopped. The broth culture was then diluted 1:10 with a freshly prepared nutrient broth to give a count of approximately 10^5 colonies per millimeter.

Inoculation of the test plates

Within 20 min of the growth reaching final turbidity, each of the isolates was uniformly and aseptically inoculated into a different Mueller-Hinton agar plates by spread plate method using sterile cotton wool. A sterile cotton wool was allowed to soak in the broth culture, squeezed by the side of the bottle before streaking over the sensitivity plates. The appropriate antibiotic multi-discs (either Gram positive or negative) were aseptically placed on the agar using sterile forceps. The plates were then incubated at 37°C for 24 h. The degree of susceptibility of the test isolate to each antibiotic was determined by measuring the diameter of the zones of inhibition (Baur et al., 1996; Ebie et al., 2001; NCCLS, 2002; Cheesbrough, 2002, 2003, 2006; Coyle, 2005; Okonko et al., 2009a, b).

Preparation of the control sensitivity disks

The method described by Pratt and Fekety (1986) was used. In this method the sterilized filter paper disks were impregnated with the various dilutions (l0 mcg and 25 mcg) of the test antibiotics in duplicates. With the aid of a sterile forceps, the impregnated disks were carefully placed on the inoculated plates and firmly pressed unto the agar with the sterile forceps to ensure complete contact with the agar. The disks were distributed evenly at 24 mm distance and in a manner as to be no closer than 15 mm from the edge of the Petri dish. The standard antibiotic disks were also placed on separate plates seeded with the test organisms. The plates were covered with the tops, inverted and incubated immediately at 37°C for 24 h. The standard positive commercial disks included gram positive, gram negative and broad spectrum disks while the negative control disks were impregnated with sterile distilled water. For S. *pyogenes,* Mueller-Hinton agar with 5% sheep blood was used. After incubation, the zones of clearance of organisms around the disks were also measured and recorded (Baur et al., 1996; Ebie et al., 2001; NCCLS, 2002; Cheesbrough, 2002, 2003, 2006; Coyle, 2005; Okonko et al., 2009a, b).

Statistical analyses

Data were analyzed using the general linear model procedure and

independent t-test to compare the mean diameters. Multiple comparisons were also carried using ANOVA to compare the responses. Indicator of statistical significance is P ≤ 0.05.

RESULTS

The microbiological characteristics of the different test organisms used in this study for survey of efficacy and quality of some of the antibiotics sold in Nigeria is presented in Table 1. The cultural, morphological and biochemical characteristics of the isolates indicated that they were *Enterobacter sp., Escherichia coli, Klebsiella pneumoniae*, *Proteus mirabilis, Pseudomonas aeruginosa*, *Proteus mirabilis*, *Salmonella sp., Staphylococcus aureus* and *Streptococcus pyogenes* (Table 1).

The results of the antibiotic susceptibility tests are presented in Tables 2 through 7. The efficacy of some of the antibiotics sold in Nigeria as they differ in brands or manufacturers were determined using different test organisms. The sensitivity testing of the test antibiotics were compared with that of the standardized commercial sensitivity disks analyzed using *Escherichia coli, Staphylococcus aureus, Pseudomonas aeruginosa*, *Klebsiella pneumoniae* and *Streptococcus pyogenes* as presented in Tables 2 to 7 respectively. These levels of efficacy were determined from the interpretation of the diameter of the zones of inhibition of these antibiotics on the microorganisms.

Table 2 shows the activities of the different brands of ampicillin tested against the test organisms and their zones of inhibition. The mean zones of inhibition for the test organisms ranged from 13.0 ± 1.00 - 40.0 ± 2.00, with 30.5 ± 1.50 for *E. coli*, 13.0 ± 1.00 for *K. pneumoniae*, 35.0 ± 1.00 for *P. aeruginosa*, 40.0 ± 2.00 for S. *aureus and* 36.3 ± 1.75 mm for S. *pyogenes.* It can be deduced that the 2 brands of ampicillin (Reichlin and Vitacillin) were comparable to the control standard in effect/efficacy on *E. coli*, *K. pneumoniae*, *S. aureus* and *S. pyogenes*. The sensitivity of these organisms were similar for both the test and control standard (P = 0.095, P = 0.095, P = 0.130 and P = 0.081). There were no significant differences (P > 0.05) between their mean zones of inhibition of the test antibiotics and standard controls for the drugs tested against the test organisms. However, for *P. aeruginosa*, the brands of ampicillin (with mean diameter of 35.0 ± 1.00 mm) were significantly (P = 0.027) less effective compared to the standard with mean diameter of 41.0 mm (Table 2).

