

Is flu stronger, or are we weaker?

Scott Ewan, BMN News

What is it about the recent deadly flu infection, Fujian flu, which makes



it so virulent and dangerous?

'We don't really know,' said Wendy Barclay, a lecturer in microbiology at the University of Reading (<http://www.reading.ac.uk>), 'but there are two possibilities.' If Fujian flu is truly more virulent, it could be that the virus has changed in some way.

What is different, the virus or man?

However, many virologists do not believe that Fujian flu is unusually virulent. It is feasible that the patients' themselves are different somehow, and are more susceptible to the effects of the infection.

Barclay continued, 'The number of deaths with this strain so far is still small.' Every year there are unexplained deaths with whatever strain is prevalent, and this year is no different.

'We shouldn't be alarmed,' said John Oxford at Queen Mary's School of Medicine in London, UK (<http://www.smd.qmul.ac.uk/>), who is also Scientific Director of the virological research and clinical trials company, Retroscreen Virology (<http://www.retroscreen.com/>). We have 'background' immunity against flu, which was developed over years of living alongside the disease and provides some protection against infection or related complications.

'There's not been much flu over the past few years,' said Oxford. This unusual coincidence means that the very young may not have had the chance to develop their background

immunity and could have resulted in the higher incidence of flu-related deaths this year in young children.

Antigen mutations

Indeed, it is known that the Fujian flu currently emerging in the UK, northern Europe and North America, is essentially the same as the prevailing flu observed for the past 30 years. The flu virus undergoes antigenic drift – mutating slightly to ensure its survival against immunity. These mutations take the form of simple changes to the amino acid sequences of two antigens on the viral surface. These antigens – haemagglutinin and neuraminidase – control the virus' entry and exit from the host cell. The antigens are the points at which drug development has focused.

The two modern drugs currently on the market – GlaxoSmithKline's Relenza and Roche's Tamiflu – both inhibit neuraminidase's normal function of releasing the virus from the host cell.

Oxford remains concerned however. 'We've still not taken on board the

WHO's [World Health Organization; <http://www.who.org>] recommendation that we should be prepared for a pandemic.' We have not coped well with Fujian 'flu, he says, which is essentially a 'normal' flu. What will happen, he wonders, when the next true pandemic occurs?

Vaccines and drugs are available to us to combat flu – but economics, logistics and lack of public knowledge are hindering their effective deployment. With an estimated 69 million workdays lost to flu in the USA each year, and at an estimated cost of over US\$14 billion, it is almost inconceivable that the best use of these agents is not being made.

New therapies

Research is still ongoing into developing new therapies. Oxford envisages a future where a single treatment might be effective against a range of respiratory tract infections – including flu. 'By identifying common responses to different viruses, it may be possible to block the pathway, preventing spread of a number of infections,' he predicted.

Mouse transcripts inform on human splice sites

Henry Nicholls, BMN News

Comparison of data from the mouse and human genome projects predicts the existence of a multitude of splice sites that analysis of the human genome alone has failed to detect.

Specific splice sites

Databases of human transcripts reveal evidence for hundreds of thousands of splice sites, specific places where RNA is edited following its transcription from