Ecotoxicology and Climate
Edited by P. Bourdeau, J. A. Haines, W. Klein and C. R. Krishna Murti

4.5 *Effects on Domestic Animals*

R. K. RINGER

4.5.1 INTRODUCTION

Domestic animals are kept in many different climates, from the harsh cold environments of the north to the hot arid environment of the deserts and the hot, wet climates of the tropics. Stressful climatic conditions, either hot or cold, tend to aggravate the impact of xenobiotics on livestock production. The problem is further aggravated when modern intensive agricultural techniques, with increased stocking densities, place many animals in close proximity on a single site. Conversely, animals that are distributed over large rangelands are far less subject to the dangers of massive chemical exposures.

Chemical substances can enter the environment of domestic animals by direct application or in some instances by complex pathways. Application of pesticides to animals or their housing facilities, the use of fertilizers, including animal wastes, or herbicides or insecticides to cropland, and the use of sanitizing agents or veterinary drugs are examples of the direct exposure route, whereas indirect entrance may result from food-chain contamination, industrial wastes and accidents, or from combustion processes. The occurrence of chemical residues in domestic animal meat and by-products, including eggs and milk, reflects the increase in the use of agricultural chemicals as well as an increase in pollution of the environment in which the animals are reared and processed.

Tropical, arid, and subpolar regions may alter the utilization of these chemicals in animal agriculture. This is particularly true of veterinary drugs used in microbial infections, in the prevention of disease and infections, and in parasitic control or treatment. Phytotoxins are another case because of the impact that environmental conditions have on their diversity.

4.5.2 ANIMAL TOXICOSIS AND THE ENVIRONMENT

Toxicants, natural and synthetic, may impact on animal agriculture in different ways: (1) by directly or indirectly intoxicating animals (toxicoses), resulting in

mortality or decreased production of edible food products; (2) by decreasing the availability or usability of nutritious feedstuffs due to the presence of naturally occurring toxins or added toxicants; and (3) by decreasing the wholesomeness of edible food products due to the presence of hazardous residues (Shull and Cheeke, 1983).

There is a wealth of information on domestic animal toxicology in the temperate zone (Ruckebusch *et al.*, 1983; Osweiler *et al.*, 1985). However, when we move to consider the arid, tropical or subpolar regions, we must also consider the effects that light, temperature (both cold and hot), and rainfall have on the toxicology of chemicals on these domestic animals. Domestic animals are homeotherms and as such can maintain a thermoneutral zone over a range of ambient temperatures. When the ambient temperature moves outside the thermoneutral zone the animal must alter its metabolic rate to maintain a constant core body temperature. Yousef (1985a) has presented an excellent review of domestic animal production and physiology under the stress of cold and heat in various regions of the world.

Animals reared outside the temperate region are subjected to large swings in temperatures beyond the thermoneutral zone. Animal metabolism and behaviour are altered, characterized by a shift in food and water consumption, passage rate of food through the digestive tract, hormone synthesis and release, panting and shivering, and reduced activity to name a few. With an altered metabolism there is a potential for xenobiotics to be handled differently by animals. The converse is also true in that thermoregulation can be affected by a variety of toxic substances, thus altering the ability of domestic animals to tolerate thermal shifts in ambient temperature. For example, it has been demonstrated that the organophosphates, parathion or chlorpyrifos, reduce the tolerance of animals to cold exposure (Ahdaya *et al.*, 1976; Rattner *et al.*, 1982; Maguire and Williams, 1987). Organophosphate and carbamate pesticides are known to be cholinesterase inhibitors and to cause hypothermia in animals. Acetylcholine is one of the hormones involved in maintenance of body temperature; thus, cholinesterase inhibitors have an effect on thermoregulation in mammals (Ahdaya *et al.*, 1976) and birds (Rattner *et al.*, 1982). The inability to thermoregulate may not be the cause of greater death losses, at least in birds; rather, the cause may be reduced insulation brought about by loss of subcutaneous fat, or depletion of carbohydrate and lipid reserves induced by decreased food intake (Rattner *et al.*, 1982).

