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EFFECTS OF AIR POLLUTION ON ANIMALS

Introduction

Interest in the effects of air pollution on animals has generally developed as a corollary to the concern about this influence on human health. Studies in which animals have been exposed experimentally to air pollutants have been prompted by the need for specific information concerning the toxicity of various known pollutants. Such information will assist in defining the effects on man. Moreover, animal morbidity and mortality following air pollution disasters have been recorded, at least partially, to seek a better understanding of the human injury.

There has been a direct concern about the injurious effects of air pollution on animals because of the considerable economic loss which has been claimed in some instances and is potentially associated with this hazard. This interest is illustrated by the research which has been devoted to the problem created by airborne fluorides.

The concern over the threat of air pollution to the health of man and animals has developed for the most part during the past decade. With the exception of the reports dealing with fluorosis, there is little to be found in the veterinary literature about air pollution as a cause of disease. Most of the information concerning the natural exposure of animals to air pollution is contained in the reports of some major air pollution disasters—e.g., Donora, London, Poza Rica. Recently, considerable information has been reported from medical research laboratories which describes the results of experimental exposure of small animals to various air pollutants. The advent of nuclear fission has added a new category of pollution to be studied and evaluated.

This chapter will attempt to appraise the reports which developed from investigations of the air pollution disasters at Donora, London and Poza

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Rica. Attention will also be given to the laboratory reports concerning air pollutants to which animals have been naturally exposed. The information concerning animal fluorosis will be reviewed, as will observations pertaining to the effects of radioactive fall-out on animals.

Major Air Pollution Disasters

A detailed investigation of both human and animal injury was conducted following the air pollution incident at Donora, Pennsylvania in 1948 (*Publ. Hlth Bull. (Wash.)*, 1949). A retrospective survey disclosed that an appreciable number of animals were reported to have become ill and that some died during the week of intense smog. The information concerning animal morbidity and mortality was obtained from the owners of both pets and farm animals by personal interviews conducted by registered nurses among the urban population and by a veterinarian among the farmers. Reports obtained by the nurses were subsequently investigated by the veterinarian.

Corroborative evidence was sought through conferences with three local veterinarians, technicians in a local dairy-cattle breeding association, two county agents, three local poultry dealers and a slaughterhouse operator. Four retail milk plants in the area were visited to examine milk production records during and after the smog. This group of observers was unable to indict the intense smog as being responsible for any noticeable effect upon the health or performance of animals. Milk production did not decline during nor immediately after the smog, according to the dairy operators.

The reports obtained through interviews with animal owners and caretakers indicated that the different species varied in their susceptibility to injury. Dogs were reportedly the most susceptible species. Of 229 dogs included in the survey, 15.5 % were reported as having become ill and 10 canine deaths were attributed to the smog. Nearly all of these fatalities occurred among dogs of less than one year of age.

The investigators grouped the signs of the canine illnesses attributed to air pollution into three syndromes. Signs of the respiratory syndrome, which occurred most frequently, included cough, sneezing, conjunctival congestion, dyspnoea and nasal discharge. Signs of the digestive syndrome included emesis, retching and diarrhoea. The third syndrome consisted of anorexia which occurred with or without lassitude. With the exception of the fatal cases, the canine illnesses were generally mild and short-lasting. The mean duration of the illnesses reported for 31 dogs was between three and four days.

Twelve of 165 cats which were included in the animal investigation at Donora were reported as having become sick during the smog interval.

Three cats died after developing signs which were similar to those described in dogs.

There were few observations of illness and death among poultry. Mild signs of respiratory distress were reported as occurring in two of 43 urban flocks. Four rural poultrymen observed that their chickens became sick and that approximately 40 % of the affected birds had died.

The larger farm animals including horses, cattle, sheep and swine were generally unaffected by the Donora smog. Three dairy farmers reported, however, that their cows had developed a cough which coincided with the occurrence of intense air pollution. At one farm five cases of calf pneumonia were said to have occurred immediately after the smog.

In appraising the reports of animal injury during the intense smog at Donora several considerations appear to be important. It is significant that professional and experienced observers of animal health failed to regard the smog as a factor in the morbidity and mortality which occurred among animals at that time. It should also be noted that while it is entirely possible that the reported illnesses could have been caused by the inhalation of irritating gases and fumes, the signs which were observed are consistently associated with common animal diseases occurring in this locality.

