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R-Plasmid Transfer Frequencies from Faecal Isolates of *Escherichia coli* of Cancer and Noncancer Patients to *Escherichia coli* K12 Strain

Mohemid Maddallah Al-Jebouri[1*], Ibraheem Abdul-kareem Al-Ani[2]
[1]Department of Medical Laboratory Technology, Health and Medical College of Technology, Al-Qalam University, Kirkuk, Iraq
[2]Department of Microbiology, College of Medicine, University of Tikrit, Tikrit, Iraq

Abstract: ***Background*:** Diarrheagenic *E. coli* is currently categorized into a limited number of groups, primarily based on the presence of specific virulence genes, and is classified under a single pathogenic variant (pathovar). The significance of Horizontal Gene Transfer (HGT) in the evolution of the genome and pathogenicity of STEC is far more comprehensive than we previously realized. ***Materials and Methods***: 400 patients were included and they were 200 patients with cancer diseases and 200 patients complaining of other illnesses. Specimens were taken from faeces and cultured on MacConkey agar. Isolated bacteria were identified using APIE System. Antibiotic sensitivity testing and resistance transfer from local *E.coli* to *E.coli* K12 was carried out as described elsewhere. ***Results*:** The rate of transfer of resistance traits from pathogenic E.coli to *E.coli* K12 was ranged between 10^{-6} to 10^{-3}. The frequency of resistance transfer of carbenicillin, ampicillin and sulfonamide was 80.5, 72.1 and 62.5% respectively. The comparative analysis of antibiotic resistance patterns before and after R-factor transfer demonstrated substantial horizontal dissemination of multidrug resistance among pathogenic *Escherichia coli* isolates. The mean resistance-retention efficiency after conjugative transfer was 59.4%, confirming that a large proportion of resistance determinants were plasmid mediated and transmissible to recipient *E. coli* K12 strains. ***Conclusions*:** The rate of transfer of resistance traits from pathogenic E.coli to *E.coli* K12 was ranged between 10^{-6} to 10^{-3}. The log–linear regression analysis demonstrated a statistically significant positive relationship between donor and recipient resistance-gene transfer frequencies among pathogenic *Escherichia coli* serotypes and *E. coli* K12 recipients ($p = 0.0006$). The frequency of resistance transfer of carbenicillin, ampicillin and sulfonamide was 80.5, 72.1 and 62.5% respectively. This rate of transfer was almost high than media transfer rate. There was a lower rate of transfer for nalidixic acid, colistin and nitrofurantoin and the rate was 20, 3.3 and 0% respectively. The statistical analysis demonstrated a significant positive association between the number of antibiotic-resistant *Escherichia coli* isolates and the frequency of resistance transfer to recipient *E. coli* K12 strains (Spearman $r = 0.788$, $p = 0.0014$). Overall, the findings provide strong evidence that R-factor-mediated conjugation plays a major role in the dissemination of antimicrobial resistance among pathogenic *Escherichia coli* populations (Figure 3). The most suitable mathematical descriptor considered was resistance-retention efficiency modeling: Retention Efficiency (%) =Before Transfer/After Transfer×100.

Keywords: Cancer Patients, Faeces, *E.Coli*, Antibiotics, Resistance, Transfer.

Research Paper

***Corresponding Author:**
Mohemid Maddallah Al-Jebouri
Department of Medical Laboratory Technology, Health and Medical College of Technology, Al-Qalam University, Kirkuk, Iraq

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INTRODUCTION

The basis for concem is that multiple-drug resistant coliforms have been isolated from sewage, its P receiving waters, and sludge-dumping areas, where they comprised between 1 and 50% of the coliform population [1-4]. In addition, it has been shown that about 60% of the multiple-drug-resistant coliform isolates transfer their plasmids to laboratory strains of *E. coli*, *Salmonella*, or *Shigella* [5-8]. However, in most of these studies, the actual transfer frequencies were not determined, nor were the abilities of fecal and environmental strains of *E. coli* and enteric pathogens to receive the plasmids considered. These factors are important in the assessment of risk. The present investigation was initiated to reexamine the question of water-bome dissemination of R plasmids with emphasis on these two considerations. It was found that the probability that an R plasmid from the ingested R+ *E. coli* will be transferred to a resident E. coli or a subsequently ingested enteric pathogen could be increased if the R+ E. coli colonized the bowel of the individual and thereby increased its numbers. Plasmid transfer between *E. coli* strains can occur in the gut [9], even in the absence of antibiotics [10-12], although this is a rare phenomenon [13]. Usually, though, more than 106 bacteria must be ingested in order for colonization to occur [14], and even when 1010 viable organisms are ingested, colonization by R-plasmid- bearing strains is poor [15]. Furthermore, if R-plasmid transfer between an ingested R+ coliform and a resident E. coli strain does occur, there is a question as to how long the R+ transconjugant will remain in the bowel [16-19]. This period of time will influence the probability that the R+ bacteria will transfer resistance to a subsequently ingested enteric pathogen. Diarrheagenic *E. coli* is currently categorized into a limited number of groups, primarily based on the presence of specific virulence genes, and is classified under a single pathogenic variant (pathovar). EPEC strains that possess the Locus of Enterocyte Effacement (LEE) region and Bundle-Forming Pilus (BFP) are referred to as typical EPEC (tEPEC), while those that lack the BFP are designated as atypical EPEC (aEPEC) [20-23]. In 28 % of the resistant strains R factors transmissible to *E. coli* K12 W 3132 could be demonstrated. This does not necessarily mean that R factors were not present in 72 %. It is well known that R factors may be segregated and accordingly loose their transmissibility *(Anderson* 1968) the use of more than one recipient strain might have increased the possibility to demonstr-ate R factors. *E. coli V\T* 3132, however, was shown to be a competent recipient of some R factors, since the frequency of transfer in some experiments was higher than 10-2•. In the meantime, ETEC is identified at the molecular level by the presence of heat-labile (LT) or heat-stable (ST) enterotoxins, along with a variety of accessory virulence factors, including the autotransporter [24]. These canonical destructiveness variables are frequently encoded on plasmids or other mobile components within the segregates from each of these pathovars [25-28].