Table 3 shows the levels of efficacy of chloramphenicol as determined using test organisms. The mean zones of inhibition for the test organisms ranged from 11.3 mm - 27.8 ± 2.25, with 27.8 ± 2.25 for *E. coli*, 26.5 ± 1.50 for *K. pneumoniae*, 11.3 ± 2.25 for *P. aeruginosa*, 12.3 ± 1.75 for *S. aureus and* 19.5 ± 2.50 mm for *S. pyogenes.* It can be deduced that the mean zones of inhibition of both brands of chloramphenicol (elisca and duban) on the test organisms were comparable to the standard controls in effect/efficacy (P > 0.05) except for *K. pneumoniae*. There were significant differences (P = 0.049) between the mean zones of inhibition of test antibiotics and standard controls of the drugs tested against the *K. pneumoniae* (Table 3).

Table 4 shows the activities of the different brands of erythromycin tested against the test organisms and their zones of inhibition. The mean zones of inhibition for the test organisms ranged from 04.5 ± 1.50, - 45.0 ± 1.00, with 04.5 ± 1.50, for *E. coli*, 45.0 ± 1.00 for *K. pneumoniae*, 22.3 ± 0.75 for *P. aeruginosa*, 41.8 ± 1.75 for *S. aureus and* 12.3 ± 2.25 mm for *S. pyogenes.* Mycin-500 showed higher zones of inhibition on the test organisms compared to stearate erythromycin. However, there were no significant differences (P > 0.05) between the mean zones of inhibition and standard controls of the drugs tested against the test organisms except for *K. pneumoniae* (P = 0.057) and *P. aeruginosa* (P = 0.038). Both brands of erythromycin (stearate and mycin-500) on the test organisms were comparable to the standard controls in effect/efficacy on test organisms except for *K. pneumoniae* and *P. aeruginosa* (Table 4).

Table 5 shows the activities of the different brands of septrin (contrimoxazole) against the test organisms and their zones of inhibition. The mean zones of inhibition for the test organisms ranged from 24.5 ± 1.50, -47.5 ± 1.50, with 37.5 ± 1.50 for *E. coli*, 47.5 ± 1.50 for *K. pneumoniae*, 27.3 ± 1.25 for *P. aeruginosa*, 38.8 ± 0.75 for S. *aureus and* 24.5 ± 1.50 mm for S. *pyogenes.* Wellcome septrin had higher zones of inhibitions on the test organisms compared to emtrim. However, there were no significant differences (P > 0.05) between the zones of inhibition of the 2 brands of septrin (wellcome and emtrim) and standard controls of the drugs tested against the test organisms (Table 5). Both brands of septrin (wellcome and emtrim) on the test organisms were comparable to the standard controls in effect/efficacy (Table 5).

Table 6 shows the activities of the different brands of vancomycin tested against the test organisms and their zones of inhibition. The mean zones of inhibitions for the test organisms ranged from 00.0 ± 0.00 – 33.5, with 09.0 ± 1.00 mm for *E. coli*, 00.0 ± 0.00 for *K. pneumoniae*, and 5.0 ± 0.00 mm for *P. aeruginosa*, 33.5 ± 1.50 for S. *aureus and* 15.8 ± 1.25 mm for *S. pyogenes.* It can be deduced that lincocin showed higher zones of inhibition compared to rincocin on all the test organisms except for *K. pneumoniae* and *P. aeruginosa*. Both brands showed zero zones of inhibition against *K. pneumoniae* (Table 6). However, there were no significant differences (P > 0.05) between their zones of inhibition and standard controls of the drugs tested against the test organisms. Both brands of vancomycin (lincocin and rincocin) on the test organisms were comparable to the standard controls in effect/efficacy (Table 6).

Table 7 shows the overall multiple responses of the organisms of the test organisms to different antibiotics.

Table 1. Morphological and biochemical characteristics of bacteria isolates used in this study.