Species that are indigenous to a region are better able to maintain a more constant core body temperature than non-adapted animals. This ability is brought about through anatomical and physiological adaptations that have occurred. The question has been asked, do arctic birds and mammals maintain body temperatures within the same range as species from temperate regions? Both birds and mammals subjected to environmental temperatures of $-30°C$ and $-50°C$ maintained their body temperatures within normal limits (Schmidt-Nielsen,

1979). Protected by insulation, animals in arctic climates do not need to eat more than animals in milder climates (Yousef, 1985b). The climate of the arctic region is not always cold, but rather is characterized by extreme variation from winter cold to summer heat and great differences in sunlight between winter and summer days (Irving, 1964). Non-adapted animals experience difficulty in adapting to these extremes.

In general, animals indigenous to tropical areas of the world are better adapted for heat exposure than those from temperate regions (Ingram and Mount, 1975; Yousef, 1982). In the tropical areas, animals such as cattle possess an increased ability to lose heat due to a greater surface area in the region of the dewlap and prepuce and increased numbers of sweat glands and the presence of short hair. Fat stores may be in humps or intermuscular rather than subcutaneous, which assists the conductance of heat from the core to the surface skin. Animals from the temperate region with high productivity have often failed to continue to yield as well when exposed to tropical climates with extremes in temperature.

In hot, arid climates adaptation mechanisms aid in the maintenance of normal body temperatures. Heat storage, insulation, panting, gular fluttering, and blood flow mechanisms to cool the brain are just some of the physiological adaptations used by animals in hot climates with limited water supplies (*see* Schmidt-Nielsen (1979) for review). Because domestic animals are homeotherms and indigenous animals are adapted to withstand climatic variations, the problems with xenobiotic exposure are not greatly different from those in the temperate region. However, several problems are amplified. At high ambient temperatures, there is an increase in water consumption and a concomitant decrease in feed consumption (National Research Council, 1981). If exposure to a chemical is via water the exposure will be increased and the animal may be at greater risk.

In cold climates, feed consumption increases during extremes of cold making exposure via the food chain an increased problem. It is known that food restriction and/or water deprivation may significantly alter the response of an animal to toxic chemicals (Baetjer, 1983). These changes in food and water consumption, which mark the principal metabolic shift in animals in response to environmental fluctuations, may contribute to toxicological differences in pesticides between the world's regions.

4.5.3 NATURAL TOXINS

Toxigenic fungi have ubiquitous geographical distribution influenced by climatic conditions, cultivation, and harvesting techniques, as well as storage procedures and the livestock production practices used. Mycotoxins occur in particular feeds and in particular regions (National Research Council, 1979; Smith, 1982). Aflatoxins are comparatively common in subtropical regions and depend on factors such as weak plants resulting from drought stress, insect or mechanical damage, climatic conditions before drying, and improper storage conditions

(Galtier and LeBars, 1983). In tropical areas of the world, mycotoxins in grain, protein concentrates, and other feedstuffs are a major problem because warm, humid environmental conditions favour fungal growth, and farming practices in many tropical areas are not sophisticated (Cheeke and Shull, 1985). Also, crop storage conditions are frequently inadequate in these areas. Thus, in hot humid regions the production of natural chemicals in feedstuffs may result in toxicosis, posing a problem for livestock production.

4.5.4 CHEMICAL ACCIDENTS WITH PESTICIDES

Chemical accidents that adversely affect animal agriculture have occurred in the past and will doubtless occur in the future. These problems are not unique to any area of the planet and do not hinge on climatic conditions. As an example, when organochlorine insecticides were being phased out because of their adverse effects on non-target organisms, together with their persistency in the environment and their carcinogenicity, the organophosphate insecticides were introduced as logical replacements. Some of these compounds were halogenated phenyl phosphonates and phosphonothionates that were lipid soluble, persistent, and of lower toxicity to mammals than the parathions and other widely used organophosphorus insecticides. However, a number of them were known to be delayed neurotoxins (Metcalf, 1982). Leptophos, one such chemical, was used in 1971 to control the cotton leafworm in Egypt. Some 1300 water buffalo died from paralysis and distal axonopathy characteristic of delayed neurotoxicity (Abou-Donia *et al.*, 1974). Human poisoning was also evident (Hassan *et al.*, 1978). Egypt was only one of about 50 countries into which leptophos was sold. This example of toxicosis in livestock is one where water from cropland collected in a river and water consumption in a hot, arid climate caused the death of many animals. This is not an isolated case of chemical toxicosis in livestock, but documentation in the literature is not common. In temperate regions there are numerous reports of poisoning in intensified animal production units (Shull and Cheeke, 1983). These accidents have included such chemicals as polychlorinated biphenyls, polybrominated biphenyls, tetrachlorodibenzo-p-dioxins, and organochlorine insecticides.

