

NUMBER CRUNCH

33% is the proportion of Americans following science and technology news "very closely" during 1986–89, according to the snappily titled Pew News Interest Index.

16% is the corresponding figure for 2000–06.

17% is the number of news junkies with a keen interest in celebrity scandals, showing that the world of science is now measurably less popular than Paris Hilton, though perhaps not as much as Sidelines might have thought.

ZOO NEWS**Bridge birds**

A build-up of corrosive pigeon guano on the support struts of the bridge in Minneapolis that collapsed on 1 August might have played a role in the disaster. Some experts think that ammonia in the droppings could have weakened the steel beams.



J. INGLIS/ALAMY

WORDWATCH**Slime-Snake-Monkey-People**

Evangelist Robert Bowie Johnson has coined this term for darwinists. He suggests that Christians should use it to 'shame' those who accept evolution over Genesis. Talk us through the snake-to-monkey step, would you, Bob?

SCORECARD**Chewing gum**

Archaeologists at Harvard University have obtained 2,000-year-old DNA from wads of plant matter that Native Americans apparently used as chewing gum.

**Chewing gum**

Meanwhile, the oldest-ever 'chewing gum' has been found in Finland. The 5,000-year-old morsel, complete with toothmarks, was a distinctly unappetizing lump of birch bark tar.

Sources: Pew Research Center, Associated Press, Christian News Wire, Science, The Guardian

Cheaper approaches to flu divide researchers

As governments race to stockpile Tamiflu and other antivirals in preparation for the next influenza pandemic, scientists are starting to look at a different approach — drugs that modulate the immune system.

Proponents argue that these abundant and cheaper drugs, such as statins, might be a useful option where antivirals are unavailable or have been given too late. But sceptics fear that they might do more harm than good because they suppress the body's immune response to the virus. With the latest studies seeming to contradict each other, the flu community is divided about the best way to proceed.

The high mortality associated with viruses such as avian flu is thought to be because the immune system has a massive over-reaction to the infection. The body hikes its production of immune molecules called cytokines, which flood into the bloodstream, causing what's known as a cytokine storm. This triggers inflammation and lung damage, and can lead to multiple organ failure and death. So rather than the usual approach of targeting the virus, researchers hope that mortality could be reduced by using drugs that regulate this immune response.

At present, planning for pandemics is focused on stockpiling antivirals. But because these drugs, such as Tamiflu, are expensive and difficult to manufacture, there may not be enough to go round by the time another pandemic strikes, and poorer countries may not have access to them at all.

"If we are to be serious about confronting a pandemic, we must urgently evaluate agents that are already available and affordable to people in all countries," says David Fedson, former director of medical affairs at Paris-based Aventis Pasteur, now Sanofi-Aventis.

This month, Ian Clark's group at the Australian National University in Canberra published a study¹ investigating the effectiveness of another immune-modulating drug, called gemfibrozil, in mice infected with the influenza virus. Gemfibrozil is a fibrate, a class of drug widely prescribed to lower lipid concentrations

in the blood. But it also inhibits the release of inflammatory cytokines such as tumour-necrosis factor, interferon- γ and interleukin-6, and stimulates production of the anti-inflammatory cytokine interleukin-4. Clark's team infected mice with H2N2 — the flu strain that caused the 1957 pandemic. Four days later, once the rodents had become sick, they injected 46 mice with 60 milligrams per kilogram of body weight of gemfibrozil once a day for six days. Twice as many of the treated mice (52%) survived compared with controls (26%).

Tread with caution

"The paper should spark some interest, but even more caution," says Erik De Clercq, a virologist at the Catholic University of Leuven in Belgium. He points out that it is difficult to

extrapolate from Clark's H2N2 mouse model to the H5N1 strain in humans. For example, he says, it did not show that increased survival was directly due to inhibition of the cytokine storm. Clark agrees and is planning further studies to rule out the possibility of confounding effects, such as the drug having a direct antiviral effect.

However, two other studies suggest that the cytokine storm might not be as fatal as thought. In one study², mice that had one cytokine pathway knocked out showed no reduction in mortality compared with normal mice after they were infected with H5N1. "These results refute the

popular paradigm that the cytokine storm is the cause of death during H5N1 infection," says Robert Webster, who carried out the study with his colleagues at St Jude Children's Research Hospital in Memphis, Tennessee.

In the second study³, by Jacqueline Katz's group at the Centers for Disease Control and Prevention in Atlanta, Georgia, mice deficient in the inflammatory cytokine interleukin-1 receptor had worse mortality, higher viral loads and more inflammation than did controls when infected with the HK/486 virus.

But both studies tested only three to five mice in each experiment, and looked at only a few of the many overlapping cytokine path-



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