# Researchers find chink in the armor of viral 'tummy bug'

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Researchers at Griffith University's Institute for Glycomics in collaboration with colleagues at the University of Melbourne have moved a step closer to identifying a broad spectrum treatment for the dreaded 'viral tummy bug' or rotavirus.

These highly-infectious viruses are the leading cause of severe diarrhoea in young children, responsible for thousands of hospitalisations in the developed world, and hundreds of thousands of deaths each year in developing countries.

Institute Executive Director Professor Mark von Itzstein said research findings published in the world-leading Chemical Biology journal *Nature Chemical Biology* this week demanded a total rethink of how these viruses work.

"Rotaviruses are thought to infect the bodies by sticking to certain types of sugars called sialic acids on the surface of our stomach cells. They then enter cells and reproduce rapidly, causing illness," he said.

"Rotavirus vaccines are still in their infancy, as problems emerged with the first vaccine that was trialled a number of years ago. While other vaccines are now in clinical use, new directions are required in the development of potential drugs to prevent or treat this deadly virus."

He said that to better understand how carbohydrates are involved in rotavirus infection, researchers had focussed on treating mammalian

cells with a protein called sialidase which cuts these surface sugars so the virus cannot attach.

Previous to his group's work most scientists believed only some of the many strains of rotavirus infection could be prevented with sialidase treatment while others were apparently immune to its effects.

This led to the conclusion that some viruses depend on sialic acid to infect the body while others were thought to cause infection independent of sialic acid.

"Unsuccessful attempts to reduce rotavirus infection with this treatment led scientists to group rotaviruses into two classes: 'sialidase-sensitive' and 'sialidase-insensitive' strains," he said.

The team used nuclear magnetic resonance spectroscopy, 3D modelling and cell-based assays to observe the interaction between the virus and host cells.

"We found that a human strain previously through insensitive to sialidase does in fact recognise and bind to sialic acid, but it is a sialic acid not accessible to sialidase treatment." Professor von Itzstein said.

"This reveals that there is a common chink in the armour of these rotaviruses.

"This discovery is the first step in designing a broad-spectrum drug able to exploit this weakness to combat many types of human and animal rotaviruses."

Source: Research Australia