The significance of the animal effects reported at Donora is further clouded by the inability to indict specific air pollutants as likely causes for the morbidity and mortality which occurred. The chief contaminants of the Donora smog were later estimated to have been well within the range of their respective toxic levels. The farm animals and poultry were not located in the centre of intense pollution and so relative exposures are unknown.

It is impossible to make a fair estimate of the relative susceptibility of man and animals in this episode. Although the reactions of the animals are not biased by subjective reflection, the reports of the owners may be. In man, 10 % were severely affected and an additional 17 % moderately affected. The 15 % of dogs reported as having become ill might have consisted only of those with rather severe reactions, making the dogs' susceptibility roughly equivalent to that of man. It seems unlikely that any of the species of animals affected at Donora would provide a sensitive biological indicator for the degree or nature of human illness caused by this type of air pollution.

During the interval of the London fog in 1952, a number of prize cattle were reported as having been severely affected (*Brit. med. J.*, 1953; Joules, 1954). These animals, which were grouped for exhibition, developed acute respiratory distress. Five of them died, 11 were subjected to emergency slaughter and over 40 others developed signs which were attributed to the fog. Post-mortem examinations revealed acute bronchiolitis, emphysema and right heart failure. Sulfur dioxide was the chemical component of the

London fog which was proposed as the cause for the morbidity and mortality (Drinker, 1953).

A related sulfur compound, hydrogen sulfide, has been held responsible for another major air pollution disaster at Poza Rica, Mexico, 1950 (McCabe & Clayton, 1952). Following this incident, an investigation disclosed that the animal population apparently had been affected. An undetermined number of canaries, chickens, cattle, pigs, geese, ducks and dogs were exposed to the polluted atmosphere at Poza Rica. The reports of animal injury during this disaster disclosed that 100 % of the canaries in the area had died. It was estimated that 50 % of the other exposed animals had died during the period of air pollution.

Exposure of Laboratory Animals

A considerable body of information is beginning to become availabel from laboratory experimentation with small animals. Such experimentation is capable of providing more specific and more reliable information in three basic categories. First, the animals themselves can be of known genetic constitution, with relatively well defined physiological constants and susceptibilities to infectious disease (Russell, 1955) and, eventually, to air pollutants. Secondly, the exposures to air pollutants may be controlled both qualitatively and quantitatively. Thirdly, responses of the animals to the exposures may be measured in detail and by techniques not readily applicable to man. Laboratory experiments serve both in the basic field of testing known contaminants and as a follow-up to field epidemiological studies, to determine whether true causal relationships exist between potential causes and effects noted in the field.

Most available results from laboratory experiments define the reactions of animals to substantial concentrations of specific chemical air pollutants. The study of potential health hazards from air pollution as it occurs today must be concerned more with chronic low-level exposure than with the acute episode of short duration and high concentration. Although further acute episodes may be expected and anticipated, and may even produce local disasters of consequence, it is the long-term, widespread exposure to daily air pollution which must be examined thoroughly for delayed effects upon the health of animals and man. Epidemiological investigation of "natural" pollution may be so confounded by environmental factors and other intercurrent pathology that no specific effects can be differentiated. It is upon this thesis that the need for experimental animal epidemiology is predicated.

Animal species have been shown to vary in their susceptibility to various air pollutants. The observations at Poza Rica suggest that avian species, especially canaries, may be considerably more susceptible to hydrogen

sulfide than are the more common laboratory animals. The fate of fattened cattle during the London fog elicits the conjecture that increased stress associated with the rapid deposit of considerable fat may alter the cardio-respiratory functions, thereby increasing susceptibility to air pollutants.

Rats exposed to silica and feldspar dust did not exhibit increased susceptibility to lobar pneumonia (Baetjer & Wintinner, 1944). Comprehensive studies have been made of the effects of sulfur dioxide and sulfuric acid on guinea-pigs (Amdur, 1954; Comar et al., 1957). A combination of these two had a much more marked effect than equivalent concentrations of either alone. Effects were demonstrated on growth, lung pathology, and airway resistance. Similar physiological effects have been noted in man (Amdur, Melvin & Drinker, 1953; Greenwald, 1954), and similar signs of illness and post-mortem pathology were noted in the cattle exposed to the London fog (Pattle & Cullumbine, 1956). Hyperpnoea, dyspnoea, and depression are exhibited commonly by most laboratory animals. Guinea-pigs are recorded as being more susceptible to these toxins than mice, rats or goats (Pattle & Cullumbine, 1956). Necropsies of exposed guinea-pigs have revealed the presence of pulmonary haemorrhages, oedema, and, in addition, consolidation and hepatization (Amdur, Schulz & Drinker, 1952). Studies in progress in this same laboratory show that the combination of sulfur dioxide and a sodium chloride aerosol produce airway resistance effects many times greater than sulfur dioxide alone.

