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Toxicology, Environmental Health, and the "One Health" Concept

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Abstract The One Health concept promotes collaboration among veterinarians, physicians, scientists, and other professions to promote human, animal, and ecosystem health. One Health illustrates the interconnectedness and interdependence of human, animal, and ecosystem health. This concept has traditionally focused on zoonoses that are infectious diseases, not on chemical- or poison-related illnesses in animals and their relationship to the detection and prevention of human illness. The purpose of this article is to describe key experiences of scientists in the Health Studies Branch within the National Center for Environmental of Health of the Centers for Disease Control and Prevention in which the study of animal illness facilitated a public health investigation into an outbreak of chemical-associated human disease. The experiences highlight how utilizing the One Health approach may improve chemical-associated outbreak investigations and facilitate appropriate intervention strategies. An appropriate One Health approach in toxicology and environmental health in outbreak settings should include consideration of the common environments and food sources shared by humans and animals and consideration of the potential for contaminated animal products as food sources in human exposures.

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Introduction

The One Health concept is a collaborative effort among multiple disciplines working locally, nationally, and globally to attain optimal health for people, animals, and our environment [1]. This concept encourages interdisciplinary collaboration among veterinarians, physicians, and ecologists [2]. The One Health concept has historically focused on zoonoses that are infectious diseases [3]. Far less attention is paid to toxic exposures in animals, the relationship between this exposure and chemical-associated human illness, and how the One Health concept might apply in these situations.

Animal illnesses have alerted the medical community to toxicological disasters often well in advance of the appearance of adverse health effects in humans [4]. In 1956, an outbreak of unusual and severe neurological illness occurred in Minamata Bay, Japan. The etiology was ultimately determined to be ingestion of locally caught seafood containing extremely elevated concentrations of methylmercury. The most severely affected individuals were exposed in utero (through maternal consumption of affected fish during pregnancy). These children were born with various central nervous system deficits including blindness, seizures, and profound developmental delays. In the 6 years preceding this outbreak, it was noted that cats in the community exhibited abnormal neurologic behavior followed by death. This behavior included ataxia and convulsions and was ultimately determined to be due to methylmercury poisoning [5]. The great London fog of 1952, in which a temperature inversion caused a severe and

long-lasting smog to hang over the city, was responsible for more than 4,000 human deaths. The etiology was not recognized until nearly a year after the event. In retrospect, a cluster of respiratory-related sudden death in cattle at a stock show in London in 1952 was indicative of the air pollution problem before the human deaths were recognized. If the cattle deaths had been recognized as the harbinger of this public health threat, it might have helped inform public health response to minimize human illness [6]. Similarly, in 1971 in Times Beach, Missouri, an outbreak of equine sudden death alerted public health officials to the largest community-based dioxin exposure in the USA [7]. Differences in routes of exposure, susceptibility, and latency phases of illness often make animals a more sensitive indicator of chemical-associated public health threats, which, if recognized as such, can inform and aid in public health action to minimize or eliminate a threat.

The Health Studies Branch (HSB) of the National Center for Environmental Health, Centers for Disease Control and Prevention (CDC) has participated in several investigations and activities involving chemical-associated illness in humans in which animal disease and death have simultaneously or previously occurred, aiding in etiology identification and characterization of clinical features of the human outbreak. Our past experience has identified the following topic areas, which should be carefully evaluated for their potential relationship to human health, in outbreaks of chemical-associated illness in animals: common environments, common food sources, and consumption of contaminated animal products. Our objective is to describe each topic area and how it relates to the One Health approach for chemical-associated illness.

Discussion

Common Environments

The shared environment of animals and humans allows potential exposure to the same toxic agents. When outbreaks of illness in humans and animals occur concurrently, studying the disease in animals in addition to humans can provide insight into the etiology. In April 2009, the Bangladesh Ministry of Health (BMOH) requested epidemiological and toxicological assistance from CDC. HSB/CDC deployed an epidemiologist and a medical toxicologist to assist the BMOH in investigating a cluster of illnesses in children in the Dhamrai Sub-district of Dhaka, Bangladesh. The illness was characterized by sudden onset of respiratory distress and altered or loss of consciousness in children of two adjacent villages. The clinical signs and affected populations were similar to an outbreak that occurred in April 2008, also in Dhaka. The cases in 2008 tested negative for various infectious causes of respiratory and neurologic illnesses, including Japanese encephalitis, Nipah virus, and influenza. As in the 2008 outbreak, the 2009 cluster of illnesses was preceded by the sudden deaths of calves and puppies in the affected villages and surrounding agricultural fields, suggesting an environmental etiology. The clinical signs in both animals and humans were suggestive of cholinesterase inhibitor pesticide toxicity. Laboratory testing of human specimens ultimately revealed the likely agent to be carbofuran, a carbamate-type pesticide. Testing of animal specimens to confirm a common etiology is pending. More importantly, a system for investigating animal deaths is under development in Bangladesh to help identify human populations at risk for disease.

