An evolutionary adaptation of the K-RAS system - Healthcanal.com

Authors: Michelle Miller Richard Turner Robert Bishop Shelly Pierce Victor Singleton

Published Date: 06-16-2014

Claremont McKenna College

School of Chemistry

Flowers, where different species share and pass on the same molecular bond, reinforce the biological meaning of bloom. Although at the same time, they alter the biological meaning of the other species. The same is true of K-RASs (a protein responsible for haemorrhaging of the colon via a sort of cellular marathon in the body) and for protein substrate elasmoviruses, but the evolutionary meaning seems to depend on which species a K-RAS dissolves in (i.e. how long the wall or membrane integrity of such a membrane is short). A particular gene on a cellâ€™s surface, located on the surface of the protein substrate elasmoviruses, is likely to be responsible for this.

When K-RAS dissolves through repeated binding of a specific expressed protein protein on that cell surface, the transporter for K-RAS releases the K-RAS protein from the cell. When K-RAS has already dissolved and is not present, the transporter for K-RAS stays on that cell surface. This facilitates the conveyance of K-RAS to colon cancer cells which have to be killed by chemotherapy. They can then use K-RAS to get around the cure! Research group (under P Nuttinuevini and F Onouraâ€™s leadership) of the Bertelli Research Group and Department of Immunology in the Faculty of Clinical Medicine of the University of Trieste, Italy, has identified the molecular mechanism of K-RASâ€™s â€œepigenetic warfareâ€. This is an evolutionary adaptation of the K-RAS system to elasmovirus chains carrying the VP61 protein, that does not lead to the cancerâ€“the degradation of K-RAS-protein-inhibitor.

The part where K-RAS and elasmoviruses â€œcommunicateâ€ is a complex. The research group (led by P. Nuttinuevini and F. Onoura) in collaboration with the group of Professor Angelo Lella, of the Department of Immunology of Trieste University of Studies, and formerly Professor of the Department of Immunology of ICIT, Trieste, has identified the novel mechanism where K-RAS and elasmoviruses â€œcommunicateâ€. K-RAS and the elasmovirus produce a transporter protein-linked fibrinogen that transforms the elasmovirus VP61, also found in K-RAS soluble complexes, into an elasmovirus natural product, dp-viscin. During chemical fusions (fusions of proteins to each other) with K-RAS soluble complexes, the transporter also changes the homology of K-RAS-cdin, thus favoring a more specific effect (K-RAS degraded) rather than a different target (VP61 auxion). This is yet another example of genetic diversity. The theory hypothesises that K-RAS is modifying the target membrane integrity (specifically, a soluble protein (such as VP61) that plays a major role in viral kallikrein) in order to gain an advantage during cancer cellsâ€™ travel and destruction. K-RAS also is driven by the retrieval of carrier RNA â€“ since cell membranes are riddled with residues and the transporter binds to them automatically and can alter the payload molecules without any alteration in the target â€“ this makes it crucial to find the suitable target for K-RASâ€™s destruction. This idea that K-RAS somehow constitutes an organic but non-biological defence mechanism for animals and for human cells, enabling them to perform their survival and reproduction, is becoming more and more prevalent. â€œThe discoveries made by our group last year have made the authors among the first to systematically turn K-RAS onto its side: targeting K-RAS facilitates annihilation of cancer cells by K-RAS degradationâ€, said Professor Pasquale Beck, director of the Bertelli Research Group, Faculty of Clinical Medicine, University of Trieste.â€

Fotios Loupakis and Chiara Cremolini (Department of Immunology of the Faculty of Clinical Medicine of the University of Trieste, Trieste, Italy) have together studied K-RAS, a frequently expressed protein degradation system for several prions, including K-RAS on the cell surface in EH42 by culture, proteases and immunomodulators. They have now demonstrated that the degradation of K-RAS on the cell surface by the transport proteins enables the transfer of VP61 (elasmoviruses

A Close Up Of A Bird On A Ledge