

SUMMARY

SUMMARY AND CONCLUSION

Ps. aeruginosa has been considered to be one of the causes of serious diseases in man and it infrequently is the cause of infection in normal individuals unless they have suffered from ~~major~~ trauma or burns. It is involved in respiratory, cutaneous and disseminated infections in individuals who have defective host defenses of cutaneous barriers, granulocytes or immunoglobulins.

Several plasmids encoding resistance to one or more antibiotic could be identified from clinical isolates *Ps. aeruginosa*.

The present investigation was carried out in order to clarify the genetic basis of *Ps. aeruginosa* antibiotic resistance.

Five *Ps. aeruginosa* isolates were used, two isolates; 1 & 2 were isolated from the urine of two patients with chronic urinary tract infection and three isolates, 3, 4, and 5, were isolated from patients with cancer bladder.

The results obtained showed that all five isolates were equally sensitive to amikacin and were sensitive to tobramycin with different degrees. The most resist-

ant isolate to tobramycin was no. 1. The most resistant isolate to kanamycin was isolate no. 1 followed by isolate; 4, 3 and 5 respectively while isolate no. 2 was sensitive. Also isolate no. 1 was highly resistant to streptomycin followed by isolates; 4, 5 and 3, respectively, while isolate no. 2 was sensitive. The four isolates; 1, 2, 4 and 5 were resistant to gentamicin. Isolate no. 1 was the most resistant one to gentamicin followed by isolate no. 4, while the isolates 2 and 5 were equal in their resistance to gentamicin and less than isolate no. 4. Isolate no. 3 was sensitive on the border line.

Isolate no. 1 which was resistant to the four antibiotics had the lowest MIC of amikacin which reached 50 $\mu\text{g/ml}$ followed by Tm and Gm which reached for either 400 $\mu\text{g/ml}$, while those for Km and Sm more than 400 $\mu\text{g/ml}$. MIC for isolate no. 2 to ; Am, Tm, Km, Sm and Gm were; 25, 50, 100, 200 and 100 $\mu\text{g/ml}$ respectively. Also MIC for isolate no. 3 to these antibiotics were, 12.5, 100, 400, >400 and 50 $\mu\text{g/ml}$ respectively, for isolate no. 4 they were 25, 100, >400, >400, and 50 $\mu\text{g/ml}$ respectively and for isolate no 5 were; 12.5, 25, 100, 200 and 100 $\mu\text{g/ml}$ respectively.

Single colonies of isolate no. 1 showed great variations in their levels of resistance to the differ-

ent antibiotics indicating that the resistant genes are located in different plasmids. Isolate no. 2 single colonies were similar in their resistance to gentamicin levels indicating chromosomal mode of inheritance. In isolate no. 3, Km and Sm resistant genes were encoded by the same plasmids. Isolates no. 4 and 5 single colonies showed different levels of resistance to, Km, Sm and Gm with great agreement for resistance levels indicating the existence of the genes in the same plasmids.

The percentages of *E. coli* k12 transformants which resist 10 $\mu\text{g/ml}$ of each antibiotics ranged from 1 - 1.75 when plasmid DNA was used as donor. The highest levels of resistance appeared for *E. coli*, k12 transformants to streptomycin followed by those; Km, Gm and Tm respectively.

Transformation of the Gram positive *S. aureus* and *B. subtilis* with plasmid DNA isolated from *Ps. aeruginosa* was unsuccessful.

E. coli k12 transformants indicated that the lowest resistance to Km which can be acquired with a single plasmid is probably 10 μg Km/ml medium. The level of 50 μg Km resistance can be obtained from double dose of plasmid as no. 20, 30 or 40 μg Km/ml

resistance could be obtained. This increase in the double dose had been attributed to gene interactions and gene regulatory mechanisms. Three plasmids were responsible for the level of kanamycin resistance reached 100 $\mu\text{g/ml}$, 4 plasmids for the level of 200 $\mu\text{g/ml}$.

One plasmid was estimated to give 50 μg Sm/ml resistance levels, 2 for the level of 100 μg , 3 and 4 plasmids for the levels of 150 and 200 $\mu\text{g/ml}$ respectively.

Each plasmid gave a resistance level of μg Gm/ml medium. The possibility of gene interactions can also be existed. the same appeared for tobramycin.

Positive correlation between bilharzia, cancer high bacterial counts and high phage contents and no clear correlation was noticed between phage contents and the resistance of any of the antibiotics studied.

In conclusion plasmids play an essential role in antibiotics resistance. To solve this problem it is essential to look for plasmid curing. Curing can occur spontaneously during growth and cell divisions or following treatments with some agents such as elevated temperatures, acridine hydrochloride, ascorbic acid and the best therapeutic treatments might be through safe vaccinations.

REFERENCES