One of the predicted water-related consequences of climate change is a global increase in the frequency and distribution of toxin-producing harmful algal blooms (HAB). This increase is likely to affect the incidence of HAB-related health events directly through exposure to toxic cyanobacteria and indirectly through consumption of shellfish that ingest these toxins. Clinical signs of HAB-toxin exposure range from mild skin or respiratory irritation (resulting from inhalational or skin contact) to severe gastrointestinal illness. Exposure often also causes death in animals that have ingested water with HAB. In fact, these animal deaths are often the first sign that toxins have reached a level for public health concern and action [8]. The Florida Red Tide program has piloted an early detection system in which lifeguards monitor beaches for dead fish, dead animals, and skin and respiratory irritation among beachgoers. The lifeguards report any instances to a central system that issues public health warnings based on this information [9]. The National Center for Environmental Health, in collaboration with partner organizations, created and maintains a unique surveillance system—the HAB-related Illness Surveillance System—to capture human and animal health data, as well as physical characteristics of HABs in a single database [10]. These data comprise a historical record of the occurrence of HABs of public health importance and will eventually allow CDC to assess how predicted changes in the occurrence of HABs affect environmental health.

Common Food Sources

Shared sources of food and water for humans and animals are a common route of toxin exposure. A One Health concept in this topic area can improve public health response. In May 2004, CDC, the Kenyan Ministry of Health, and the World Health Organization responded to an outbreak of jaundice with a high mortality rate in central Kenya [22]. Several well-known infectious causes of jaundice are present in Kenya, and an infectious etiology

was initially suspected. However, recognizing concomitant deaths in animals fed the same maize that affected humans had eaten helped public health officials identify aflatoxicosis as the primary differential diagnosis. Aflatoxicosis is caused by consumption of a toxin produced by the Aspergillus fungus and is characterized by gastrointestinal illness and liver dysfunction, which, in severe cases, may cause death resulting from liver failure. Chickens, uniquely susceptible to aflatoxicosis, typically eat maize or corn also intended for human consumption [11–13]. Surveillance for jaundice/death in animal populations such as chickens in regions known to be endemic for aflatoxicosis could identify communities at risk for human illness.

The Ethiopian Ministry of Health asked HSB epidemiologists and medical toxicologists to help investigate an outbreak of chronic liver disease in a rural community of Northern Ethiopia in 2007 [14]. Clinical signs and symptoms among case patients were vague but included fever, epigastric pain, ascites, and organomegaly. Hepatitis, schistosomiasis, visceral leishmaniasis, and other infectious agents with similar clinical presentations were endemic to the area and therefore initially thought to be the etiology. However, the simultaneous recognition of similar signs, including ascites in local livestock, suggested an environmental cause [15]. The public health investigation is ongoing, and the current leading hypothesis with regard to etiology includes pyrrolizidine alkaloid (PA) intoxication from consumption of local vegetation [14].

Consumption of Contaminated Animal Products

Much work in the One Health field has highlighted human infectious illness attributed to consumption of contaminated animal products. Globally, animals are an important part of the human diet, and recognizing the potential for human illness from animals contaminated with chemicals is vital. PAs are produced by plants and thus most commonly a contaminant of grains or plant-derived products such as teas and herbal medicines. If eaten by production animals, PAs can be excreted in milk and can accumulate in certain organs, such as the liver, which may be consumed by humans [16]. If the etiologic agent in the Ethiopia liver disease outbreak proves to be PAs, milk and organ meat from sick animals could be an exposure source for humans, and subsequently, an appropriate area for public health intervention.

In the early 1970s, cattle and chickens in Michigan were inadvertently fed grain contaminated with poly-brominated biphenyls (PBB). This flame retardant is also a persistent organic pollutant that bioaccumulates in adipose tissue [17]. People were exposed to PBB through the consumption of meat, milk, and eggs of affected animals. PBB consumption caused devastating health consequences in heavily contaminated cattle [18]. Though PBB was manufactured for only a short time, the health effects in humans exposed through the food chain were recognized several years later [19]. Earlier recognition of the problem in animals could have led to a faster diagnosis and prevented human consumption of contaminated animal products. Scientists from HSB partnered with epidemiologists from the Emory Rollins School of Public Health to study the endocrine system abnormalities occurring in individuals with high body burdens of PBB resulting from either direct consumption of contaminated animal products or mother-to-child transmission of PBB [20]. Thorough characterization of low-dose and trans-generational effects of persistent organic pollutants will allow us to better identify thresholds and contaminants of concern in the food chain to better protect public health. This intergenerational exposure scenario has contemporary relevance to environmental health as demonstrated by the recent dioxin contamination of animal products in Europe [21], and it highlights the global nature of chemically contaminated animal products and human health.

Conclusion

Our experience suggests that public health officials should be vigilant in recognizing that outbreaks of animal illness may be indicative of a potential public health threat to humans. Simultaneous clusters or outbreaks of illness in both humans and animals living in close proximity to each other can suggest an environmental etiology and may be the result of chemical exposures. Public health officials should consider common environments, common food sources, and potential for consumption of contaminated animal products in these incidents when determining risk to human populations. These determinants may in turn facilitate identification of the etiology and help resolve the outbreak. The investigations, events, and activities described here highlight the benefit of a One Health approach in suspected and known outbreaks of chemical-associated illness in order to minimize the effects on human health.

Disclaimer The findings and conclusions of this report are those of the author(s) and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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