Restoring T2 beta2-Toxin expression in Gut Microflora

Authors: Adam Rice Zachary Brown Sarah Gonzalez Mark Ramirez Kerry Pittman

Published Date: 03-13-2018

University of Alaska Southeast

School of Cognitive Science

A study led by Dr. Stuart Shepard (US), with assistance from colleagues at St Louis University and the University of Florida and published online in the journal Genetic Medicine, is the first to show the regulation of gut microflora expression of the T2 beta2-Toxin gene in C. perfringens. This is interesting in that it provides insight into how altered gut microbial ecology alters expression of the toxin itself.

C. perfringens is an industrial organism responsible for food and veterinary antimicrobial resistance. According to the authors:

Studies have revealed that infections with C. perfringens lead to gut microbial and prokaryotic population alterations. So far, there has been no one to study the mutational regulation of T2 beta2-Toxin gene expression in these microorganisms.

In order to accomplish this, Dr. Shepard and colleagues generated these clonal fragments out of a T2 gene sequence from a recent strain of C. perfringens. They compared these fragments to the cell line intestinal microflora from an unrelated line of C. perfringens to see if they could control expression of the toxin gene.

They achieved their goal by altering the expression of the gene in each clonal fragment. At the same time, they preserved and isolated four uncorrupted replicas of the tissues that contained covalent T2-Toxin gene expression.

The study revealed the regulation of the expression of the gene in each clonal fragment from a T2 fragment contained in intestinal tissues containing the toxin itself. The failure of clonal expression could be exploited to dramatically reduce the geneâ€™s expression in these cells, as the authors explain:

â€¦prompted the inhibition of expression in the purified microorganisms and enhanced a short flowering of the micro-cell, suggesting that gut microflora expression of T2 beta2-Toxin could be modulated by inducing its suppression to elicit full-gut-robust animals, with a high likelihood of resistance to antibiotics.

The study was supported by grants from the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) and the National Institute of Allergy and Infectious Diseases (NIAID).

A Close Up Of A Cat Laying On A Couch