New Directions in Plastic Debris

THE LARGEST EVER MEETING FOCUSING ON plastic debris in the environment was recently held in Redondo Beach, California (*1*). It is evident that plastic waste presents major concerns in aquatic habitats worldwide. However, this meeting differed from previous efforts/gatherings because representatives from industry, government, academia, and nongovernment organizations were united in their desire to identify solutions to reducing waste. There has been a switch in the types of litter recorded, from shipping- and fishing-related debris to land-based sources. This was poignantly underscored by reports of islands of plastic debris swept into the sea by Hurricane Katrina.

Polymer scientist A. Andrady explained that all the plastic introduced into the oceans remains unmineralized as either entire objects or fragments, some of which are less than 20 μm in diameter (*2*). Large items of debris cause entanglement, impaired feeding, and mortality to birds, turtles, and mammals. Microscopic fragments are also ingested, but the consequences are unknown. H. Takada and C. Moore presented evidence on the ability of plastic to accumulate PCBs, DDE, and nonylphenol (*3*), and the potential for toxic chemicals to transfer to the food chain was identified as a key research direction. It was also recognized that better understanding of effects at an organismal level is required before consequences at population and ecosystem levels can be examined.

In terms of solutions, much could be achieved by reductions in packaging. Keynote speaker W. McDonough made the case for a "cradle to cradle" (*4*) strategy to ensure that plastics are retained in a product-specific recycling loop—turning debris from a waste disposal liability into feedstock for production. Although debris can be removed from drains and rivers by physical separators, there is also a key role for education to help reduce littering. The importance of social research to establish the public's willingness to engage with these solutions was also clearly recognized.

RICHARD THOMPSON,[1]* CHARLES MOORE,[2] ANTHONY ANDRADY,[3] MURRAY GREGORY,[4] HIDESHIGE TAKADA,[5] STEPHEN WEISBERG[6]

[1]School of Biological Sciences, University of Plymouth, Plymouth, Devon PL4 8AA, UK. [2]Algalita Marine Research Foundation, 148 North Marina Drive, Long Beach, CA 90803, USA. [3]Research Triangle Institute, 3040 Cornwallis Road, Research Triangle Park, NC 27709–2194, USA. [4]University of Auckland, Private Bag 92019, Auckland, New Zealand. [5]Tokyo University of Agriculture and Technology, Tokyo 183 8509, Japan. [6]Southern California Coastal Research Project, 7171 Fenwick Lane, Westminster, CA 92683, USA.

*To whom correspondence should be addressed. E-mail: r.c.thompson@plymouth.ac.uk

CREDIT: DAVID QUINN/AP PHOTO

Toy cars amid debris in New Orleans after Hurricane Katrina.

References

1. Plastic Debris Rivers to Seas, organized by the California Coastal Commission, 7 to 9 Sept 2005.
2. R. C. Thompson *et al.*, *Science* **304**, 838 (2004).
3. Y. Mato *et al.*, *Environ. Sci. Technol.* **35**, 318 (2001).
4. W. McDonough, M. Braungart, *Cradle to Cradle* (North Point Press, New York, 2002).

Preparing for the Worst-Case Scenario

THE REPORT "CONTAINING PANDEMIC influenza at the source" by I. M. Longini Jr. *et al.* (12 Aug., p. 1083) was encouraging that an avian pandemic can be contained if proper intervention is carried out promptly. N. M. Ferguson *et al.* published similar findings (*1*). However, further investigation is needed before we can celebrate.

A valid conclusion from a model requires a careful selection of the parameter values. Longini's article assumed that Tamiflu (oseltamivir) was useful in a pandemic. Yet, Tamiflu may not be effective on all new avian flu viruses, which can have 80% mortality; Tamiflu was ineffective in 50% of patients in Thailand (*2*). Moreover, although the basic reproduction number (R_0) below 2 was a reasonable estimation in Longini's models, previous flu pandemics have had an R_0 up to 3 (*3*). Apart from these technical parameters in a hypothetical model, the logistics in a real situation can also be fluid. Although developing countries with pharmaceutical factories can issue a compulsory license to make generic copies of patented drugs in the event of a medical emergency, in reality when there is a pandemic occurring, developing areas without such facilities (perhaps those that most need Tamiflu) will be in a difficult position to secure enough supply.