	Isolates							
Parameters	I	II	III	IV	V	VI	VII	VIII
Grams reaction	-	-	+	-	-	-	+	-
Cellular morphology	Rods	Rods	Cocci	Small rods	Straight rods	Rods	Cocci in chains	Small rods
Growth on Blood agar (colony)	Large greyish-white mucoid	Large greyish-white partially mucoid	Creamy white	Greenish	Large, flat spreading and circular mucoid	Greyish-white	Creamy/colourless, mucoid in chains with zones of complete haemolysis	Swarming with fishy smell
Growth on MacConkey agar	Pink Mucoid	Mucoid	N/A	Pale	Smooth Red/Pink	Pale	Pink	
Growth on Mannitol Salt agar	N/A	N/A	Bright yellow	N/A	N/A	N/A	N/A	N/A
Motility	-	+	-	+	-	+	-	+
Catalase test	+	-	+	+	+	+	-	+
Coagulase test	N/A	N/A	+	N/A	N/A	N/A	-	N/A
Citrate test	+	-	+	+	+	+	+	+
Oxidase test	-	-	-	+	-	-	-	-
Indole test	-	+	-	-	+	-	-	-
Urease activity	+	+	+	-	-	-	-	+
Methyl Red	+	+	+	-	-	+	+	+
Voges Proskauer	+	-	-	+	-	-	-	-
Bacitracine	N/A	N/A	N/A	N/A	N/A	N/A	+	N/A
Growth on KIA Medium:								
Slope	Yellow	Yellow	Yellow	Yellow	Yellow	Yellow	Yellow	Red-pink
Butt	Yellow	Yellow	Yellow	A	Yellow	Yellow	Yellow	Yellow
Hydrogen Sulphide (H_2S)	-	-	+	-	-	+	+	+
Gas production	-/G	-/G	-/G	-	-/G	-/G	-/G	-/G
Sugar fermentation test:								
Glucose	A/G	A/G	A/G	-/-	A/G	A/G	A/G	A/G
Lactose	A/-	A/-	A/-	A/G	A/-	A/G	-	-/-
Sucrose	A/-	A/-	A/-	A/G	A/-	A/G	A/G	A/-
Mannitol	A/-	A/-	A/-	A/G	A/-	A	-/-	-/-
Maltose	A/-	A/-	A/-	-/-	A/-	A/G	-/-	-/-
Most probable organism	*Klebsiella pneumoniae*	*Enterobacter sp*	*Staphylococc us aureus*	*Pseudomonas aeruginosa*	*E. coli*	*Salmonella sp*	*Streptococcus pyogenes*	*Proteus mirabilis*

Keys: N/A = Not applicable, - = No growth, + = Growth, A/G = Acid production and gas production, A/- = Acid production only and no gas production, -/G = Gas production only, -/- = No acid and gas production, Yellow = Acidic reaction, Red-pink = Alkaline reaction.

Table 2. Activities of the different brands of ampicillin tested against the test organisms and their zones of inhibition.

			Zones of inhibition (mm)			Test statistics	
Code	**Test organism**	**Brand**	**Test antibiotics**	**Standard controls**	**Mean + SE (n=2)**	**t- value**	**P value**
I	*E. coli*	Reichlin (25 mcg)	32.0	35.0			
		Vitacillin (25 mcg)	29.0	35.0	30.5 ± 1.50	-3.00	0.095
II	*K. pneumoniae*	Reichlin (25 mcg)	14.0	16.0			
		Vitacillin (25 mcg)	12.0	16.0	13.0 ± 1.00	-3.00	0.095
III	*P. aeruginosa*	Reichlin (25 mcg)	36.0	41.0			
		Vitacillin (25 mcg)	34.0	41.0	35.0 ± 1.00	-6.00	0.027*
IV	*S. aureus*	Reichlin (25 mcg)	42.0	45.0			
		Vitacillin (25 mcg)	38.0	45.0	40.0 ± 2.00	-2.50	0.130
V	*S. pyogenes*	Reichlin (25 mcg)	38.0	42.0			
		Vitacillin (25 mcg)	34.5	42.0	36.3 ± 1.75	-3.29	0.081

Key: SE = Standard Error of Mean, * = Significant.

Table 3. Activities of the different brands of chloramphenicol tested against the test organisms and their zones of inhibition.

			Zones of inhibition (mm)			Test statistics	
Code	**Test organism**	**Brand**	**Test antibiotics**	**Standard controls**	**Mean + SE (n=2)**	**t- value**	**P value**
I	*E. coli*	Elisca (25 mcg)	30.0	36.0			
		Duban (25 mcg)	25.5	36.0	27.8 ± 2.25	-3.67	0.067
II	*K. pneumoniae*	Elisca (25 mcg)	28.0	33.0			
		Duban (25 mcg)	25.0	33.0	26.5 ± 1.50	-4.33	0.049*
III	*P. aeruginosa*	Elisca (25 mcg)	13.5	17.0			
		Duban (25 mcg)	09.0	17.0	11.3 ± 2.25	-2.56	0.125
IV	*S. aureus*	Elisca (25 mcg)	14.0	18.0			
		Duban (25 mcg)	10.5	18.0	12.3 ± 1.75	-3.29	0.081
V	*S. pyogenes*	Elisca (25 mcg)	22.0	27.0			
		Duban (25 mcg)	17.0	27.0	19.5 ± 2.50	-3.00	0.095

Key: SE = Standard Error of Mean, * = Significant.

Table 4. Activities of the different brands of erythromycin tested against the test organisms and their zones of inhibition.