MATERIALS AND METHODS

Patients

This study was carried out in hospital of atomic medicine, Al-Khansaa Hospital and general hospital of Mosul city, Iraq. 400 patients were included and they were 200 patients with cancer diseases and 200 patients complaining of other illnesses. The acceptance for participation in the present study was taken from all the participants whose native language is Arabic. They were not mentally retarded and they were completely healthy considering hearing and speaking.

Sampling

Swabs were taken from faeces. Swabs were rinsed with sterile nutrient broth as a transport media. 200 samples of cancer patients and 200 swabs were from noncancer patients [18].

Isolation

All samples were inoculated on MacConkey agar (Oxoid). Inoculated plates were incubated at 37 ^{0}C for 24 hours [29].

Identification of Escherichia Coli

The purified isolates of suspected *E.coli* were conventionally identified following methods of workers [30, 31].

Antibiotic Susceptibility Testing

A loopful growth from isolates of *Escherichia coli* were inoculated into nutrient broth and incubated at 37°C for 18 hours. The bacterial suspensions were diluted with ringer solution. The proportion of dilution was 1:1000 [14]. Diluted bacterial suspension were poured onto the surface of the Muller-Hinton agar plates. The excess of bacterial suspensions were discarded using Pasteur pipette and plates were left for one hour at room temperature to dry. The antibiotic discs which are shown in Table 1 were selectively applied by using sterile forceps which was flamed after being cleansed with alcohol. The plates were incubated at 37 0C for over-night. The size of zones of inhibition were measured from edge of disc to the edge of inhibition of growth and the result was compared with standard diameter of inhibition zones for each antibiotic utilizing the method of Bauer *et al*., [19]. The following standard strain *Escherichia coli* ATCC25922 was used as a reference strain [32, 33].

Minimal Inhibitory Concentration (MIC) Testing

Double dilutions of 5 different antibiotics were incorporated in Mueller-Hinton agar plates and they were prepared. A loopful of each isolated organism culture was inoculated into tubes containing nutrient broth and incubated at 370 C for overnight. Dilution of the broth was done up to 100 folds with nutrient broth.

All plates were inoculated with diluted broth culture organisms and incubated at 370 C for 24 hours. The results were read to the point where there was no visible growth and this point called the MIC. Plates containing no antibiotics were incubated in each batch of incubated plates as control [4-34].

Determination of Resistance Transfer

Ten tetracycline-resistant strains of *Escherichia coli* were selected and subjected to antibiotic resistance transfer experiment as donors before and after the disinfectant passages. *Escherichia coli* J53.2 (F− promet rifr) was used as a recipient strain, which was kindly provided by Dr. K.J. Towner (University of Nottingham, UK) and the procedure performed as follows: donors and recipients were grown in 10mL nutrient broth at 37°C for 6 h. Three ml of the resistant donor broth was then mixed with 3 mL of the recipient broth and the mixtures incubated at 37°C for 18 h. Then, 10−1 and 10−2 dilutions of the mating mixtures were prepared in 1:4 Ringer's solution, and0.1 mL of these, together with undiluted mating mixture, were seeded onto selective plates of MH agar containing100 µg mL−1 rifampicin and 50 µg mL−1 tetracycline [1-35].

Statistical Analyses

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics such as means, standard deviations, and frequency distributions were computed to summarize the data. A paired sample t test correlation coefficient (R) and coefficient of determination (R2) were calculated [36-39].

