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NSW-ACT Branch

Syntrophy

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From the editor

By Tim Newsome

Dear all,

Welcome to your latest issue of Syntrophy, and welcome back from any Easter break you may have been lucky enough to enjoy, work permitting.

It's been a topsy-turvy couple of weeks on the COVID-19 front. We have seen some great results with vaccine rollout in curbing the spread of the pandemic in some countries (Israel stands out here), and some setbacks on the local front as our strategy shifts away from the AstraZeneca vaccine towards the Pfizer. It has been fantastic seeing prominent Australian microbiologists, virologists and immunologists communicating the science about SARS-CoV-2.

In this issue, Jai Tree (UNSW) has written a fantastic piece on the relationship between disease symptoms and the evolution of bacterial and viral pathogens, taking us on a journey from *E. coli* to SARS-CoV-2. We also have a meeting report from our annual Nancy Millis Awards Night. We made the decision, based on Zoom fatigue, to limit the number of finalists to four, which means we were treated to an embarrassment of riches in high quality talks. It is always great to get a tour of the latest science taking place across NSW and ACT. Congratulations to all the students who made the finals, and to the ASM Nancy Millis Student Travel Award 2021 recipient, and the ASM NSW-ACT Branch Student Travel Award recipient. Keep reading to find out who they are! You will be hearing from all the finalists in future Syntrophy issues throughout the course of this year.

Best wishes.

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Organisations with research opportunities, or companies seeking to fill positions are welcome to place an advertisement in an upcoming issue of Syntrophy. Please contact the Syntrophy Coordinator with your details for inclusion.

Focus Article

Why do pathogens cause disease?

Dr. Jai Tree

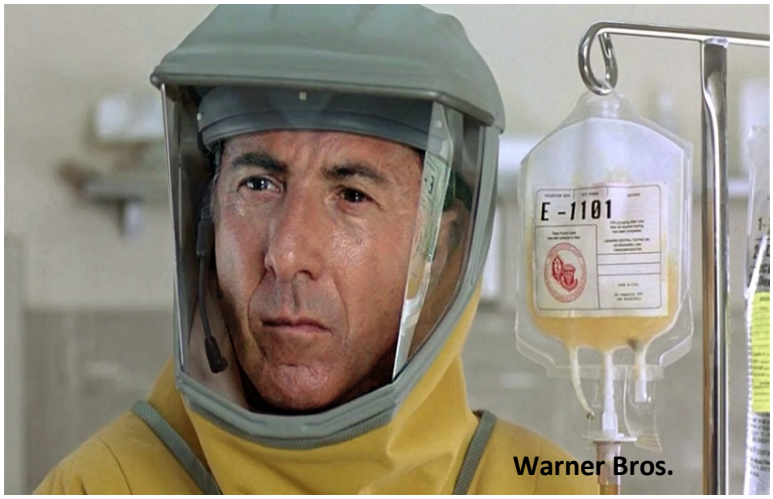
School of Biotechnology and Biomolecular Sciences, University of New South Wales

In the middle of a global pandemic, it's tempting to ask "why"? i.e. why do pathogens cause disease in humans at all, and what is the purpose of this, from the pathogen's perspective?

Where do pathogens come from?

It may be small comfort to know that it is, in fact, quite difficult to infect a human and that only a tiny fraction of microorganisms have the appropriate tools to cause disease. Of the estimated one trillion microbial species on Earth, only about 1400[1] cause disease in humans – certainly enough – but a tiny minority.

As exemplified by the emergence of SARS CoV-2 from (most likely) a population of bats, an estimated 75% of human infectious diseases originate in animals[1,2] – a process termed zoonosis. Within animal reservoirs, pathogens can be amplified, selecting for combinations of virulence traits that help them thrive in these hosts. Occasionally this selects for traits that allow them to overcome our immune defences. When this happens, the pathogen is poised for a "host jump" or "spill-over" event that may lead to the emergence of a new human disease.



Warner Bros.

Some pathogens are generalists and utilise pathways for infection that are present in a relatively wide range of host species. These pathogens, including influenza and coronaviruses, are more likely to infect a new host species, like humans. Some pathogens already have a broad host range and spill-over into the human population is quite common. For example, each year more than a million people contract food poisoning caused by Salmonella[3] that colonises livestock – particularly chickens – and enters the food chain where it infects humans.

In other cases, pathogenicity appears to be an accident, causing severe disease in humans with no appreciable benefit to the pathogen because they are only rarely transmitted from the initial person. An example of these "evolutionary accidents" is the hamburger bug, *E. coli* O157:H7. This pathogen releases a powerful toxin that causes kidney failure and neurological damage, but is mainly transmitted through food contaminated with ruminant faeces rather than person-to-person. The disease symptoms in humans have little benefit to the pathogen, and it seems the real function of the toxin may be to kill amoeba that feed on *E. coli* O157:H7 in the gut of cattle and sheep[4]. In this case, we are – sadly – collateral damage.

Given the important role that animal-to-human transmission plays in the emergence of new human diseases, there is now a strong focus on coordination between human health, animal health, agriculture, and food safety (termed One Health) to detect and stop pathogen spread and spill-over before human cases even arise. Surveillance programs that monitor influenza strains in animals and antimicrobial resistance profiling of bacteria in livestock all provide an important early warning system for the emergence of new, virulent pathogens but it is by no means a perfect system.

Is there such thing as a perfect pathogen?

Do pathogens "mellow" with age? The conceptual argument for a pathogen to become less virulent is that severe disease limits the pathogen's opportunity for transmission – most acutely seen for pathogens like Ebola with case fatality rates that are at around 50%. Better transmission is achieved if a pathogen causes mild or no symptoms allowing it to "fly under the radar" and infect more people.

