Editorials

Dioxins in Belgian feed and food: chickens and eggs

The scare over dioxin contamination of Belgian food in May to June 1999 erupted when European consumer confidence in food was particularly low. Both public alarm and official response were intense. The European Union banned sales of Belgian poultry, eggs, fatty beef, pork, butter, and all byproduct foods. In terms of the actual risks to consumers from this particular increment in dose, the response was almost certainly excessive. Nevertheless, as risk experts remind us, the perceived risk reflects both the assumed actual hazard and the attendant level of outrage. There was great public outrage because of the toxic notoriety of dioxins, the invisibility of the hazard, and the sense that officialdom had conspired against consumer interests.

Meanwhile, beyond the immediate drama is an important chicken or egg type of question. What was the underlying cause: poor commercial technology, misguided governmental priorities, or the intensification of food production for a mass consumer market? The public debate over the dioxin episode reflected concerns over each of these issues. In particular, the succession of recent food scares has engendered a growing perception that human interventions in nature—whether via intensified food production, genetic engineering, or excessive fossil fuel combustion—seem to make things go wrong. We do not merely create transitory hazards; we disrupt our habitat.¹

An additional anxiety is that the human health effects of dioxin are not obvious. At least with *E coli* 0157, salmonella food poisoning and Malaysia's Nipah virus, the human health consequences are evident—infected people get very ill or die. Health outcomes were also overt with earlier toxicological food disasters, especially the Spanish cooking oil episode of 1981, which killed 400 people and left many thousands ill from exposure to adulterated rapeseed oil,² and the 1985 episode when anti-freeze chemicals were illegally added to Austrian and Italian wines. Thus, the consequences of many such infectious and toxicological events can be tabulated. But with dioxin, how will we know who has been affected and when the excess risk has been dissipated? Although the estimated dioxin exposure levels exceeded the WHO standards by two orders of magnitude, no acute health effect in humans was expected or observed.

Europe's other recent spectacular public health food crisis, bovine spongiform encephalopathy (or "mad cow disease"), has been somewhat intermediate to these two contrasting situations. The resultant human neurological disorder, variant CJD, has been fairly well characterised clinically and pathologically, and compelling epidemiological, animal experimental and molecular biological evidence of a causal relation has now accrued. However, the course of the attributable epidemic of variant CJD remains incomplete and uncertain.

Note that each of these episodes surfaced because of some observable adverse effect in humans or in the animals they consume. In an era of intensive food production, long distance transport, and an urban culture of fast food and convenience foods, more thorough surveillance of food quality is needed. Consumer organisations increasingly want to know what lies below the iceberg's water line. Are we subject to some form of "baseline drift" as background levels of certain contaminants in food increase? In fact, levels of dioxins in food have declined in several European

countries in recent years, as mirrored in falling levels in breast milk—such as the 65% fall in Germany between 1989 and 1997.³ Even so, against the backdrop of several decades of accrued uptake by humans from trace exposures to dioxins in foods in modern industrial urban society, the estimated average increment in dioxin uptake because of this recent episode is actually quite small. (Interestingly, the preoccupation with this acute exposure episode resembles the prevailing approach of epidemiologists to air pollution and health: most attention has been focused on the acute health consequences of short-term fluctuations, with relative neglect of the cumulative consequences of long term exposure to increased air pollutant levels.⁴)

The dioxins comprise a family of over 100 closely related polychlorinated chemicals: the dibenzodioxins (PCDDs), dibenzofurans (PCDFs) and biphenyls (PCBs). A minority of these, and especially 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), are highly toxic, causing dermal toxicity, reproductive impairment, disturbance of fetal-neonatal immunological and neurological development, endocrine disruption and, probably, teratogenicity and carcinogenicity.⁵ It is those black sheep family members that have given "dioxin" its sinister image. Dioxin is absorbed through skin or gut; an estimated 90% of human exposure to dioxin is through food. It bioaccumulates in fat tissue. Dioxin is a member of the POPs club: the persistent organic pollutants.

Dioxins rarely occur in nature, other than via volcanic eruptions and forest fires. Trace amounts form as byproducts of smelting, paper-pulp bleaching and pesticide manufacture, from the manufacture of PCBs and other chlorinated chemicals, and, in particular, when chlorinated compounds are incinerated at high temperature. They are ubiquitous in the environment. They have long been suspected of causing cancer.⁵ The latest follow up data from occupationally exposed cohorts suggest that cancer risks might be generally but modestly increased—although, with the exception of an increased lung cancer risk at the highest exposure level, there are no clearcut findings for specific cancers.⁶ The International Agency for Research on Cancer (WHO) has recently classified dioxin as a "known cancer-causing compound".⁷

Dioxin gained notoriety during the Vietnam war, when suspicion fell on it as a potent toxic contaminant in the "Agent Orange" herbicide, widely sprayed over that benighted countryside, its people and animals. Debate continues about its role in congenital abnormalities in Vietnam, where environmental exposures persist. The Seveso disaster in Italy in 1976, when a chemical factory exploded, acutely exposed the local community to unprecedentedly high levels of dioxin. The first observed outcome was toxicity in small domestic animals. Painful and disfiguring chloracne soon appeared in several hundred people, although there was little other evidence of permanent organ damage.⁸ Two years after Seveso, in upstate New York, USA, dioxin was found in a dumping ground, Love Canal, which—despite the absence of clear evidence of human health detriment—became a cordoned off ghost town.

