SCIENCE'S COMPASS



Penny Wise, Pound Foolish— A Retrospective

The News of the Week article about an independent panel's report on bovine spongiform encephalopathy (BSE), usually called mad cow disease, lays blame disproportionately on "incorrect assumption[s] by a scientific panel" for which "the best people [were not] recruited to give advice" ("Report flags hazards

of risk assessment" by Helen Gavaghan, 3 Nov., p. 911). As one of those BSE advisory scientists in a 1989 meeting, I know that the scientists who were recruited represented all the major laboratories engaged in transmission experiments, in addition to other respected neurovirologists. From my perspective, unwelcome scientific advice about an epidemic spread of BSE worldwide, and especially about the undeniable possibility of transmission of the BSE agent to humans, was dismissed. During that meeting, I proposed killing all cows on farms where infection had been found. Other scientists considered that removal of bovine offal from the mammalian (or at least

cow) food chain would be sufficient.

The reasons for not initiating a kill of "healthy" cows, and for implementing the offal ban only half-heartedly, were economic rather than scientific. Even the payment of farmers for sick (or almost sick) cows was to be half their market price. As many of us warned, this policy would encourage farmers to rapidly push suspect cows to market for full price. Commercial economic interests were also pivotal for the British government.

Scientific opinion was also captured in the process. It was convenient, for example, for policy-makers to work under the assumption that the "species barrier" was impermeable. An argument based on the dominant prion theory presumed that host prion protein differences would control transmission across species. The lesson here is the risk of ignoring powerful data when they contradict a popular theory. It was repeatedly shown, since the 1960s, that both scrapie and variant Creutzfeldt-Jakob disease agents

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can silently infect many distant species, with disease becoming evident if one waits long enough. Thus, there was a clear risk of cross-species BSE transmission, particularly in long-lived species such as humans. Prion theory at that time also did not acknowledge distinctive agent strains. Because such strains could change with passage, it was obvious that a more virulent agent for humans could be selected after passage in



A recent report examines how the British government handled the mad cow disease outbreak.

cows (1). Given this history, it is unsettling that Lord Andrew Phillips wants a "research 'supremo" to cull scientific advice.

Another lesson was the risk of secrecy. All scientists had to promise not to reveal new BSE data discussed, including export of cows and contaminated feed worldwide and recognition that more than 1000 farms had infected cows. Nevertheless, I felt obligated as a physician to inform the U.S. Department of Agriculture (USDA) upon my return to the United States and strongly urged my USDA contact to ban further imports of feed. The USDA acted promptly, and the United States still has had no evidence of the U.K. BSE agent in its cattle. The promise of confidentiality was, however, detrimental for public disclosures. I did not speak about these matters in scientific lectures, and I felt obliged to wait until 1994 to publish my concerns that BSE could be transmitted to people (2). This same article also warned of associated problems from contaminated medical products, an issue now being rediscovered.

• LETTERS

Because BSE transmission to humans and other species has become a public reality since 1996, open scientific discourse and policy deliberations are critical. The conclusions by the current independent panel that BSE was "unavoidable" and that it "may have emerged...from a genetic mutation that went unnoticed in a single cow" are surely open to question. Moreover, narrow public instruction in "spontaneous generation" of an "infectious protein" can delay important initiatives and preventive measures. Stopping the ongoing spread of BSE should be of common concern, theories notwithstanding. Many economically disadvantaged countries have received contaminated U.K. feed and cattle, and only informing them about the real risks and precautions and supplying necessary aid will limit the further penetration of BSE worldwide.

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Missing Persons Identification: Genetics at Work for Society

The genetic molecular tools that have been applied to the forensic field have had a beneficial impact, including the exoneration of innocent people, identification of offenders, and establishment of criminal databases. However, regardless of the anonymous nature of DNA profile data and the security measures in place, misuse of the data and mishandling of samples are possible threats to social and individual rights, as Andrew Watson discusses in his News Focus article "A new breed of high-tech detectives" (11 Aug., p. 850). But one application that does not compromise social or personal rights offers a valuable tool for the identification of missing persons and human remains (1).

In 1999, Spain started a national program to try to identify cadavers and human remains that could not be identified with traditional forensic approaches. Named for the classical Greek myth, the Phoenix Program is based on the fact that mitochondria, which are present in every cell and have their own mitochondrial DNA (mtDNA), are inherited strictly from the mother. Two mtDNA databases are being generated: a reference database, which contains mtDNA sequences from maternal relatives of missing persons who provide the samples (buccal swabs) voluntarily after signing an informed consent form, and a questioned database, which contains mtDNA data from unknown human remains and cadavers. These data can be automatically compared to search for identical or similar mtDNA sequences (2). Although the first phase of this program

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(typing of all unidentified human remains) will not be completed until December 2001, positive identifications have already been made. International collaboration in this endeavor-the generation of internationally compatible databases-could make this effort valuable worldwide.

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Eurasian Air Pollution Reaches Eastern North America

Pollutants of Eurasian origin cross the Pacific Ocean via the westerly winds and affect the west coast of North America, as Kenneth E. Wilkening, Leonard A. Barrie, and Marilyn Engle discuss in their Perspective (Science's Compass, 6 Oct., p. 65). However, neither anthropogenic pollutants nor natural dusts of trans-Pacific origin stop their transit at or near the western North American coast.

We have found that the continental dust in the Greenland Summit ice cores from 1500 to 44,000 years before the present, and probably beyond, is of Eastern Asian provenance (1). We are finding the same sources in recent (past decade) dust extracted from snow pits at the North Greenland Ice core Project site (75°N 43°W), including dust from the April 1998 outbreak shown in the

figure in Wilkening et al.'s Perspective (see the figure) (2). The fluxes of dust to Greenland have been generally about a few millligrams per square centimeter per thousand years, compared with fluxes about an order of magnitude greater upwind in the eastern North Pacific (3). Consequently, we expect that the decrease in the flux of dust and as-

sociated pollutants between western and eastern North America might be less than that factor of ~10 and therefore of significance to levels of pollution.

As Asian economic expansion increases the quantities of pollutants injected into the westerly winds, their levels in western



Satellite remote sensing images show how wind currents distribute dusts and pollutants on a global scale.

> North American cities, like those cited by Wilkening et al., will also rise, as will those levels eastward and all across the continent.

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