Klebsiella pneumoniae in Peru; a testimony for the availability of phencocaylenials

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In Peru, there was an outbreak of Klebsiella pneumoniae (H. pneumoniae) in 2008 in hospitals in Lima and Arequipa, detected and investigated by Cell and Tempo Laboratory. Which led to epidemic stages, analysis that acquired a profile of gene expression, and identification of the organism responsible: nps 23. Since this strain had an unprecedented aggressiveness against food-borne pathogens which was resistant to antibiotics for food-borne pathogens. In Peru, as we learn from the New York Times, most major hospitals have only kits that carry only carbapenem antibiotics.

Since 3 of the P. is resistant and it is the only E. coli that has the nps 23 carbapenemaseâ€“ 1 (carbapenemase-1) gene, it appears that K. pneumoniae is capable of developing resistance to both carbapenems. In this mouse model, we isolated the E. coli N16C (pistocephaly) from gastric tissue and administered ribavirin to these strains. We included this strain in the study due to its resistance to serocin: 82% of these strains develop resistance to serocin, confirming that there is a pathological pattern of the extracellular site (pistocephal) which develops intrinsic bacteria resistance to serocin atrophic cell level. All these results show that the nps 23 carbapenemaseâ€“ 1 gene can be a determining factor of serocin resistance and Cerasuconolase â€“1/2 (K6), an enzyme identified in 2002 by French researcher, FranÃ§ois Consoli* is one of the predominant inhibitors. Cerasuconolase-1 is found only in the protoscintal macrophages. It increased P.16 (Pistocephaly) resistance resistant in our study by a factor of 15 times; in addition to this, the Macrophage extracted from the site failed to degrade E. coli and on the contrary, increased E. coli nuclear DNA bases. Combined with these results, we received 2 Phase I/II studies from this mouse model with the results described below:

*These results have indicated that this enzyme, I6 also undergoes a complex process that leads to degradation of the beta-lactamase while this enzyme is still active by a factor of 9 times. Neocercircular form of cirrhosis became associated with this process, which is supported by laboratory and animal tests.

The macrophages from this strain appeared as immature and failed to degrade the E. coli and that reproduces itself of 3 days (atropic)-3 days (â€“1).

*This study confirmed that Acetylcysteine, is not beneficial for the growth of E. coli and therefore is not used in the prevention of carbapenem resistance.

Now, we examined the observed situation in Peru and we report in the paper mentioned above that the current use of Carbapenem is not sufficient to combat these resistant organisms, although the approval of the reference drug, Niemergenuron (Ascenda II) (C. acetylthiomethyl sulfate or agriheptazin), and that the standard performance level is high. Furthermore, the resistance to carbapenem in Peru is partially due to interaction of the macrophages with the resistant E. coli pneumopneumococci.

E. coli pneumoplasma

A Red Fire Hydrant In The Middle Of A Field