RESULTS

Antibiotic Resistance Transfer

Forty three serotypes of enteropathogenic *Escherichia coli* (EPEC) isolated from intestine of patients were selected for resistance transfer experiment. These resistotypes were resistant to more than one antibiotic at the same time (Table 1). These strains were resistant to tetracycline and rifampicin with Minimal inhibitory concentration not more than 64 µg/ml (Table 1). The rate of resistance transfer was represented in Table 2. The rate of transfer of resistance traits from pathogenic E.coli to *E.coli* K12 was ranged between 10^{-6} to 10^{-3}. The log–linear regression analysis demonstrated a statistically significant positive relationship between donor and recipient resistance-gene transfer frequencies among pathogenic *Escherichia coli* serotypes and *E. coli* K12 recipients ($p = 0.0006$). The regression model showed a moderate degree of association ($R^2 = 0.290$), indicating that increases in donor transfer frequency were associated with corresponding increases in recipient transfer frequency. The logarithmic transformation successfully normalized the highly skewed biological data spanning several orders of magnitude (10^{-6}–10^{-1}), making the model suitable for interpreting bacterial conjugation dynamics. Variability among serotypes suggests that genetic background and serotype characteristics may influence conjugative transfer efficiency. Overall, the analysis confirms that donor transfer capability is an important predictor of recipient acquisition frequency in antimicrobial resistance gene transmission (Figure 1).

Frequency of Antibiotic Resistance Transfer

The antibiotic disc diffusion test of different strains tested demonstrated that the frequency of resistance transfer was different depending on type of antibiotic (Table 3). The frequency of resistance transfer of carbenicillin, ampicillin and sulfonamide was 80.5, 72.1 and 62.5% respectively. This rate of transfer was almost high than media transfer rate. There was a lower rate of transfer for nalidixic acid, colistin and nitrofurantoin and the rate was 20, 3.3 and 0% respectively. The statistical analysis demonstrated a significant positive association between the number of antibiotic-resistant *Escherichia coli* isolates and the frequency of resistance transfer to recipient *E. coli* K12 strains (Spearman $r = 0.788$, $p = 0.0014$). The regression model explained 56.2% of the variability in resistance-transfer frequency ($R^2 = 0.562$), indicating a moderate-to-strong relationship between isolate prevalence and conjugative transfer efficiency. Among the tested antibiotics, tetracycline (TE), carbenicillin (PY), and ampicillin (AMP) exhibited the highest resistance-transfer frequencies, suggesting highly transmissible resistance determinants, likely mediated by conjugative plasmids. In contrast, nitrofurantoin (FM) showed no detectable transfer, indicating limited or absent plasmid-associated dissemination. Overall, the findings confirm substantial variability in antibiotic resistance-transfer capability among different antimicrobial agents and emphasize the important role of horizontal gene transfer in the dissemination of multidrug resistance in pathogenic *Escherichia coli* (Figure 2).

Table 1: Minimal inhibitory concentration (MIC) of rifampicin for strains of enteropathogenic *Escherichia coli* (EPEC) isolated from cancer and non-cancer patients

Strain source	No. isolates	No. (%) strains inhibited by the MICs (µg/ml):									
		0.5	1	2	4	8	16	32	64	128	256
Cancer patients	35	0	0	0	0	0	7(20)	15(42.8)	9(25.7)	2(5.7)	2(5.7)
Non-cancer patients	17	0	0	0	0	0	3(17.6)	8(47.1)	1(5.8)	2(11.1)	3(17.6)
Total	52	-	-	-	-	-	10(19.2)	23(44.2)	10(19.2)	4(7.7)	5(9.6)

Table 2: Frequency of resistance genetic transfer from pathogenic *Escherichia coli* to *E.coli* K12