			Zones of inhibition (mm)			Test statistics	
Code	**Test organism**	**Brand**	**Test antibiotics**	**Standard controls**	**Mean + SE (n=2)**	**t- value**	**P value**
I	*E. coli*	Stearate (10 mcg)	03.0	08.5			
		Mycin-500 (10 mcg)	06.0	08.5	04.5 ± 1.50	-2.67	0.117
II	*K. pneumoniae*	Stearate (10 mcg)	44.0	49.0			
		Mycin-500 (10 mcg)	46.0	49.0	45.0 ± 1.00	-4.00	0.057*
III	*P. aeruginosa*	Stearate (10 mcg)	21.5	26.0			
		Mycin-500 (10 mcg)	23.0	26.0	22.3 ± 0.75	-5.00	0.038*
IV	*S. aureus*	Stearate (10 mcg)	40.0	48.0			
		Mycin-500 (10 mcg)	43.5	48.0	41.8 ± 1.75	-3.57	0.070
V	*S. pyogenes*	Stearate (10 mcg)	10.0	15.0			
		Mycin-500 (10 mcg)	14.5	15.0	12.3 ± 2.25	-1.22	0.346

Key: SE = Standard Error of Mean, * = Significant.

The overall mean zones of inhibition for the test organisms ranged from 20.2 – 33.3 mm, with 21.9 mm for *E. coli*, 26.4 mm for *K. pneumoniae*, 20.2 mm for *P. aeruginosa*, 33.3 mm for S. *aureus* and 21.7 mm for S. *pyogenes.* The multiple responses (LSD) of the test organisms to different antibiotics showed that there were

Table 5. Activities of the different brands of septrin (contrimoxazole) tested against the test organisms and their zones of inhibition.

			Zones of inhibition (mm)			Test statistics	
Code	**Test organism**	**Brand**	**Test antibiotics**	**Standard controls**	**Mean + SE (n=2)**	**t- value**	**P value**
I	*E. coli*	Wellcome (25 mcg)	39.0	40.0			
		Emtrim (25 mcg)	36.0	40.0	37.5 ± 1.50	-2.67	0.238
II	*K. pneumoniae*	Wellcome (25 mcg)	49.0	50.0			
		Emtrim (25 mcg)	46.0	50.0	47.5 ± 1.50	-4.00	0.238
III	*P. aeruginosa*	Wellcome (25 mcg)	28.5	30.0			
		Emtrim (25 mcg)	26.0	30.0	27.3 ± 1.25	-5.00	0.159
IV	*S. aureus*	Wellcome (25 mcg)	39.5	40.0			
		Emtrim (25 mcg)	38.0	40.0	38.8 ± 0.75	-3.57	0.238
V	*S. pyogenes*	Wellcome (25 mcg)	22.0	22.0			
		Emtrim (25 mcg)	19.0	22.0	24.5 ± 1.50	-1.22	0.423

Key: SE = Standard Error of Mean.

Table 6. Activities of the different brands of vancomycin tested against the test organisms and their zones of inhibition.

			Zones of inhibition (mm)			Test statistics	
Code	**Test organism**	**Brand**	**Test antibiotics**	**Standard controls**	**Mean + SE (n=2)**	**t- value**	**P value**
I	*E. coli*	Lincocin (10 mcg)	10.0	12.0			
		Rincocin (10 mcg)	08.0	12.0	09.0 ± 1.00	-3.00	0.095
II	*K. pneumoniae*	Lincocin (10 mcg)	00.0	02.0			
		Rincocin (10 mcg)	00.0	02.0	00.0 ± 0.00[a]	a	a
III	*P. aeruginosa*	Lincocin (10 mcg)	05.0	07.0			
		Rincocin (10 mcg)	05.0	07.0	05.0 ± 0.00[a]	a	a
IV	*S. aureus*	Lincocin (10 mcg)	35.0	38.0			
		Rincocin (10 mcg)	32.0	38.0	33.5 ± 1.50	-3.40	0.095
V	*S. pyogenes*	Lincocin (10 mcg)	17.0	20.0			
		Rincocin (10 mcg)	14.5	20.0	15.8 ± 1.25	-3.40	0.077

Key: SE = Standard Error of Mean, [a] = t cannot be computed because the standard deviation of both group is zero.

significant differences between ampicillin and erythromycin; ampicillin and septrin; ampicillin and vancomycin. There was no significant difference (P = 0.279) between the zones of inhibition observed for ampicillin (30.5 mm) and chloramphenicol (27.8 mm) and no significant difference (P = 0.104) between vancomycin (9 mm) and erythromycin (4.5 mm) against *E. coli*. There was no significant difference (P = 0.186) between the mean zones of inhibition showed by septrin (47.5 mm) and erythromycin (45 mm) against *K. pneumoniae*. *P. aeruginosa* responses in terms of their zones of inhibitions were highest for ampicillin with 35 mm and least for vancomycin (5 mm) respectively. It was significantly (P = 0.001) more sensitive to ampicillin (mean 35 mm) than to vancomycin (mean 0.0 mm); it was also significantly (P = 0.001) more sensitive to septrin (mean 27.3 mm) than to chloramphenicol (mean 13 mm); in the same vein the *K. pneumoniae* was significantly (P = 0.001) more sensitive to chloramphenicol (mean 26.5 mm) than to ampicillin (mean 13 mm). In the case of *S. aureus*, *S. aureus* was significantly (P = 0.001) more sensitive to erythromycin (mean 41.8 mm), ampicillin (mean 40 mm) and septrin (38.8 mm) than to vancomycin (mean 33.5 mm) and chloraphenicol (mean 12.3 mm) as shown in Table 7. For *S. pyogenes*, *S. pyogenes* was significantly (P = 0.002; P = 0.000, P = 0.001, P = 0.002) more sensitive to ampicillin compared to chloramphenicol, erythromycin, septrin and vancomycin respectively (Table 7).