While there are examples of pathogens that appear to have mellowed with age, this is not the rule and different pathogens appear to increase, decrease, and maintain disease severity after their initial host jump. This likely reflects the fact that the interaction between humans and pathogens is multifaceted and pathogens have found a variety of solutions to the problem of subverting our defences and transmitting to a new person. This is exemplified by the viruses myxomatosis and rabbit haemorrhagic fever disease that were released in Australia to control the rabbit population. The mortality rate of myxomatosis initially declined in the years after release whereas the virulence of rabbit haemorrhagic fever appears to have increased. The difference is likely linked to different modes of transmission between these viruses (rabbit haemorrhagic fever is transmitted from carrion by flies) and the changes in rabbit resistance to the diseases.


Universal pictures

For SARS CoV-2 it seems that some of the more transmissible variants that have recently emerged are also associated with higher mortality (eg: B.1.1.7)[5,6]. Early evidence to support the virus becoming less virulent was likely confounded by increased efficiencies in healthcare and changing demographics of those infected during 2020.

Why do pathogens cause disease symptoms?

The measure of a pathogen's success is its ability to grow in its host, and spread to as many hosts as possible. Some disease symptoms are a by-product of tissue damage caused by pathogen growth, or our own over-exuberant immune responses to the pathogen. These responses can be extremely advantageous for the pathogen – for respiratory viruses like SARS CoV-2, inflammation in the nose and airways induces sneezing and coughing that are important for pathogen spread. Wearing a mask significantly reduces the pathogen's advantage.

During infection with Salmonella (one of the most common causes of food poisoning), inflammation in the intestine clears away our protective normal flora and reduces competition. Pathogens can also actively induce disease symptoms, for example the bacterium that causes cholera induces diarrhoea using a toxin – a key survival tactic for a pathogen that spreads through faecal-oral transmission.

The purposes of other disease symptoms are less clear and may be the unfortunate consequence of an interaction that is often blind to events that occur on the macroscopic scale. For instance, patients infected with *Mycobacterium leprae*, the causative agent of leprosy, lose feeling in their extremities. How the bacterium would benefit from this is unclear and may simply be a terrible corollary of the fact that our nerve cells are (unfortunately) good places to hide from our immune system.

Winning the war on disease

Beyond the COVID-19 pandemic we face continued challenges from pathogenic microorganisms. Key among these is the emergence of antimicrobial resistance, a slower burning pandemic that threatens to nullify some of our most effective tools for combating infectious disease. If nothing else, 2020 has taught us that our ability to effectively combat disease underpins our way of life. Given the enormous human and financial toll that the pandemic has taken on us all, it will hopefully re-invigorate national and global interest in retaining and expanding our arsenal of effective vaccines and antimicrobials.

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About the author

Jai Tree is a Senior Lecturer in Microbiology at the University of New South Wales. He completed his PhD at the University of Queensland and undertook post-doctoral studies at the University of Edinburgh, UK and University of Melbourne. His research aims to understand the molecular pathways that control disease progression in bacterial pathogens. A major focus of the lab is to understand how regulatory non-coding RNAs control and co-ordinate expression of genes that are required for pathogenesis and antibiotic resistance. He is also enthusiastic about undergraduate learning and teaches Microbiology, Molecular Biology, and Bacterial Genetics. Website: treelab.science Twitter: @jaitreeau

ASM Nancy Millis Travel Awards night

By Mohammad Hamidian and Nick Coleman

The ASM Nancy Millis Travel Awards night was held via Zoom on March 24, and was attended by a total of 35 academics and HDR students from Universities across NSW and ACT. Yet again, highlights of this year's competition were the extremely quality of abstracts and the large number of applications received (14 abstracts) by the ASM NSW-ACT branch, making the task of choosing the finalists to speak on the night very difficult. All finalists received a $250 cash prize.

Five applications for the ASM Nancy Millis Travel Award were received from the University of New South Wales (UNSW), five from the University of Technology Sydney (UTS), one from the University of Sydney, one from Australian National University, one from the University of Wollongong and one from Southern Cross University. Three independent examiners carefully reviewed all applications, and four finalists were selected to present their work at the awards night.

The talks given by the four finalists, Winton Wu (UNSW), Ahmed Mohammad (UTS), Mozammel Hoque (UTS), and Elizabeth Peterson (UTS), highlighted both the diversity and the high quality of microbiology research being undertaken by local microbiology PhD students. Each talk was followed by a Q&A session, following which the three judges, Prof Ruiting Lan (UNSW), A/Prof Willa Huston (UTS) and A/Prof Sally Partridge (The Westmead Institute for Medical Research), carefully examined and ranked the talks.

The ASM Nancy Millis Student Travel Award 2021 went to **Elizabeth Peterson (UTS)**, for her talk "*Shape-shifting bacteria are key to infection*". **Winton Wu (UNSW)** was the winner of the ASM NSW-ACT Branch Student Travel Award for his talk "*Uncovering the small RNA interactome and its role in antibiotic tolerance in* Staphylococcus aureus". Elizabeth will present her talk at a special symposium at the ASM 2021 annual meeting, and both Elizabeth and Winton have the registration, accomodation and travel costs for this meeting reinbursed. Elizabeth and Winton are highlighted in green frames in the screenshot below. All finalists received a $250 cash prize.

The ASM NSW/ACT Branch Committee thanks all the award applicants for their hard word in preparing their abstracts and also thanks everyone who made time to attend the session. Hopefully in 2022, we will return to a live talks format.