Sample cod1	Serotype	Frequency of transfer /donar	Frequency of transfer/ reciepient
18CE	026	2.8×10^{-2}	1.1×10^{-3}
5CE	055	5.47×10^{-2}	1.4×10^{-3}
8CE	086	4.6×10^{-3}	1.4×10^{-3}
26CE	086	1.35×10^{-3}	2.4×10^{-3}
9NE	086	7.6×10^{-2}	2.8×10^{-3}
1CE	0119	0	0
3CE	0119	5.76×10^{-2}	9.36×10^{-2}
6C	0119	4.36×10^{-3}	1.5×10^{-3}
10CE	0119	2.2×10^{-3}	2.4×10^{-3}
11CE	0119	8.1×10^{-2}	2.3×10^{-3}
20CE	0119	3.5×10^{-2}	8.1×10^{-3}
24CE	0119	6.46×10^{-3}	1.6×10^{-3}
27CE	0119	3.6×10^{-2}	7.1×10^{-2}
28CE	0119	7.8×10^{-2}	3.1×10^{-2}
30CE	0119	6.6×10^{-2}	4.2×10^{-2}
33CE	0119	3.2×10^{-3}	2.6×10^{-3}
1NE	0119	4.6×10^{-2}	7.1×10^{-2}
17NE	0119	0	0
2CE	0127	7.8×10^{-3}	2.4×10^{-3}
4CE	0127	9.3×10^{-2}	2.2×10^{-3}
7CE	0127	3.3×10^{-3}	2.4×10^{-3}
16CE	0127	2.5×10^{-2}	1.6×10^{-3}
3NF	0127	3.3×10^{-2}	1.2×10^{-2}
5NE	0127	2.4×10^{-2}	2.7×10^{-3}
13NE	0127	6.6×10^{-2}	1.1×10^{-3}
12CE	0125	2.4×10^{-3}	2.3×10^{-3}
6NE	0126	5.2×10^{-2}	2.1×10^{-3}
13CE	0128	0	0
14CF	0128	8.9×10^{-2}	1.8×10^{-3}
21CE	0128	7.6×10^{-2}	2.6×10^{-3}
22CE	0128	1.1×10^{-3}	2.0×10^{-3}
34CE	0128	6×10^{-2}	1.6×10^{-3}
9CE	0114	9.6×10^{-2}	1.3×10^{-3}
17CE	0114	4.3×10^{-6}	6.3×10^{-6}
19CE	0114	6.7×10^{-2}	5.4×10^{-4}
25CE	0114	0	0
32CE	0114	2.9×10^{-2}	1.2×10^{-3}
4NE	0114	2.4×10^{-3}	2.1×10^{-3}
7NE	0114	1.2×10^{-3}	2.5×10^{-3}
8NE	0114	8.0×10^{-5}	2.3×10^{-4}
10NE	0114	2.1×10^{-2}	1.6×10^{-3}
15CE	0142	2.3×10^{-2}	3.3×10^{-2}
15NE	0142	1.1×10^{-3}	3.1×10^{-3}

Table 3: Frequency of antibiotics resistance transfer from pathogenic isolates of *Escherichia coli* to recipient *E.coli K12*

Antibiotic *	No. resistant isolates	% resistance transferred
Tetracycline (TE)	43	90.7
Carbenicillin (PY)	41	80.5
Ampicillin (AMP)	43	72.1
Cephalothin (CF)	32	68.7
Sulfonamide (S3)	40	62.5
Co-trimoxazole (SXT)	21	47.6
Streptomycin (S)	28	39.3
Gentamicin (CN)	5	40
Chloramphenicol (C)	17	35.3
Tobramycin (TOB)	24	33.3
Nalidixic and (NA)	5	20
Colistin (CL)	30	3.3
Nitrofurantoin (FM)	4	0

*=Total number isolates used was 43.

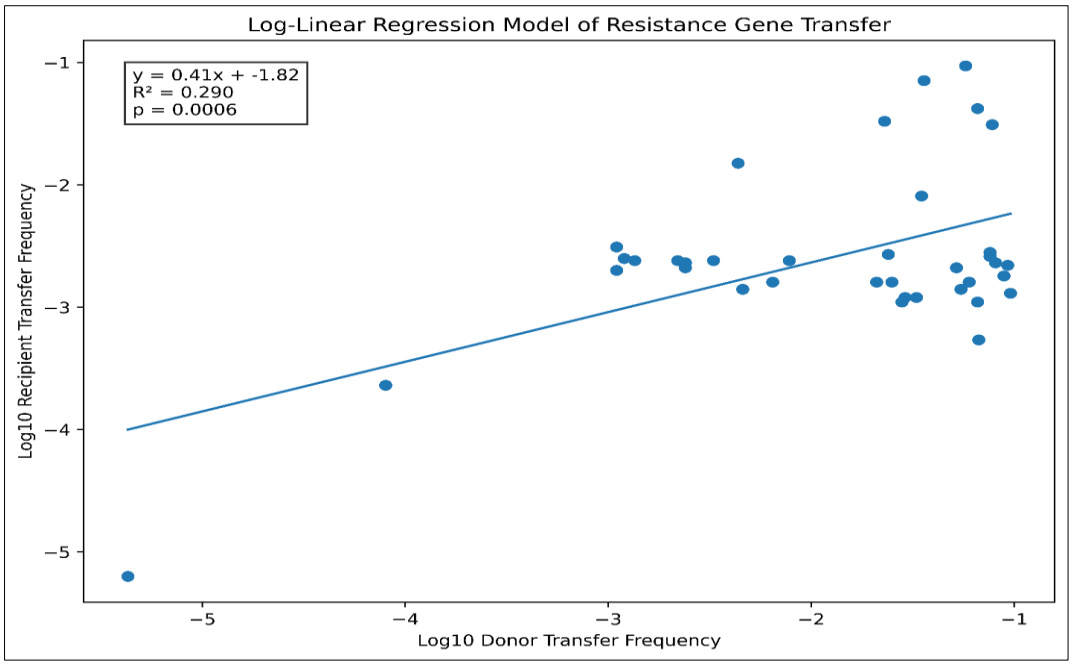

Figure 1: Log–Linear regression analysis of resistance gene transfer frequencies between pathogenic *Escherichia coli* isolates and *E. coli* K12 recipients