DISCUSSION

In testing the efficacy of 10 brands of 5 different antibiotics sold in Calabar, Nigeria, a total of 5 different bacteria were used. The identified test organisms included; *E. coli, K. pneumoniae*, *P. aeruginosa*, *S. aureus* and *S. pyogenes.* This susceptibility test measures the ability of the different antibiotics to inhibit bacterial growth. It involves the *in vitro* testing using disk paper diffusion technique and measuring their zones of inhibition. From the results, the overall mean zones of

Table 7. Multiple responses of the test organisms to different test antibiotics.

LSD and ANOVA					
Test organisms	**(I) Brandgrp**	**(J) Brandgrp**	**Zones of Inhibition (mm)**	**Mean Difference (I-J) + SE**	**P value**
E. coli	Ampicillin	Chloramphenicol	30.5 ± 2.26	2.75 ± 2.26	0.279
		Erythromycin		26.00 ± 2.26(*)	0.000
		Septrin		-7.00 ± 2.26(*)	0.027
		Vancomycin		21.50 ± 2.26(*)	0.000
	Chloramphenicol	Ampicillin	27.8 ± 2.26	-2.75 ± 2.26	0.279
		Erythromycin		23.25 ± 2.26(*)	0.000
		Septrin		-9.75 ± 2.26(*)	0.008
		Vancomycin		18.75 ± 2.26(*)	0.000
	Erythromycin	Ampicillin	04.5 ± 2.26	-26.00 ± 2.26(*)	0.000
		Chloramphenicol		-23.25 ± 2.26(*)	0.000
		Septrin		-33.00 ± 2.26(*)	0.000
		Vancomycin		-4.50 ± 2.26	0.104
	Septrin	Ampicillin	37.5 ± 2.26	7.00 ± 2.26(*)	0.027
		Chloramphenicol		9.75 ± 2.26(*)	0.008
		Erythromycin		33.00 ± 2.26(*)	0.000
		Vancomycin		28.50 ± 2.26(*)	0.000
	Vancomycin	Ampicillin	09.0 ± 2.26	-21.50 ± 2.26(*)	0.000
		Chloramphenicol		-18.75 ± 2.26(*)	0.000
		Erythromycin		4.50 ± 2.26	0.104
		Septrin		-28.50 ± 2.26(*)	0.000
K. pneumoniae	Ampicillin	Chloramphenicol	13.0 ± 1.61	-13.50 ± 1.61(*)	0.000
		Erythromycin		-32.00 ± 1.61(*)	0.000
		Septrin		-34.50 ± 1.61(*)	0.000
		Vancomycin		13.00 ± 1.61(*)	0.000
	Chloramphenicol	Ampicillin	26.5 ± 1.61	13.50 ± 1.61(*)	0.000
		Erythromycin		-18.50 ± 1.61(*)	0.000
		Septrin		-21.00 ± 1.61(*)	0.000
		Vancomycin		26.50 ± 1.61(*)	0.000
	Erythromycin	Ampicillin	45.0 ± 1.61	32.00 ± 1.61(*)	0.000
		Chloramphenicol		18.50 ± 1.61(*)	0.000
		Septrin		-2.50 ± 1.61	0.182
		Vancomycin		45.00 ± 1.61(*)	0.000
	Septrin	Ampicillin	47.5 ± 1.61	34.50 ± 1.61(*)	0.000
		Chloramphenicol		21.00 ± 1.61(*)	0.000
		Erythromycin		2.50 ± 1.61	0.182
		Vancomycin		47.50 ± 1.61(*)	0.000
	Vancomycin	Ampicillin	0.0 ± 1.61	-13.00 ± 1.61(*)	0.000
		Chloramphenicol		-26.50 ± 1.61(*)	0.000
		Erythromycin		-45.00 ± 1.61(*)	0.000
		Septrin		-47.50 ± 1.61(*)	0.000
P. aeruginosa	Ampicillin	Chloramphenicol	35.0 ± 1.81	23.75 ± 1.81(*)	0.000
		Erythromycin		12.75 ± 1.81(*)	0.001
		Septrin		7.75 ± 1.81(*)	0.008
		Vancomycin		30.00 ± 1.81(*)	0.000
	Chloramphenicol	Ampicillin	11.3 ± 1.81	-23.75 ± 1.81(*)	0.000
		Erythromycin		-11.00 ± 1.81(*)	0.002
		Septrin		-16.00 ± 1.81(*)	0.000
		Vancomycin		6.25 ± 1.81(*)	0.018
	Erythromycin	Ampicillin	22.3 ± 1.81	-12.75 ± 1.81(*)	0.001
		Chloramphenicol		11.00 ± 1.81(*)	0.002

Table 7. Contd.