At this time a number of studies are well under way to determine the effects of single or mixed air pollutants on the enzyme systems of specified tissues or cells. Preliminary reports of a number of these projects are included in the proceedings of the Air Pollution Research Planning Seminar (US Department of Health, Education, and Welfare, 1956).

Exposure to Oxidizing Air Pollution

The natural exposure of animals to atmospheric pollution characterized by the reaction of unsaturated hydrocarbons and oxides of nitrogen in the presence of sunlight has been investigated but superficially to date. This type of air pollution is exemplified by that which mars the atmosphere of Los Angeles (Haagen-Smit & Fox, 1956).

In 1954, an intensive survey of the air pollution problem in Los Angeles was conducted. During this time the effects on animal health were cursorily investigated. The information obtained was apparently regarded as inconclusive since it was not included in the published report of the survey (California State Department of Public Health, 1955). More recently, it has been discovered through the medium of a questionnaire that veterinarians in Southern California occasionally incriminate air pollution as a cause of

illness among animals (Catcott, 1957, unpublished data). Signs of ocular and upper respiratory tract irritation among dogs and pet birds are rather frequently attributed to atmospheric contaminants.

Artificial exposure of laboratory animals has provided some evidence of the effects caused by some of the chemical agents present in this type of smog. The studies of the carcinogenic properties of ozonized hydrocarbons illustrate this information (Kotin, 1956). Both dermal and pulmonary cancers have been produced in mice artificially exposed to an irradiated mixture of ozone and unsaturated hydrocarbons. Skin painting with aromatic hydrocarbons produced skin tumours in both C57 black and strain-A mice (Kotin, Falk & Thomas, 1956). Skin painting with aliphatic hydrocarbons also produced skin tumours in C57 black mice. Of more interest and probably more significance is the finding that pulmonary tumours were produced in strain-A mice after their exposure to an atmosphere of ozonized gasoline (Kotin & Falk, 1956) In these mice tumours developed in 41 % under washed air conditions and in 80 % in polluted air. Results on the C57 black mice under similar exposure are reported by Kotin & Falk (1958). The control animals showed a very low percentage of lung tumours, whereas over one-third of those exposed to polluted air produced tumours. Additional biological effects on these mice will be reported in detail. At the moment it has been noted that the mice housed in a polluted atmosphere showed a consistent weight deficit in comparison with the controls.

These laboratory observations are at present being complemented with a field survey of pulmonary pathology in dogs (Catcott, McCammon & Kotin, 1958). In this study, dogs of known age and period of residence in the Los Angeles area are being examined post mortem. Both gross and microscopic observations are being made of the major organs, particular attention being given to pulmonary tissues.

Ozone was considered to be an important component of the Los Angeles type of air pollution. Rather extensive studies on the toxicology of ozone have been conducted and are still being pursued (Stokinger, 1954; Stokinger, Wagner & Wright, 1956; US Department of Health, Education and Welfare, 1956). The irritant qualities of ozone have been demonstrated in dogs, cats, rabbits, mice and guinea-pigs. Exposures at toxic levels have produced marked pulmonary changes, including death. Oedema and haemorrhage characterize the morphological changes of lungs which have been injured with ozone. In nearly all experiments, the concentrations of ozone have been greater than those observed to occur naturally. Although ozone was considered to be a representative constituent of the oxidizing type of smog, opinion is now developing that ozone itself is not the responsible agent in this type of smog. Experiments in progress indicate that animals may develop a tolerance to ozone, or that they may be made more susceptible through the medium of environmental stress.