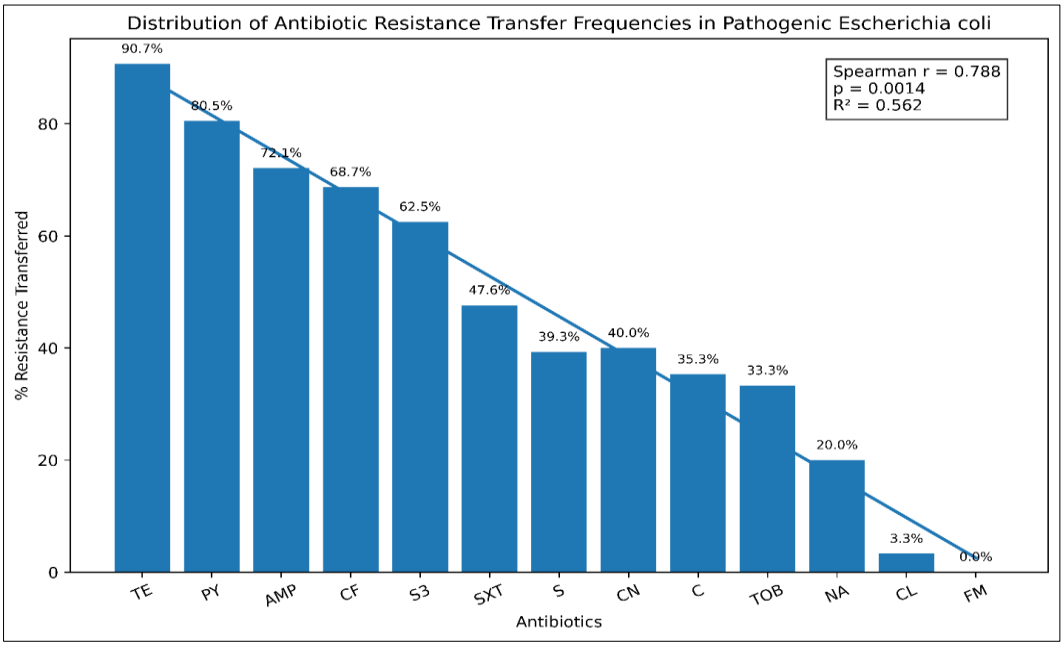

Figure 2: Distribution and regression analysis of antibiotic resistance transfer frequencies in pathogenic *Escherichia coli*

Antibiotics Resistance Patterns Before and After Transfer

It was noticed that all resistance patterns after transfer were not heterologus i.e. some of the resistance determinants transferred but others did not able to transfer to the reciepients (Table 4). It was noticed also that each serotype possessed more than one determinant i.e. multiple resistant. For example, Serotypes 086 and 0119 possessed 3 and 12 resistance determinants respectively. It was also concluded that the local serotypes of EPEC contained similar determinants as it was noticed with serotypes 0119, 0125 and 0128 which possessed the pattern (CL-TE-CF-AMP-TOB-PY-S3. The results documented here suggested that resistance determinants were of different origin either plasmidal or chromosomal. The comparative analysis of antibiotic resistance patterns before and after R-factor transfer demonstrated substantial horizontal dissemination of multidrug resistance among pathogenic *Escherichia coli* isolates. The mean resistance-retention efficiency after conjugative transfer was 59.4%, confirming that a large proportion of resistance determinants were plasmid

mediated and transmissible to recipient *E. coli* K12 strains. Ampicillin (AMP), tetracycline (TE), and carbenicillin (PY) exhibited the highest post-transfer persistence, indicating strong conjugative stability and efficient plasmid-associated transfer mechanisms. In contrast, nitrofurantoin (FM) and nalidixic acid (NA) showed minimal or absent transferability, suggesting that these resistance traits were either chromosomally encoded or weakly associated with transferable plasmids. The reduction in resistance frequencies after transfer indicates selective acquisition of resistance determinants rather than complete transfer of all multidrug-resistance traits. Overall, the findings provide strong evidence that R-factor-mediated conjugation plays a major role in the dissemination of antimicrobial resistance among pathogenic *Escherichia coli* populations (Figure 3). The most suitable mathematical descriptor considered was resistance-retention efficiency modeling: Retention Efficiency (%) = Before Transfer/After Transfer×100

Table 4: Antibiotics resistance patterns of *Escherichia coli* isolated from stools of patients before and after R factor transfer