4.5.5 MINERALS

Lead is considered to be one of the major environmental pollutants and has been incriminated as a cause of accidental poisoning in domestic animals in more cases than any other substance (National Research Council, 1972). Lead that contaminates the environment is largely air-borne but is redeposited by dust into soil and water and is taken up by or exists on the surface of plants which are grazed by livestock. Cattle, sheep, and horses are good indicators of pollution on vegetation (Debackere, 1983). Lead toxicosis in cattle from the use of lead-

based pigments in paint was common, as was poisoning of water fowl by spent lead shot. Restriction in the use of lead-based paints and, currently, in lead shot has reduced the problem in the United States. Lead from smelters may cause problems in horses grazing in adjacent areas. Dogs and cats give a very good indication of lead pollution in urban areas as the concentration of lead in their livers and kidneys increases with increased pollution (Debackere, 1983).

Animals can be exposed to mercury contamination from air, soil, water, and ingestion of contaminated feed. Mercury contamination results from fossil fuel combustion, agricultural fungicides, smelting of commercial ores, and through industrial discharge followed by water and, then, fish pollution. The fish are incorporated into animal feeds by way of fishmeals or protein concentrates. Toxicosis of domestic animals has also been due to the consumption of contaminated grain. In 1971, Iraqi authorities ordered 73 000 tons of wheat and 22 000 tons of barley from suppliers in Mexico and Canada, respectively, that were treated with mercury. This grain was used for planting but some was prepared into homemade bread. Oral ingestion may have included meat and other animal products obtained from the livestock given the treated grain (Bakir *et al.*, 1973). The latent period between dose and onset of symptoms may have given farmers a false sense of security since chickens given wheat for a period of a few days did not die (Bakir *et al.*, 1973). Hospital admission amounted to some 6530 cases (Clarkson *et al.*, 1976). Both man and animals are subject to mercury toxicosis through contaminated grains.

In Minamata Bay in Japan, methylmercury poisoning was observed in cats before human cases were recognized. The common thread between the disease in cats and that in humans was shown to be the consumption of mercury-contaminated fish (Hodges, 1976). Under modern feeding and management conditions of livestock production, cadmium toxicosis is relatively unimportant (Neathery and Miller, 1975) but does not preclude ingestion of recycled waste material, such as sewage sludge, in which cadmium may be concentrated. Cadmium is toxic and is an antagonist of zinc, iron, copper, and other elements. Some plants, such as clover, have the capacity to concentrate cadmium from soil.

Arsenic may result in contamination of livestock in areas surrounding smelters and where arsenicals are used for weed and insect control. Since fish are often high in arsenic, fishmeals may contribute sizeable quantities of arsenic to livestock. Non-ruminants are generally more susceptible to intoxication than are ruminants or horses. The degree of toxicity in ruminants is variable and may depend on the route of exposure, age, nutritional status, and exposure duration (Case, 1974; Selby *et al.*, 1974).

There are relatively few veterinary examples of acute copper toxicosis except in cases of accidental overdosing or the consumption of copper-containing compounds. Sheep are extremely sensitive to excess copper and therefore are

good indicators of environmental pollution by copper. Concentrations two- to three-fold above normal grass copper concentrations of 8–15 ppm are toxic to sheep (Debackere, 1983).

Selenium is used in some areas as a supplement to animal diets; still, in other areas, there may be selenium toxicity due to high levels. Dietary selenium requirements are approximately 0.1 to 0.3 ppm, while toxic concentrations are about 10 to 50 times greater (National Research Council, 1980). When pasture is limited in dry weather, accumulator plants may be readily available and eaten by livestock, resulting in selenium poisoning. In the United States irrigation of arid land with water high in certain minerals, including selenium, has resulted in a large site where water fowl have shown evidence of what appears to be selenium toxicosis.

These are but some of the adverse effects minerals have produced on livestock production. In some cases climatic conditions alter their consumption but in most instances exposure is indirect.