		Septrin		-5.00 ± 1.81(*)	0.040
		Vancomycin		17.25 ± 1.81(*)	0.000
	Septrin	Ampicillin	27.3 ± 1.81	-7.75 ± 1.81(*)	0.008
		Chloramphenicol		16.00 ± 1.81(*)	0.000
		Erythromycin		5.00 ± 1.81(*)	0.040
		Vancomycin		22.25 ± 1.81(*)	0.000
	Vancomycin	Ampicillin	05.0 ± 1.81	-30.00 ± 1.81(*)	0.000
		Chloramphenicol		-6.25 ± 1.81(*)	0.018
		Erythromycin		-17.25 ± 1.81(*)	0.000
		Septrin		-22.25 ± 1.81(*)	0.000
S. aureus	Ampicillin	Chloramphenicol	40.0 ± 2.27	27.75 ± 2.27(*)	0.000
		Erythromycin		-1.75 ± 2.27	0.476
		Septrin		1.25 ± 2.27	0.606
		Vancomycin		6.50 ± 2.27(*)	0.036
	Chloramphenicol	Ampicillin	12.3 ± 2.27	-27.75 ± 2.27(*)	0.000
		Erythromycin		-29.50 ± 2.27(*)	0.000
		Septrin		-26.50 ± 2.27(*)	0.000
		Vancomycin		-21.25 ± 2.27(*)	0.000
	Erythromycin	Ampicillin	41.8 ± 2.27	1.75 ± 2.27	0.476
		Chloramphenicol		29.50 ± 2.27(*)	0.000
		Septrin		3.00 ± 2.27	0.244
		Vancomycin		8.25 ± 2.27(*)	0.015
	Septrin	Ampicillin	38.8 ± 2.27	-1.25 ± 2.27	0.606
		Chloramphenicol		26.50 ± 2.27(*)	0.000
		Erythromycin		-3.00 ± 2.27	0.244
		Vancomycin		5.25 ± 2.27	0.069
	Vancomycin	Ampicillin	33.3±2.27	-6.50 ± 2.27(*)	0.036
		Chloramphenicol		21.25 ± 2.27(*)	0.000
		Erythromycin		-8.25 ± 2.27(*)	0.015
		Septrin		-5.25 ± 2.27	0.069
S. pyogenes	Ampicillin	Chloramphenicol	36.3 ± 2.69	16.75 ± 2.69(*)	0.002
		Erythromycin		24.00 ± 2.69(*)	0.000
		Septrin		15.75 ± 2.69(*)	0.002
		Vancomycin		20.50 ± 2.69(*)	0.001
	Chloramphenicol	Ampicillin	19.5±2.69	-16.75 ± 2.69(*)	0.002
		Erythromycin		7.25 ± 2.69(*)	0.043
		Septrin		-1.00 ± 2.69	0.726
		Vancomycin		3.75 ± 2.69	0.223
	Erythromycin	Ampicillin	12.3 ± 2.69	-24.00 ± 2.69(*)	0.000
		Chloramphenicol		-7.25 ± 2.69(*)	0.043
		Septrin		-8.25 ± 2.69(*)	0.028
		Vancomycin		-3.50 ± 2.69	0.251
	Septrin	Ampicillin	24.5 ± 2.69	-15.75 ± 2.69(*)	0.002
		Chloramphenicol		1.00 ± 2.69	0.726
		Erythromycin		8.25 ± 2.69(*)	0.028
		Vancomycin		4.75 ± 2.69	0.139
	Vancomycin	Ampicillin	15.8 ± 2.69	-20.50 ± 2.69(*)	0.001
		Chloramphenicol		-3.75 ± 2.69	0.223
		Erythromycin		3.50 ± 2.69	0.251
		Septrin		-4.75 ± 2.69	0.139

Key: SE = Standard mean error, * = The mean difference is significant at the 0.05 level.

inhibition for the test organisms ranged from 20.2 – 33.3 mm, with 21.9 mm for *E. coli*, 26.4 mm for *K. pneumoniae*, 20.2 mm for *P. aeruginosa*, 33.3 mm for S. *aureus and* 21.7 mm for S. *pyogenes.* The potency or activity per milligram of a chemotherapeutic agent is usually expressed on the basis of the lowest concentration of minimal inhibitory concentrations (MICs) or higher zones of inhibition (Nnela and Cox, 1988). Bacterial resistance to beta-lactam antibiotics is primarily due to the production of beta-lactam ring of the antibiotics rendering them inactive (Akpan, 1992).