Now, in 1999, we face another dioxin scare, in Belgian food. This problem was hugely compounded by the

misguided behaviour of officialdom. The dioxin contamination of animal feed was apparently known by agricultural authorities for several months before the health ministry was alerted. The original episode, in mid-January, entailed the contamination of commercial feed preparation process, apparently by dioxin contaminated waste industrial oils. Impaired egg laying and neural disorders were soon noticed in chickens. By April, officials knew that dioxin level in chickens were extremely high with some measurements being of the order of 700 pg/g of lipid, resulting in body burdens of around 50 ng per chicken.³ Farm ministry staff worried about the effects on food trade—but not about consumer safety. In late May the hazard to humans was acknowledged, and the public scandal broke.

This narrative mirrored the primacy given to producer interests in Britain's mad cow episode, at the expense of consumer interests. By the time the reprehensible behaviour of Belgian officialdom came to light, over 1000 Belgian farms had been endangered by contaminated animal feed. Several hundred other farms in the Netherlands, France and Germany also came under surveillance as authorities struggled to trace tainted products. The EU ordered the destruction of millions of pounds of chicken and egg products exported from Belgium after 15 January. But by then the damage was done. The USA announced an immediate ban on all meat and dairy products from Europe. Various European countries banned imported Belgian meat products. Many other countries introduced partial bans—Middle Eastern countries, Singapore, South Korea, Canada. Meanwhile, distressed Belgian shoppers crossed into France, Germany and England to seek untainted food for their weekend dinners.

The immediate political and toxicological dramas were, of course, fascinating. The dioxin crisis coincided with the dispute between the USA and Europe over the latter's refusal to accept hormone treated beef imports from US producers. The USA claimed this was a breach of international trade rules. Europe argued that it was protecting the public's health against carcinogenic and other hazards (which, conveniently, also enabled the continued protection of its own less efficient beef producers). Meanwhile, there were widespread consumer fears of poisoning, cancer and birth defects. Although the risks of cancer reproductive disorders and neural damage from dioxin traces in food are quite low, WHO's recent quantitative risk assessment of dioxin suggests there are particular health risks to human infants.⁹ Furthermore, such exposure episodes contribute to the wider build up of chemical burdens in human bodies, from diverse sources in the modern industrial world. Those burdens may have adverse subclinical effects on neurological, immune and reproductive systems.

There is, though, a more profound dimension to this episode, arising out of modern human economic practices and our relationships with the natural world.¹⁰ As urban consumerism grows, as deregulated markets respond to competition, as trade networks globalise, and as national governments seek competitive advantage in a free market global economy, so food health crises tend to multiply. In particular, the emergence or spread of infectious agents in the human food chain is facilitated.^{11 12} Many of our food production methods have deviated from natural processes. There is a heightened discontinuity between food production and consumption: food has been progressively commodified.¹³ Today, food is principally grown, processed and marketed for profit by large, often transnational, agribusiness corporations, and it is often produced by poorer countries as export cash crops to lessen national debt. The public health ramifications are legion. This line of analysis

connects with the current debate over genetically modified foods.¹⁴ Whose interests are really being served? How should we assess the risks to consumers? Indeed, how can we get off the population/consumption escalator, as compared with trying to run up it ever faster? Overall, then, we must seek to have food production, transport and distribution moderated by social consensus and regulation in favour of practices that are both health supporting and ecologically sustainable.

Meanwhile, more immediate actions should be taken. Epidemiologists should conduct ad hoc orthodox studies, where they are potentially informative. Studies of immune system function in babies born to persons likely to have been particularly exposed during this recent episode would be a good starting point. Epidemiologists must also carry out population health risk assessments based on existing, albeit imperfect, knowledge and theory.³

Good research will illuminate the policy making and regulatory processes. In matters of food safety, in particular, there must be full disclosure of available information to the public and transparent regulatory procedures. Within Europe, international cooperation should be strengthened by extending, standardising and coordinating the national surveillance of food safety and foodborne health disorders. The controls should be located within the public health sector, not in the production or trade sectors. Links between food quality control laboratories and clinical laboratories should be strengthened. Culturally attuned health education for food preparers, school children and the general public should be promoted.

We face a difficult decade or two as modern food production methods evolve, as trading networks extend, as human mobility increases, as eating habits and consumer preferences change, and as deregulated market forces tend to distort good public health practice.^{10 13 15} The increasingly frequent food safety fiascos of the 1990s, however, have created a political opportunity to assert the primacy of public health as the guiding criterion of food policy in Europe.

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