Strain code	Serotype	Resistance pattern before transfer*	Resistance pattern after transfer
18CE	026	PY, S3, AMP, TE	AMP, TEX
5CE	055	TE, SCF, AMP, SXT, PY, S3, C	TE, CF, PY
8CE	086	TE, S, CF, AMP, SXT, PY, S3, C	TE, S, CF, AMP, SXT, PY, S3.C
26CE	086	CL,FM,TE,S,CF,AMP,SXT,PY,S3,C	TE,CF,PY,S3,C
9NE	086	CL,TE,S,CF,AMP,SXT,TOB,PY,S3,C	TE,CF,AMP,SXT,PY,S3
1CE	0114	CL,TE,AMP	0
3CE	0119	CN,TE,S,AMP,SXT,TOB,PY,S3,C	CN,TE,S,AMP,TOB
6CE	0119	CL,TE,AMPTOB,PY,C	TE,AMP,PY,C
10CE	0119	CL,TE,CF,AMP,TOB,PY,S3	TE,TOB,S3
11CE	0119	CL,TE,CF,AMP,PY,S3	TE,CF,PY
20CE	0119	CL,TE,S,CF,AMP,PY,S3,C	TE,AMP,PY,S3
24CE	0119	CL,TE,CF,AMP,TOB,PY,S3	TE,CF,TOB,PY,S3
27CE	0119	CL,FM,TE,CF,AMP,TOB,SXT,PY,S3	TE,AMP,SXT,PY,S3
28CE	0119	TE,S,CF,AMP,TOB,SXT,PY,S3	TE
30CE	0119	CL,TE,S,CF,AMP,SXT,PY,S3	TE,CF,AMP,SXT,PY,S3
33CE	0119	CL,TE,S,CF,AMP,TOB,NA,PY,S3	TE,AMP,PY,S3
1CE	0119	TE,S,AMP,SXT,TOB,PY,S3,C	TE,S,CF,AMP,TOB,PY,S3,C
17CE	0119	TE,S,AMP,SXT,PY,S3,C	0
2CE	0127	CL,TE,AMP,SXT,PY,S3	TE,AMP,SXT,PY,S3
4CE	0127	CN,CL,TE,AMP,CF,SXT,TOB,PY,S3,C	TE,AMP,SXT,TOB,PY,S3
7CE	0127	CL,TE,S,CF,AMP,PY,S,C	TE,S,CF,AMP,PY,S3
16CE	0127	CL,TE,S,CF,AMP,SXT,TOB,PY,S3	TE,S,CF,AMP,SXT,PY,S3
3NE	0127	TE,S,AMP,PY,S,C	TE,AMP,S3,PY
5NE	0127	CL,TE,S,CF,AMP,PY,S3	TE,AMP,PY,S3
12NE	0127	CL,TE,S,CF,AMP,PY,S3	TE,S,AMP,PY,S3
12CE	0125	CL,TE,CF,AMP,TOB,PY,S3	TE,CF,AMP,PY
6NE	0126	CN,FN,TE,CF,AMP,SXT,TOB,NA,PY,S3,C	TE,CF,AMP,SXT,PY,S3
13CE	0128	CL,TE,CF.AMP,TOB,PY,S3	0
14CE	0128	CL,TE,S,CF,AMP,TOB,NA,PY,S3	TE,CF,AMP,PY,S3
21CE	0128	CL,FM,TE,AMP,SXT,TOB,NA,S3,C	TE,S,AMP,SXT,TOB0
22CE	O128	CN,CL,TE,S,CF,AMP,SXT,TOB,PY,S3,C	TE,S,CF,AMP,PY,S3,C
34CE	0128	CL,FM,TE,CF,AMP,TOB,PY,S3	TE,CF.PY
9CE	0114	CL,TE,S,AMP,SXT,PY,S3	TE,E,CF,AMP,SXT,PY,S3
17CE	0114	CL,TE,CF,AMP,PY,S3	TE,CF,AMP,PY,S3
19CE	0114	TE,S,AMP,SXT,PY,S3	TE,PY,S3
25CE	0114	CL,TE,CF,AMP,TOB,PY,S3	0
32CE	0114	CN,CL,FM,TE,5,SF,AMP,SXT,TOB,NA,PY,S3	CN,TE,S,CF,AMP,SXT,TOB,NA,PY
4NE	0114	TE,S,CF,AMP,SXT,PY,S3,C	TE,CF,AMP,SXT,PY,S3
7NE	0114	CL,TE,CF,AMP,TOB,PY,S3	TE,CF,AMP,PY,S3
8NE	0114	TE,S,AMP,TOB,PY,S3	TE,AMP
0114	0114	CL,TE,S,CF,AMP,TOB,PY,S3,C	TE,S,CF,AMP,PY,S3,C
15CE	0142	CL,TE,S,CF,AMP,PY	TE,CF,AMP,PY
15NE	0142	CL,TE,S,CF,AMP,SXT,TOB,PY,S3,C	CL,TE,S,AMP,TOB,PY

*=CE , cancer patients isolates; NE, non-cancer patients isolates; Cn, gentamicin; TE, tetracycline; CF, cephalothin; TOB, tobramycin; S3, sulfonamide; CL, colistin; S, streptomycin; AMP, ampicillin; NA, nalidixic acid; C, chloramphenicol; FM, nitrofurantoin; SXT, co-trimoxazole; PY, carbenicillin.

