NZMS 2010 student prize winners

The student competitions for the best oral presentations and posters are excellent avenues to showcase students' research projects.

The projects presented range from undergraduate to postgraduate levels and the standard is usually of high quality with 2010 being no exception. Following are the NZMS student prize winners of the 2010 NZMS/NZSBMB Joint Meeting (November 30-December 3, 2010) in Auckland:

Oral Presentations

1st Prize - Andrew Wood (University of Auckland)
2nd Prize - Ron Xavier (AgResearch and University of Waikato)
3rd Prize – Manpreet Dhami (University of Auckland)
Poster Presentations
1st Prize - Leo Germoni (University of Otago)
2nd Prize - Jason Ryan (Industrial Research Ltd and University of Canterbury)
3rd Prize - Barbara Govan (AgResearch)
Congratulations (albeit belated) to all winners.

All prize winners were approached to provide a short article describing their research projects and here is a selection of articles.

Investigating the microbiology of chronic sinusitis

Departments of Surgery and Molecular Medicine and Pathology, University of Auckland.

By Andrew Wood

Why worry about a snotty nose? While in themselves the symptoms of a blocked, runny nose, facial congestion and a loss of sense of smell may seem of little significance, for those who suffer with sinusitis every day, it has an impact on their quality of life comparable to angina or chronic lower back pain.

Over the years, there has been significant controversy around what might be causing chronic sinusitis (and therefore the best way to treat it) such that to date, many patients are still afflicted with ongoing problems despite the best available treatment regimes. While the use of antibacterials does produce some improvement in patients' symptoms, antibiotics are far from being a universally successful treatment, leading researchers to focus on a variety of other possible causes such as allergy or fungal infection.

In 2004, the presence of bacterial biofilms within the sinus cavities of patients with chronic sinusitis was first described. Since that time, several research groups have described their presence in sinusitis, principally using scanning electron microscopy and confocal microscopy to examine whole tissue samples. The concept of biofilms providing a reservoir of bacteria protected from antibiotics and the immune response, yet stimulating chronic inflammation, is an attractive hypothesis. While biofilms appear to be prevalent in chronic sinusitis and much less so in samples taken from control subjects, they have not been shown to directly cause the ongoing inflammation that characterises this disorder.

Our study is the first to directly relate the presence of biofilms on the surface of the sinus lining to the inflammatory response going on in the tissue. However, we have shown that not all bacteria present in the sinuses during chronic sinusitis cause inflammation. Those bacteria that are adherent to the surface of damaged tissue (adherence being generally considered a key step in biofilm formation) are associated with a more vigorous inflammatory response. This further supports the hypothesis that adherent biofilms stimulate inflammation in chronic sinusitis.

Now that we have better characterised the bacterial flora that exists in chronic sinusitis and its related inflammatory responses, we are moving towards both animal and human studies of appropriately directed therapies.

The 2010 New Zealand Microbiological Society Conference was an excellent forum both for showcasing my research but also exposure to a diverse array of microbiological research areas. I have found that it is only when you move away from meetings focused on the narrow scope of your own research to conferences such as the NZMS Conference that you generate truly novel paths to follow in your field.

I have been enormously lucky in the support that I have received in my research. I have enjoyed superb academic support from my supervisors at the University of Auckland: Dr. Richard Douglas, Prof. John Fraser and Dr. Simon Swift among others. My research has been very generously funded by the Garnett Passe Rodney Williams Memorial Foundation, The Green Lane Research and Education Fund and the University of Auckland Faculty Research Development Fund.

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It's a feast or famine –

Can Escherichia coli O157:H7 survive the rigors of starvation and go on to live another day?

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E. coli O157:H7 is a food-borne pathogen that can cause serious, often life-threatening illness. The symptoms can be relatively minor, but in some cases it can progress to bloody diarrhoea, and haemolytic uraemic syndrome (HUS). Supportive treatments are given during the acute phase of the illness but the resulting damage to the kidneys can potentially lead to long-term complications1.

Historically, E. coli O157:H7 has been regarded as the "burger bug" because of its association with undercooked beef patties. However, recent outbreaks have been attributed to other food sources, such as fresh produce and raw dairy products2. Ruminants, particularly cattle, have been identified as the main animal reservoir for this zoonotic pathogen.

Survival outside the ruminant host in soil3 and water4 has been demonstrated, widening the range of sources for transmission to humans. In New Zealand, infections are sporadic, and commonly associated with environmental and familial transmissions5. Thus, E. coli O157:H7 is now increasingly being recognised as an environmental pathogen.

The question that my research aims to unravel is how E. coli O157:H7 survive in two quite diverse environments - the ruminant host and the external environment. To emulate the cycling of E. coli O157:H7 between host and environment, a combination of shifts in temperature and starvation were used, followed by recovery using both solid and liquid media with different nutrient availability. E. coli O157:H7 survival was monitored for up to 98 days at a range of environmentally-relevant temperatures. Surprisingly, the number of potentially viable cells, i.e., cells which maintained membrane integrity, remained largely unchanged for 98 days of starvation, regardless of the holding temperature.

The real question is what proportion of the population would be in a physiological state that would allow colony formation on solid media. Interestingly, nutrient level plays an important role in the recovery of cells starved at 4°C, with recovery on nutrient-rich agar being 10,000 times higher than on nutrient-poor agar. Conversely, the recovery of cells starved at both 15°C and 25°C were less affected (less than 10-fold difference). In addition to a change in the observed number of colonies formed, there was also a change in colony size. Variation in colony size formed by cells starved at 4°C was observed, with a general decrease in colony size over the course of the 98-days starvation. This phenotypic change suggested that starvation at 4°C induced a delay in initiation of growth of E. coli O157:H7.

This change in colony size over time is dependent on available nutrients, as well as the presence of key metabolic substrates. A range of key metabolic substrates have been investigated to determine their involvement in the recovery process. Using the BIOLOG® Phenotype MicroArrayTM carbon panels, we have found that the ability of starved E. coli O157:H7 to utilise a number of carbon sources was different to that of exponential and 24-hour cultures, suggesting that specific nutrients are required for the recovery of starved cells.

In conclusion, E. coli O157:H7 can survive the temperature downshift from host to environment, as well as the transitions from nutrient-rich to nutrient-poor environments (i.e., from feast to famine). Our data suggest that temperature and nutrient availability are both important triggers used by E. coli O157:H7 to sense, respond and adapt to their surrounding environment, which in turn enable the bacteria to go on and live another day.

Ron Xavier is a third-year PhD student at the University of Waikato. His project is part of the Improved Pathogen Control Technology (IMPACT) Ministry of Science and Innovation (MSI)-funded collaboration between ESR, AgResearch and Massey University, which aims to develop new biocontrol strategies against Escherichia coli O157:H7 for the New Zealand red meat industry.

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