Animal Fluorosis

While fluorine toxicosis is usually induced by a less direct process than inhalation, the chemical agent is air-borne. Fluorosis of animals has become fairly well defined during the past ten years. Chronic fluoride poisoning probably results from the distribution of fluorine in the air. Fluorides are widely distributed in soils, water and animal feeds. It has been stated that it would be difficult to devise an animal ration which would contain less than 2-3 p.p.m. of fluorides (Phillips, 1952).

The sources of fluoride present in, or on, pasture crops include: (*a*) the natural dusts from soil in certain localities. Some top soil in Idaho has been found to contain as much as 1640 p.p.m. of fluoride; (*b*) dusts and gases from certain factories; (*c*) the combustion of coal, which results in the dispersion of fluoride-containing materials (Largent, 1952). Gaseous effluents from the enamelling, cement, and aluminium industries and from cryolite or acid phosphate production contain hydrogen fluoride or other gaseous fluorides.

The occurrence and distribution of livestock fluorosis in the USA has been contemporaneous with the development of the industries previously mentioned. The fluoride-containing effluents from these industries may contaminate the forage crops which are subsequently ingested by animals. Air contamination is greatest in the area adjacent to industrial sources and decreases with distance as the effluents become dispersed (Huffman, 1952). Sufficient forage contamination to produce the early signs of fluorine toxicosis may occur at a distance of seven miles (11 km) or more in the direction of the prevailing wind. Certain industrial establishments have made every effort to prevent this type of contamination; others are still a problem.

Fluorine is a protoplasmic poison. It has a marked affinity for calcium and interferes with normal calcification (Madsen, 1942). Livestock are observed to be more resistant than are humans to dental mottling, which is an early sign of fluorine toxicosis. Cattle and sheep are the most frequently affected animals (Largent, 1952). Experimental work has indicated that the maximum safe levels of daily fluoride ingestion are: 3 mg/kg bodyweight for cattle and sheep, and 10 mg/kg for chickens. Only one clinical case of fluorine poisoning in a horse has been reported in the USA (Largent, 1952).

The pathogenesis of fluorine toxicity consists of hypoplasia of dental enamel followed at higher levels of fluorine intake by abnormal growth of bones. The pathology is manifested by mottling, staining and pitting of incisor teeth. Generally, only the permanent teeth are affected. Excessive wearing of incisor and molar teeth with subsequent gingival injury may occur at high levels of fluorine intake. Bone lesions may develop at any age. Their appearance indicates that there has been a high fluorine intake over a long period. The pathology is manifested by isolated exostoses or by a

diffuse thickening of long bones. Lameness is a consequence when joint surfaces become involved. Signs of advanced fluorosis include anorexia, diarrhoea, weight loss, lowered fertility and reduced milk production. Positive diagnosis of fluorosis is based on a chemical determination of fluoride levels in bones or teeth. Normal fluoride levels in bone ash of unaffected animals range from 200 to 800 p.p.m. About 2 mg daily of fluorine per kg of bodyweight will produce dental mottling in cattle within four to five years. The toxicity of fluorine appears to be greater in forage contaminated with gaseous effluents than with rock phosphate. The storage of fluorine in the soft tissues of cows on high level intakes is reportedly low. Therefore, neither meat nor milk from such animals is likely to be injurious to humans. The prospect of controlling industrial fluorosis of livestock appears to be good. The element is so widely distributed in nature, however, that its complete eradication as an industrial waste is unlikely (Huffman, 1952).

The exposure of rabbits and guinea-pigs to high concentrations of hydrogen fluoride has been observed to be irritating to the respiratory tract (Machle et al., 1934). Slowing of the respiratory rate was observed at low concentrations. Increased exposure caused corneal erosion, necrosis of turbinate bones and marked conjunctival and nasal discharges. Death of these laboratory animals following their exposure to hydrogen fluoride was usually due to bronchopneumonia.

Exposure to Ionizing Radiation

Radioactive fall-out from nuclear weapons testing is a unique air pollutant in that the associated biological effects occur as a result of ionizing radiation rather than chemical toxic action. The effects of radiation on animals are similar qualitatively to those in humans (Hollaender, 1954; National Academy of Sciences, 1956a). They can arbitrarily be categorized as acute radiation effects and the delayed or long-term effects.

Signs of acute radiation injury develop within a period of hours to weeks following exposure. Only fall-out occurring close to the weapon's test site can produce radiation levels sufficiently high to result in acute radiation effects (Cronkite, Bond & Dunham, 1956; National Academy of Sciences, 1956b; US Department of the Army, 1957).