In this present study, the potency of the standard drugs when compared to test antibiotics and the mean potency of the antibiotic on the organisms determined showed differences in efficacy and quality of the various brands of antibiotics sold in Nigeria. From our study, there were no significant differences (P > 0.05) between the zones of inhibition and control standards for the drugs tested against the test organisms. However, for *P. aeruginosa*, the two brands of ampicillin and erythromycin were significantly (P = 0.027; P = 0.038) less effective compared to the control standards while for *K. pneumoniae*, the two brands of chlorampenicol and erythromycin were significantly (P = 0.049; P = 0.057) less effective compared to the control standards. This showed that some of the brands of ampicillin, chloramphenicol and erythromycin sold in Calabar, Nigeria may be fake or adulterated (Adejoh, 2000), which is a reflection of what goes on in many developing countries, in particular, in sub-Saharan Africa is considerable and within those countries, economically disadvantaged persons are most likely to contract communicable diseases and least likely to access appropriate treatment (Schellenberg et al., 2003; Bates et al., 2004; Okeke et al., 2007; Okonko et al., 2009 a, b).

From our study, the multiple responses analysis of the test organisms to different antibiotics showed that there were significant differences between ampicillin and erythromycin; ampicillin and septrin; ampicillin and vancomycin. *E. coli* and *K. pnemuoniae* responses in terms of their zones of inhibitions were highest for septrin with 37.5 and 47.5 mm and least for erythromycin (4.5 mm) and vancomycin (0.0 mm) respectively. *P. aeruginosa* response was highest for ampicillin (35 mm) and least for vancomycin (5 mm). *S. aureus* response was highest for erythromycin (41.8 mm) and least for chloramphenicol (12.3 mm) while *S. pyogenes* response was highest for ampicillin (36.3 mm) and least for erythromycin (12.3 mm). E. coli was significantly (P = 0.001) more sensitive to ampicillin (mean 30.5 mm) than to erythromycin (mean 4.5 mm); it was also significantly (P = 0.027) more sensitive to septrin (mean 37.5 mm) than to ampicillin (mean 30.5 mm); in the same vein the *E. coli* was significantly (P = 0.001) more sensitive to ampicillin (mean 30.5 mm) than to vancomycin (mean 9.0 mm). However, there was no significant difference (P = 0.279) between the zones of inhibition observed for ampicillin (30.5 mm) and chloramphenicol (27.8 mm) and no significant difference (P = 0.104) between vancomycin (9 mm) and erythromycin (4.5 mm) against *E. coli*.

K. pneumoniae was significantly (P = 0.001) more sensitive to septrin (mean 47.5 mm) than to vancomycin (mean 0.0 mm); it was also significantly (P = 0.001) more sensitive to erythromycin (mean 45 mm) than to ampicillin (mean 13 mm); in the same vein the *K. pneumoniae* was significantly (P = 0.001) more sensitive to chloramphenicol (mean 26.5 mm) than to ampicillin (mean 13 mm). There was no significant difference (P = 0.186) between the mean zones of inhibitions showed by septrin (47.5 mm) and erythromycin (45 mm) against *K. pneumoniae*. There were significant differences (P < 0.05) between the mean zones of inhibitions showed by all test antibiotics against *P. aeruginosa*. *P. aeruginosa* responses in terms of their zones of inhibitions were highest for ampicillin with 35 mm and least for vancomycin (5 mm) respectively. It was significantly (P = 0.001) more sensitive to ampicillin (mean 35 mm) than to vancomycin (mean 0.0 mm); it was also significantly (P = 0.001) more sensitive to septrin (mean 27.3 mm) than to chloramphenicol (mean 13 mm); in the same vein the *K. pneumoniae* was significantly (P = 0.001) more sensitive to chloramphenicol (mean 26.5 mm) than to ampicillin (mean 13 mm).

In the case of *S. aureus*, it was significantly (P = 0.001) more sensitive to erythromycin (mean 41.8 mm), ampicillin (mean 40 mm) and septrin (38.8 mm) than to vancomycin (mean 33.5 mm) and chloraphenicol (mean 12.3 mm) as shown in (Table 7). Chloramphenicol showed significantly (P = 0.001) lower zones of inhibittions compared to other antibiotics against *S. aureus*. Also, vancomycin showed significantly (P = 0.001) lower zones of inhibitions compared to other antibiotics against *S. aureus* except for septrin (P = 0.069). However, there were no significant differences (P > 0.05) between the mean zones of inhibitions showed by ampicillin compared to erythromycin (P = 0.476) and septrin (P = 0.606) respectively; between erythromycin and septrin (P = 0.244) and between septrin and vancomycin (0.069). For *S. pyogenes*, *S. pyogenes* was significantly (P = 0.002; P = 0.000, P = 0.001, P = 0.002) more sensitive to ampicillin compared to chloramphenicol, erythromycin, septrin and vancomycin respectively. Chloramphenicol showed no significant zones of inhibitions (P = 0.726, P = 0.223) against *S. pyogenes* compared to septrin and vancomycin respectively. Also, erythromycin showed no significant zone of inhibition (P = 0.251) against *S. pyogenes* compared to vancomycin. Septrin showed no significant zones of inhibitions (P = 0.0726, P = 0.139) against *S. pyogenes* compared to chloramphenicol and vancomycin. However, there were no significant differences (P = 0.223, P = 0.251, P= 0.139) between the mean zones of inhibitions showed by vancomycin against *S. pyogenes* compared to other antibiotics except for ampicillin (P = 0.001).