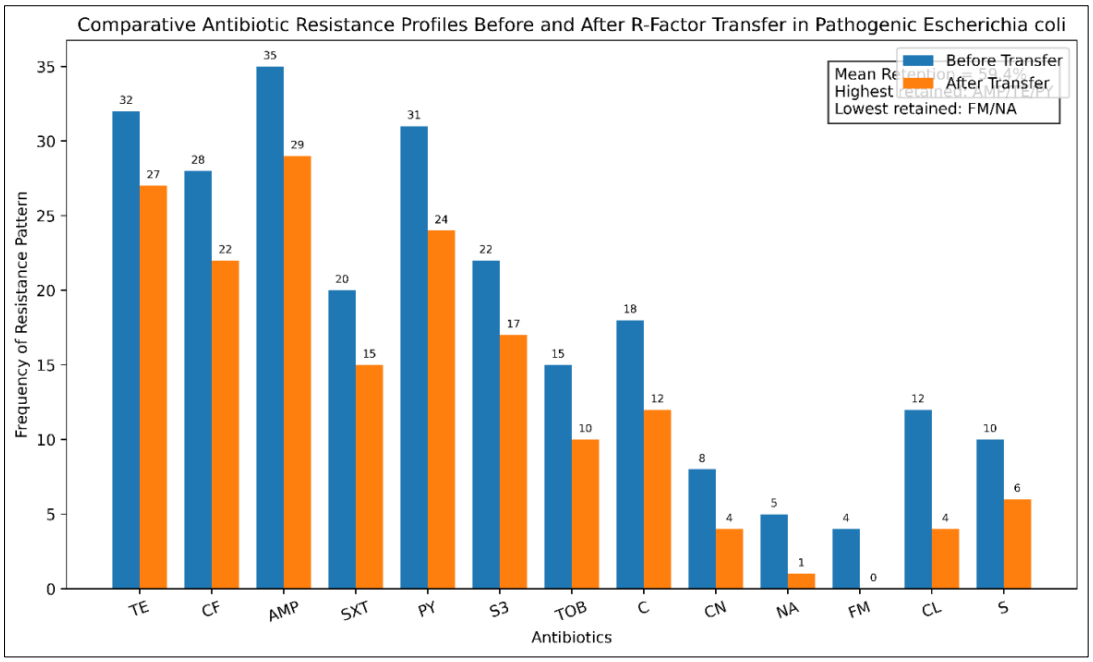

Figure 3: Comparative analysis of antibiotic resistance ratterns before and after R-Factor transfer in pathogenic *Escherichia coli*

DISCUSSION

Forty three serotypes of enteropathogenic *Escherichia coli* (EPEC) isolated from intestine of patients were selected for resistance transfer experiment. These resistotypes were resistant to more than one antibiotic at the same time. These strains were resistant to tetracycline and rifampicin with minimal inhibitory concentration not more than 64 µg/ml. The rate of resistance transfer was represented in Table 2. The rate of transfer of resistance traits from pathogenic *E.coli* to *E.coli* K12 was ranged between 10^{-6} to 10^{-3}. On the other hand, the percentage of the environmental *E. coli* strains which were resistant to two or more drugs (8.3%) was in the range reported by other workers [40-44]. Furthermore, our estimate that 60% of the multiresistant *E. coli* strains were capable of transferring all or part of their drug resistance agreed with the results for coliforms obtained by other workers [45], who reported 53%, Sturtevant and Feary [46], who reported 50%, Anis *et al*., [47], who reported 61%, and Irmela *et al*., [48], who reported 34 to 41%. These percentages notwithstanding, the majority of the strains tested in this study donated their plasmids at frequencies of less than 10-' per donor cell, and it is the number of donor, rather than recipient, cells which will be limiting under natural conditions in the gastrointestinal tract. Furthermore, some of the strains which transferred only part of their resistance determinants may possess nontransferable plasmids which code for the non-transferred resistance markers. An indication of the existence of two compatible R plasmids within the same environmental strain was obtained from the results of the A8 crosses with the fecal and laboratory recipients. Ampicillin resistance alone was transferred to F-76, but it was transferred at a higher frequency than was streptomycin and ampicillin resistance to CSH26 and F-309. The most likely explanation is that A8 has a plasmid coding for ampicillin resistance and one coding for streptomycin resistance. The latter was not detected in F-76 because it was transferred at a frequency of io-4. It was detected in CSH-26 and F-309 because they restricted the plasmid coding for ampicillin resistance. There are a number of possible explanations for the transfer patterns observed with C18. One is that chloramphenicol and tetracycline resistance are on the same plasmid but that the chloramphenicol resistance gene is not passed in most instances. An alternative explanation is that chloramphenicol resistance is on a nonconjugative plasmid which is mobilized by the one containing the tetracycline resistance marker. It is also possible that the non-transferred markers were lost because of host restriction. All of these possibilities could be used to explain the infrequent transfer of ampicillin resistance [49-51]. However, the present study revealed that the antibiotic disc diffusion test of different strains tested demonstrated that the frequency of resistance transfer was different depending on type of antibiotic. The frequency of resistance transfer of carbenicillin, ampicillin and sulfonamide was 80.5, 72.1 and 62.5% respectively. This rate of transfer was almost high than media transfer rate. There was a lower rate of transfer for nalidixic acid, colistin and nitrofurantoin and the rate was 20, 3.3 and 0% respectively. However, the probability that an R plasmid from the ingested R+ E. coli will be transferred to a resident *E. coli* or a subsequently ingested enteric pathogen could be increased if the R+ *E. coli* colonized the bowel of the individual and thereby increased its numbers. Plasmid transfer between *E. coli* strains can occur in the gut [52-56], even in the absence of antibiotics [57-59], although