Our primary interest is in the long-term effects that may be associated with global fall-out. The most important long-term effects to consider are: (*a*) cancer (including leukaemia); (*b*) shortening of life span (non-specific aging); and (*c*) the genetic or mutation effect. These effects generally require several years, or, in the case of genetic changes, generations following exposure, to be manifested. Our concern about these effects in animals is

not only in the interests of animal health, but principally for the information that can be extrapolated to man. In the USA, the National Academy of Sciences (1956a) points out:

> Laboratory studies of long-term effects have been relatively few, partly because their importance was not early appreciated and partly because they are very expensive and time consuming owing to the necessity for maintaining considerable numbers of animals for all, or most, of their normal life spans. In consequence, the data on late effects in animals are meagre in certain areas. Such data as there are in man are sufficiently in agreement with those on other mammals to lead to the expectation that extrapolation from lower mammals to man will be possible with fair accuracy.

The Academy (1956b) also reports:

> Radiation from fall-out inevitably contaminates man's food supply. Radioactive elements in the soil are taken up and concentrated by plants. The plants may be eaten by humans, or by animals which in turn serve as human food. At present the contamination is negligible. But the maximum tolerable level is not known. There is not nearly enough information about the long-term biological effects on man or animals from eating radiation-contaminated food. Research in this area is urgently needed.

We are interested in the quantities of radioactive fission products being assimilated by animals throughout the world, because such data provide a biological index of the level of environmental contamination and give warning of the build-up of potentially hazardous levels.

Compared to other air pollutants it is relatively easy to determine environmental levels of radioactive fall-out and to measure the body burden of fission products assimilated by animals. In recent years there have been numerous studies to measure levels of select fission products naturally occurring in animal tissues and animal products as a result of fall-out (Anderson et al., 1957; Comar et al., 1957; Kulp, Eckelmann & Schulert, 1957; Van Middlesworth, 1956).

Summary

Reports concerning the effects of air pollution on the health of animals have four general sources. Reports of animal injury and death have followed investigations of the air pollution disasters at Donora, London, and Poza Rica. Extensive studies of air-borne fluorides and their relation to animal health are available. Observations of the effects of air-borne radioactive substances on animals provide a third source. Finally, the reports of artificial exposure of laboratory animals have enlarged our understanding of the effects of various air pollutants on animals.

It must be conceded that the present information concerning this subject is quite inadequate. The reports of animal morbidity and mortality

which followed major air pollution episodes should be regarded critically. The investigations of these acute and intense exposures to air pollution have been done retrospectively. It is significant that the owners' reports of injury to animals could not be corroborated by professional observers at the Donora disaster. The high rate of animal mortality which allegedly occurred at Poza Rica is generally in contradiction to the information concerning the relative susceptibility to air pollutants of animal species which have been studied experimentally.

The synergistic roles of physiological and of external environmental influences on reactions to air pollution indicate that the interactions of many factors may be necessary to produce critical situations. Genetic attributes of the individual animal as well as the species may define specific parameters of physiology and nutrition within the animal, whereas meteorology and type source of air pollution define exposures. The association of animals and atmosphere may be the final requirement for specific biological effects.

There has been very little study of animals subjected to natural exposure to air pollution of the kind prevalent on the west coast of the USA. Investigations of naturally occurring effects of this form of air pollution are at present being conducted. Most of our information concerning the oxidant type of pollutant has been and currently is being obtained in experimental studies of laboratory animals.

In contrast to the paucity of information concerning natural exposure to most air-borne pollutants, the effects of fluorides on animals have been defined well. The incidence of animal fluorosis and its nature have been described. Methods of controlling this hazard to animal health are known and utilized when the threat of fluorosis occurs.

Laboratory research has provided important information concerning the effects of specific pollutants on animals. Mice, rabbits, guinea-pigs, rats and monkeys have been utilized to demonstrate the toxic properties of such air pollutants as sulfur dioxide, sulfuric acid, hydrogen sulfide, ozone, nitrogen dioxide, organic compounds, and some dusts. Information which has been obtained by artificial exposure of animals is providing some indices of both human and animal effects to be expected from natural exposures. A well-integrated attack, in the field and in the laboratory, will be necessary to divulge the true details of the biological effects of polluted air.

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