Handling the ever-changing disease pattern of pandemic avian flu requires a contingency plan to prepare for the worst scenario. A worst-case scenario model should predict the resources a public health system needs to cope with a pandemic. This model should consider that Tamiflu may be ineffective in treating the new strain, a higher-than-expected R_0 value, and the possibility of a paucity of antiviral drug/vaccines. Such a worst-case-scenario model provides valuable information for resource planning, for example, the number of ventilators, the amount of intensive care, and even funeral facilities that will be required.

PUI HONG ALEX CHUNG

Higher Specialist Trainee in Community Medicine, 10th floor, 147C Argyle Street, Mong Kok, Hong Kong SAR, China.

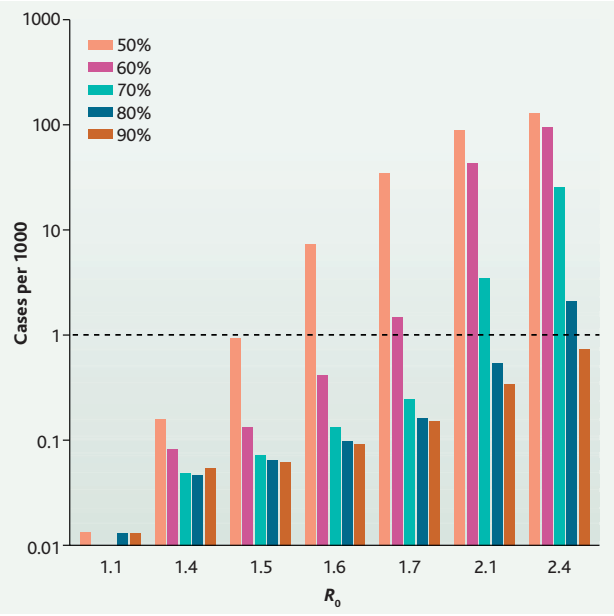
References

1. N. M. Ferguson *et al.*, *Nature* **437**, 209 (2005).
2. T. H. Tran *et al.*, *N. Engl. J. Med.* **350**, 1179 (2004).
3. B. Cooper *et al.*, poster presented at Influenza Vaccines for the World, Lisbon, 24 to 26 May 2005.

Response

CONTAINING A POTENTIAL STRAIN OF PANdemic influenza at the source will not be easy and will require intense surveillance of influenza-like illness, speedy lab work to identify isolated viruses, and feasible and effective control strategies. We showed that the targeted use of oseltamivir could contain a potential pandemic strain of influenza if the basic reproductive number (R_0) were below 1.6 and if the intervention took place 2 to 3 weeks after the first case appeared. If the efficacy of oseltamivir against the

Published by AAAS



The effectiveness of the household and neighborhood cluster quarantine started 14 days after the first case at different values of R_0. Outbreaks that result in a cumulative incidence of ≤ 1 case per 1000 are considered contained (horizontal line). The cumulative incidence per 1000 does not always decrease monotonically with increasing intervention coverage for small differences due to stochastic variability.

emergent virus were as low as 50% against illness and transmission, the targeted use of oseltamivir could still effectively contain the spread (figs. S16 to S18). For a situation in which oseltamivir is ineffective against the emergent strain, we modeled the use of voluntary household and neighborhood cluster (i.e., one small grouping of about four households) quarantine alone (figs. 3 and S13). For this intervention, the first case in a locality (i.e., a region roughly 6 to 9 km in radius) triggers a quarantine policy. Every case and a certain percentage of susceptible people restrict their movement to their household and their neighborhood cluster. The figure gives the average simulated cumulative influenza case incidence for different levels of quarantine effectiveness for different values of R_0. For $R_0 \leq 1.5$, quarantine levels as low as 50% of people being restricted to the household and neighborhood cluster could possibly contain outbreaks. For $R_0 \leq 1.6$, quarantine levels of 60% or higher could be effective. With an R_0 as high as 2.4, we would need a 90% effective quarantine for containment. For larger values of R_0, containment would not be possible with quarantine measures alone. If we consider an R_0 of 2.4 and total viral resistance to oseltamivir to be the worst-case scenario, then only an extremely tight household and neighborhood cluster quarantine could effectively contain the spread of the virus. Other forms of quarantine also could be effective (*1*). Our results are highly probabilistic, and a strategy that works in many simulations does not necessarily work in all simulations. Thus, even if countries are prepared to try to contain the outbreak, the world needs to be prepared for the event that containment fails.