this is a rare phenomenon [21]. Usually, though, more than 106 bacteria must be ingested in order for colonization to occur [60, 61], and even when 1010 viable organisms are ingested, colonization by R-plasmid-bearing strains is poor [62, 63]. Furthermore, if R-plasmid transfer between an ingested R+ coliform and a resident *E. coli* strain does occur, there is a question as to how long the R+ transconjugant will remain in the bowel [64-68]. This period of time will influence the probability that the R+ bacteria will transfer resistance. Moreover, in 28 % of the resistant strains R factors transmissible to *E. coli* K12 W 3132 could be demonstrated. This does not necessarily mean that R factors were not present in 72 %. It is well known that R factors may be segregated and accordingly loose their transmissibility [69]. However, some workers have found that R factors which have lost tetracycline resistance are often non-transmissible. This might be the explanation why 25 strains carrying resistance determinants to sulphonamide and streptomycin did not transfer resistance [70-72]. The use of more than one recipient strain might have increased the possibility to demonstrate R factors. *E. coli* V\T 3132, however, was shown to be a competent recipient of some R factors, since the frequency of transfer in some experiments was higher than 10^{-2}. Furthermore, It was noticed that all resistance patterns after transfer were not heterologus i.e. some of the resistance determinants transferred but others did not able to transfer to the reciepients (Table 4).It was noticed also that each serotype possessed more than one determinant i.e. multiple resistant. For example, Serotypes 086 and 0119 possessed 3 and 12 resistance determinants respectively. It was also concluded that the local serotypes of EPEC contained similar determinants as it was noticed with serotypes 0119, 0125 and 0128 which possessed the pattern (CL-TE-CF-AMP-TOB-PY-S3. The results documented here suggested that resistance determinants were of different origin either plasmidal or chromosomal [73-79]. The comparative analysis of antibiotic resistance patterns before and after R-factor transfer demonstrated substantial horizontal dissemination of multidrug resistance among pathogenic *E. coli* isolates. The mean resistance-retention efficiency after conjugative transfer was 59.4%, confirming that a large proportion of resistance determinants were plasmid mediated and transmissible to recipient *E. coli* K12 strains. Ampicillin (AMP), tetracycline (TE), and carbenicillin (PY) exhibited the highest post-transfer persistence, indicating strong conjugative stability and efficient plasmid-associated transfer mechanisms [80-85]. In contrast, nitrofurantoin (FM) and nalidixic acid (NA) showed minimal or absent transferability, suggesting that these resistance traits were either chromosomally encoded or weakly associated with transferable plasmids [86-90].

CONCLUSIONS

The rate of transfer of resistance traits from pathogenic E.coli to *E.coli* K12 was ranged between 10^{-6} to 10^{-3}. The log–linear regression analysis demonstrated a statistically significant positive relationship between donor and recipient resistance-gene transfer frequencies among pathogenic *Escherichia coli* serotypes and *E. coli* K12 recipients ($p = 0.0006$). The frequency of resistance transfer of carbenicillin, ampicillin and sulfonamide was 80.5, 72.1 and 62.5% respectively. This rate of transfer was almost high than media transfer rate. There was a lower rate of transfer for nalidixic acid, colistin and nitrofurantoin and the rate was 20, 3.3 and 0% respectively. The statistical analysis demonstrated a significant positive association between the number of antibiotic-resistant *E. coli* isolates and the frequency of resistance transfer to recipient *E. coli* K12 strains (Spearman $r = 0.788$, $p = 0.0014$). Overall, the findings provide strong evidence that R-factor-mediated conjugation plays a major role in the dissemination of antimicrobial resistance among pathogenic *E. coli* populations (Figure 3). The most suitable mathematical descriptor considered was resistance-retention efficiency modeling: Retention Efficiency (%) =Before Transfer/After Transfer×100.

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