**Food Poisoning Pathogen May Work with Bacilli to Break Down Sugars**

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**Abstract:**

**Food poisoning is caused by consumption of contaminated food and can cause nausea, vomiting, abdominal pain, diarrhea, and fever. It typically lasts for 1-2 days and can lead to considerable dehydration. *Campylobacter jejuni*, a common cause of food poisoning, was considered to be incapable of breaking down carbohydrates for energy. However, it was found that over half of sequenced isolates included a regulator of L-fucose that could be used to metabolize sugar. However, the steps used for the catabolism remained unknown. FucX, a *C. jejuni* enzyme, breaks down L-fucose and D-arabinose when cultured and both are broken down by fuc-operon encoded enzymes. The enzyme permits movement of both sugars along a non-carbohydrate utilizing *C. jejuni* strain. *C. jejuni* favors using amino acids over using carbohydrates. This provides evidence that a metabolic hierarchy exists within the pathogen. The study examines the nutrient metabolism of *C. jejuni*, and identifies its connections with other gastrointestinal microbes.**

**Introduction**

*Campylobacter jejuni* is commonly found in chickens and is transmitted to humans when they consume contaminated or undercooked meat. It causes infections that usually clear up on their own, but they can also be treated with antibiotics. There are known to be over 800,000 cases of food caused by *Campylobacter* itself per year in the United States according to the Centers for Disease Control and Prevention (CDC). Unfortunately, complications can occur after infection. These can include but are not limited to: irritable bowel syndrome, Guillain-Barre syndrome, reactive arthritis, and growth stunting. The growing antibiotic resistance of *C. jejuni* is cause for expanded concern, especially regarding fluoroquinolones.

In the past, *C. jejuni* was once considered to be asaccharolic. This means that it is not capable of breaking down carbohydrates for energy. This was thought because *C. jejuni* does not have key enzymes from the pentose phosphate and Entner-Doudoroff pathways that are used to metabolize carbohydrates. Instead, it gets a lot of its nutrition from amino acids that are plentiful in the gastrointestinal tract of chickens. Amino acids are the building blocks of proteins.

There is a working fuc locus for L-fructose utilization in about 65% of sequenced strains of *C. jejuni*. “L-fucose is a monosaccharide that is a common component of many N- and O-linked glycans and glycolipids produced by mammalian cells. It is the fundamental sub-unit of the fucoidan polysaccharide ([PubChem)](https://pubchem.ncbi.nlm.nih.gov/compound/L-Fucose)).” L-fucose helps to maintain the health of the host by providing nutrients, resistance to pathogens, a functional receptor for cholera toxin, and defense against Crohn’s disease. Since *C. jejuni* doesn’t have evident fucosidase homologs, it is thought that they hunt for L-fucose released by other microorganisms. Homologs are things that are similar in position or structure, but do not necessarily have similar functions.

The paper examines the transfer between *C. jejuni* and *B. vulgatus*. The two have a commensal relationship where *C. jejuni* is able to forage and metabolize L-fucose released by *B. vulgatus*. Commensal relationships occur when one organism gets a benefit from another without hurting or helping it. With knowledge of how the pathogen *C. jejuni* procures sugars from other microorganisms and also how it uses carbohydrates, scientists can better strategize future treatment and prevention options.

**Recent Progress**

Due to the research being published in January of 2020, there has not been ample time for additional research to be conducted based off of the research at hand. However, the scientists believe that their research will aid in the development of treatment options for food poisoning that is caused by *C. jejuni*.

**Discussion**

*C. jejuni* has a competitive advantage because of its ability to use L-fucose. Therefore, fucose metabolism gives the pathogen a fitness advantage in the host. It was hypothesized that instead of cleaving sugars from the host, C. jejuni scavages for free or released fucose. In order to test the hypothesis, many experiments were performed to confirm and validate the findings. Some of these included co-culture experiments with *B. vulgatus*, RNAseq experiments, crystallography, and biolog screening.

*B. vulgatus* is a gut microbe known to express fucosidases, enzymes that help break down oligosaccharides that are attached to glycoproteins and glycolipids and serve to cleave off fucose (NIH). *B. vulgates* packages catalytic enzymes into secreted outer membrane vesicles. This provides the perfect opportunity for *C. jejuni* to acquire the fucose. The findings supported that nutrient sharing is prevalent in the gut.

Understanding carbohydrate metabolism and how other carbon sources impact the interactions within *C. jejuni* can provide insight on how nutrition effects disease severity. Expanding on this research could help in the development of affordable nutrition therapies.

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