It is important to develop international cooperation around the maintenance of a mobile stockpile of oseltamivir that can be rapidly deployed to the location of an emergent strain. The World Health Organization (WHO) is currently developing such a stockpile and recently announced that Roche is donating 3 million courses of oseltamivir to its mobile stockpile (*2*). If a potential pandemic strain of influenza emerges, WHO plans to intervene, in concert with the ministry of health of the affected country and other public health organizations, along the lines that we describe above [see index 1 of (*2*)]. Such international cooperation coupled with a rapid, effective response will be necessary to have any hope of containing pandemic influenza before it spreads throughout the world, or at least slowing the spread while a well-matched vaccine is developed and sufficient quantities are deployed.

IRA M. LONGINI JR. AND M. ELIZABETH HALLORAN
Department of Biostatistics, The Rollins School of Public Health, Emory University, 1518 Clifton Road, NE, Atlanta, GA 30322, USA.

References
1. N. M. Ferguson *et al.*, *Nature* **437**, 209 (2005).
2. "Donation of three million treatments of oseltamivir to WHO will help early response to an emerging influenza pandemic" (available at www.who.int/mediacentre/news/releases/2005/pr36/en/), accessed 30 Aug. 2005.

Timing in Collection of Stool Samples

WE READ WITH GREAT INTEREST THE REPORT "Diversity of the human intestinal microbial flora" by P. B. Eckburg *et al.* (10 June, p. 1635; published online 14 Apr.). We applaud the authors for advancing this important field by undertaking comprehensive 16S rDNA sequencing to describe the composition of the microbiota in stools and at six sites of the colon in each of three human volunteers. The analyzed 13,355 prokaryotic ribosomal RNA gene sequences represent the largest 16S rDNA microflora data set reported to date in any species.

On the basis of their analysis, the authors suggest that "differences between stool and mucosa community composition" exist. We question the validity of this conclusion, based on our own observation that stool microflora composition can vary significantly in stool samples collected before and after a colonoscopy (*1*). The authors compare microflora composition in colon biopsy samples obtained during colonoscopy with a stool sample collected a month afterwards. The authors acknowledge potential problems with their interpretation because of the delayed stool collection, but a rationale for collecting delayed stool samples is not given.

This Report has significantly expanded our knowledge of the diversity of the intestinal microflora in a few subjects. However, if we ever want to correlate microflora composition with health or disease, we will have to design studies aimed at understanding the variation in the microflora composition in a large cohort of human subjects.

VOLKER MAI,* O. COLIN STINE, J. GLENN MORRIS JR.
Department of Epidemiology and Preventive Medicine, School of Medicine, University of Maryland, Baltimore, MD 21201, USA.
*To whom correspondence should be addressed. E-mail: vmai@epi.umaryland.edu

Reference
1. V. Mai, O. C. Stine, J. G. Morris Jr., unpublished data.

Response

OUR LARGE-SCALE COMPARATIVE ANALYSIS of bacterial and archaeal 16S rDNA sequences in the colon and stool revealed significant intersubject variability and patchy heterogeneity among the colonic mucosal bacterial populations. Regarding the statistical differences we reported between stool and adherent mucosal populations, subjects B and C harbored different bacterial populations in their colonic mucosa compared with their stool samples collected 4 weeks later, while the mucosal populations in subject A were subsets of the population observed in stool collected 4 weeks after colonoscopy. Each subject's stool community was more similar to the communities of their own mucosal samples than to any community from a different subject.

We acknowledged that the statistically significant difference between the bacterial composition of the stool and colonic mucosa may have been due to the 4-week delay in stool collection after colonoscopy. The collection of stool was not originally planned in the large Canadian population-based case control study from which the control subjects were selected. For this