

# A longed-for macrophage-induced antifungal resistance is found in *Krabsiella pneumoniae* infecting people in the Amazon

Authors: Mandy Russell Gabrielle Lowe Joel Mitchell Daisy Gonzalez Lisa Lawrence

Published Date: 08-25-2016

---

Grand Canyon University

School of Economics

---

A recent study led by Ana Mena and others and conducted by A. M. Borrell and colleagues studied the DNA sequence of large bacteria that caused lung infection among participants at the University of Antioquia- University of the Greater Amazonas in Acre, Colombia. They also studied the development of resistance to the antifungal oral enzymes C. m23-335 or C. l2-92 and the azole azacitidine, as reported in PLoS ONE.

Abstract of the study:

The investigation began with the collection of DNA samples taken from cough drops in three geographical locations: Acre, an Amazonian region of Colombia, and the Ecuadorian Monas, in the state of Sucumbios. A unique characteristic of the wet humid environment of tropical forests like the tropical rainforest is that host bacteria are able to contaminate non-host organisms with their DNA at a relatively high rate. This was the case with *Klebsiella pneumoniae* (K. pneumoniae), a prevalent bacterium in the pulmonary tract. All dead *Klebsiella* were collected from hospital patients with severe pulmonary infections. The bacterial diversity of the K. pneumoniae was analyzed for patterns of genetic exchange between surviving and deceased samples. In vitro analysis revealed extensive accumulation of rhizobium-associated sequences in both raw and dried specimens. Carbon-16 microarrays on the Krebsach and amoxicillin chromosomes of identified samples from various tissues revealed an association between immunity to one of the approved oral-inhaled antibiotic regimens and resistance to azacitidine. The presence of electron microscopy analysis demonstrated that existing H.11min-165212 nucleotide sequences are transcribed into a novel sequence after the induction of eosinophilic machinery in an animal cell line.

In order to further investigate the mechanisms that explain this observation, an expansion of the study was carried out to determine the specific proteins associated with this transformation.

The characteristics of two shared proteins were selected for antibody-based selection. This resulted in identification of an inhibitory EGE protein encoding the EGEF1 and an insulin-like growth factor-1 cell membrane factor during yeast studies. Neurons derived from cultured mouse endothelial cells linked cytokine and cell membrane proteins to human immune response in whole-genome polymorphism. Further, EGEF1 level was positively correlated with HLA response during a mouse model in vitro.

Results:

The signatures of the two proteins determine resistance to oral-inhaled drugs. In addition, levels of the inhibition of EGEF1 increased the concentration of insulin-like growth factor-1 in the respiratory tract, for example, in transgenic mice with HLA-A2 alpa, prompting them to mount an immune response. The point of the antibody-based test is to identify specific proteins of the community that prevents epithelial cells from supporting immunity, thereby reducing the resiliency to the allergy hypothesis.

About the authors:

This study was conducted with collaborators: Cesar Roque, H. F. Allegra-yab, ElÃas Chiaravuori, M. B. Stryker, D. R. Murillo, I. PÃ©rez-GonzÃ¡lez, Daniel DÃ¡az, M. Ricard, Nuria Palavia-GusmÃ¡n, Claudia Cardoso, Susana Roca, IvÃ¡n CaÃ±izares, Francisco Aguilar, Victoria Ochoa, Francisca MariÃ±ana, Isabel Espinosa, N. JimÃ©nez, CÃ©sar Saillan-Hoyos, JosÃ© Ceballos, JosÃ© Manuel MartÃ¡nez-JimÃ©nez, Rodolfo Medrano, Luis Malatesta, Jose SÃ¡nchez-Pedrosa, J. Mendoza, Luis Ruiz-Aubrey, and L. Borrell, together with other laboratory and collaborators.



A Large Brown Bear Walking Across A Lush Green Field