This result is similar to that reported by Reish et al. (1993) and Aiyegoro et al. (2007) who also reported that *Klebsiella* spp. showed a resistance of 66.7% against cotrimoxazole. This is also similar to the study on the outbreak of multi-resistance *Klebsiella* in a neonatal intensive care unit in a hospital in Israel in which the *Klebsiella* isolates were resistant to chloramphenicol, gentamycin, cefuroxin but sensitive to quinolenes (Aiyegoro et al., 2007). Resistance to ampicillin, erythromycin by *P. aeruginosa* and K*. pneumoniae* and to resistance to chloramphenicol by *K. pneumoniae* has been previously reported (Olowu and Oyetunji, 2003; Aiyegoro et al., 2007; Okonko et al., 2009 b). Okonko et al. (2009b) reported 100% resistance to ampicillin, chloramphenicol and cotrimoxazole by *K. pneumoniae*. Zero resistance to septrin (cotrimoxazole) reported in this study is contrary to what was previously reported. Goldraichi and Manfrori (2002) reported 6.7% sensitivity by *E. coli* and Okonko et al. (2009 b) reported that all the gram negative isolates were resistant to cotrimoxazole. Cotrimoxazole resistance remained stable, approximately 30% in a study by Oteo et al. (2005) and similar to the 27% reported by Alos et al. (1993) in Spain in 1993. Reish et al. (1993) and Aiyegoro et al. (2007) reported that 66.7% resistance to cotrimoxazole and that resistance of *E. coli* to cotrimoxazole was 57.9%. Christiaen et al. (1998) reported a resistance of 17% to cotrimoxazole and a similar result was reported for resistance to quinolones.

However, one of the cankerworms plaguing our country, Nigeria, in recent times is the menace of the sales of fake adulterated and substandard drugs which has eaten deep into fabric of our society like a bad ulcer (Popoola, 2001). Our study showed that the efficacy of antibiotics sold in Nigeria was poor with reference to the active ingredients used. Resistance due to over use and adulteration of the antibiotics has also been reported. The zones of inhibition shown by these brands of antibiotics against the test organisms indicate their potencies (Cheesbrough, 2000, 2002, 2003, 2006; Pelczar, 1998). The potencies of course have to do with the active ingredients contained in each of the antibiotics since the test were compared to the standards. Other factors that could have affected their potencies such as; storage procedure, temperature, adulteration, humidity, expiring dates, pathophysiological state of the patient, natural history of the infection, presence of R-factor, age of patient, etc. Also, the widespread counterfeiting of these antibiotics, excessive decomposition of active ingredient as a result of exposure to high temperature and humidity and poor quality assurance during the manufacturer are not exceptions, were not investigated; however, the differences in efficacy among brands of the antibiotics constitute a grave danger to health. And the implication is that many bacterial and parasitic diseases that could, until recently, be treated with inexpensive antimicrobial agents, has recently been made more expensive and less successful by the emergence and spread of resistant organisms (Okeke et al., 2007; Donbraye-Emmanuel et al., 2009; Okonko et al., 2009 a, b). However, this drug resistance has now become a large and growing problem in infections that account for most of Africa's disease burden, including malaria, tuberculosis (TB), HIV infection, respiratory and diarrhea diseases (Okeke et al., 2007).

In this study, the observed efficacies of some of the antibiotics sold in Nigeria differ in their efficacies or potencies depending to their brands or manufacturers. Some showed a higher efficacy and some a lower efficacy when compared to the standard controls used. These differences in efficacies among brands of some antibiotics constitute a potential danger to health. Since proper quality control of pharmaceutical products is a sure way of producing drugs that meet up with standard (Florey, 1998), there is therefore an urgent need for all pharmaceutical products manufacturers to make sure that the recommended active ingredients and other ingredients' expiration dates are ascertained before the drugs are distributed for public consumption. However, to enhance the consent efficacy of some antibiotics sold in Nigeria, adequate measures must be gingered to monitor the storage, distribution and manufacture of these antibiotics. The ban on the sale and use of "fake" and expired drugs should be enforced by the authorities concerned. Also, cogent awareness campaign to intimate the citizenry of the need for the proper prescription of antibiotic drugs before use should also be intensified to guide against the use of low or higher concentrations of antibiotics compared to the dose needed in the blood stream. These prescriptions should be given by their official brand names as provided in the United States Pharmacopedia (USP).

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