



Unlocking the colonization of *Actinobacillus*

Researchers have examined more than 1,000 mutant strains of the *A. suis* pathogen in an attempt to find out how it infects the pig's upper throat and tonsils

A *ctinobacillus suis* is an opportunistic bacterial pathogen of swine which can cause serious disease, especially in high health-status herds.

This organism most often causes septicaemia and diseases

such as arthritis and meningitis which come with septicaemia. *A. suis* can also cause enteritis, mastitis, metritis and abortion.

It's well known that *A. suis* is able to colonize the upper respiratory tract tissues of a very young pig, with no outward symptoms to indicate infection has occurred. However, later in the pig's life, at times of stress such as weaning, farrowing and transportation, this bacterium can become active and cause disease.

"What's not well understood is exactly how *A. suis* causes disease," says Janet MacInnes of the Department of Pathobiology at University of Guelph.



(L-R) Shivani Ojha, Technician,
and Dr. Janet MacInnes

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In order to understand better how this pathogen initially attaches and colonizes a pig's upper throat and tonsil tissues, critical events in the infection process, MacInnes and her colleagues are doing detective work at the genetic level.

Working with Shivani Ojha of the Laboratory for Foodborne Zoonoses (Guelph) and Sonia Lacouture and Marcelo Gottschalk of the Université de Montréal's Faculty of Veterinary Medicine, MacInnes has examined more than 1,000 different *A. suis* mutant strains. This has led the team to identify 30 genes necessary for colonization of the upper respiratory tract of swine.

The next step for the researchers involved conducting in-

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depth lab studies to see how some of these mutant strains attach and invade samples of swine tonsil, kidney and brain tissue. "Although the tissue (tonsil) and cell (kidney and brain) cultures used in the lab do not retain all of the features of tissues in the pig's actual body," says MacInnes, "they do possess similar characteristics and give useful results."

One of the four *A. suis* mutant strains that MacInnes and her colleagues selected to focus on in detail was of particular interest. This mutant strain lacked the major cell surface protein called OmpA. In some human pathogens, this protein has been shown to be important for bacterium to stick (adhere) to host cells and even invade some cells.

By comparing the adherence of the naturally occurring *A. suis* and the OmpA mutant strain, the research team found that the OmpA strain attached much less well to tonsil organ explants and swine kidney cells than the naturally occurring type. "We also found that the OmpA mutant strains could not invade kidney epithelial cells efficiently," says MacInnes.

The researchers also looked at the ability of the OmpA mutant strain to adhere to porcine brain cell cultures. "*A. suis* can cause meningitis," MacInnes notes, "whereas the closely related pathogen *Actinobacillus pleuropneumoniae* (the causative agent of pleuropneumoniae) can't." They found that neither the *A. pleuropneumoniae* strain tested nor the OmpA mutant could bind to the brain cultures, but the naturally occurring *A. suis* strain with its intact OmpA protein did adhere tightly to these cells.

"Discovering differences between how the OmpA protein functions in *A. suis* versus *A. pleuropneumoniae*," notes MacInnes, "will help us understand how these two very similar organisms interact with the host differently and cause very different disease profiles."

In the other major part of the investigation, the researchers wanted to find out if the OmpA protein helps *A. suis* to survive in the presence of immune system components such as serum and other stressors.

"We found that mutant cells which lack the OmpA protein are killed in the presence of serum," says MacInnes. "This suggests that the presence of the OmpA protein in naturally occurring strains helps to protect *A. suis* against the immune system in the host."

In addition, the researchers found that the OmpA mutant survived significantly less than did the naturally occurring strain in acidic and high salt conditions. These results also demonstrate that the OmpA protein may be important in protecting *A. suis* from a variety of stressors which the organism might encounter in the pig or environment.

MacInnes says that more work is needed to understand precisely how the OmpA protein functions in *A. suis*, but the tonsil culture model developed in these studies may lead to a better understanding of how a variety of important swine pathogens can establish themselves in the pig and cause disease.

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