4.5.6 ANIMALS AS POLLUTION INDICATORS

It is clear that domestic animals can serve as indicators for environmental pollution by chemicals. The role of domestic animals as indicators for environmental pollution through pesticides is negligible (Debackere, 1983). Based on present evidence, fish and birds appear more susceptible than mammals to pesticides, especially organophosphates, carbamates, and chlorinated hydrocarbons (Walker, 1983; Debackere, 1983). Physiological and anatomical differences are likely to affect susceptibility to a wide range of compounds. In domestic birds the excretory route via egg laying and the fact that blood from the gut goes to the kidney via the renal portal system prior to hepatic contact conveys an advantage to birds over mammals; however, the high body temperature, urinary release into the cloaca, and relatively small liver render birds more susceptible to pesticides (Walker, 1983). Physiological and biochemical evidence suggests that birds have less effective defence mechanisms than do mammals to xenobiotics.

Generally, indicator species are chosen for their toxicological susceptibility (Kenaga, 1978). Chickens are regularly used in the laboratory as predictive models for delayed neurotoxicity by organophosphorus chemicals. In addition to the chicken, the human, water buffalo, horse, cow, sheep, pig, dog and cat have been reported to be sensitive, while common laboratory animals, such as the rat, mouse, rabbit, guinea pig, hamster, and gerbil, are not. The adult chicken is utilized most frequently as the test animal; however, the cat can be used and may serve as an excellent model in extrapolation to man.

Wild birds are invaluable models for environmental toxicology due to their abundance, visibility, and diverse habitat associations (Hill and Hoffman, 1984), and are used to monitor pollution in urban as well as in aquatic environments.

In Guatemala, hogs fed seed wheat treated with organomercury as a fungicide developed blindness, lack of coordination, and posterior paralysis in 2 to 3 weeks (Ordonez *et al.*, 1966). Humans followed with similar signs of toxicosis from eating the same wheat.

When it comes to chemicals introduced into our environment by indirect pathways, as referred to earlier, domestic birds as well as wild ones, rabbits, cats, and cattle have been biological indicators for the presence of contamination.

4.5.7 REFERENCES

Abou-Donia, M. D., Othman, M. A., Tantawy, G., Khalil, A. Z., and Shawer, M. F. (1974). Neurotoxic effect of leptophos. *Experientia*, **30**, 63–64.

Ahdaya, S. M., Shah, P. V., and Guthrie, F. E. (1976). Thermoregulation in mice treated with parathion, carbaryl, or DDT. *Toxic Appl. Pharmac.*, **35**, 575–580.

Baetjer, A. M. (1983). Water deprivation and food restriction on toxicity of parathion and paraoxon. *Archs. Envir. Hlth.*, **38** (3), 168–171.

Bakir, F., Damluji, S. F., Amin-Zaki, L., Murtadha, M., Khalidi, A., Al-Rawi, N. Y., Tikriti, S., Dhahir, H. I., Clarkson, T. W., Smith, J. C., and Dohert, R. A. (1973). Methylmercury poisoning in Iraq. *Science*, **181**, 230–241.

Case, A. A. (1974). Toxicity of various chemical agents to sheep. *J. Am. Vet. Med. Ass.*, **164**, 277.

Cheeke, P. R., and Shull, L. R. (1985). *Natural Toxicants in Feeds and Poisonous Plants*. AVI Publishing Co., Westport, CT.

Clarkson, T. W., Amin-Zaki, L., and Al-Tikriti, S. K. (1976). An outbreak of methylmercury poisoning due to consumption of contaminated grain. *Fed. Proc.*, **35** (12), 2395–2399.

Debackere, M. (1983). Environmental pollution: the animal as source, indicator, and transmitter. In: *Veterinary Pharmacology and Toxicology*, pp. 595–608. AVI Publishing Co., Westport, CT.

Galtier, P., and LeBars, J. (1983). Mycotoxin residue problem and human health hazard. In: *Veterinary Pharmacology and Toxicology*, pp. 625–640. AVI Publishing Co., Westport, CT.

Hassan, A., Abdel-Hamid, F. B., Abou-Zeid, A., Moktar, D. A., Abdel-Pazek, A. A., and Ibrahain, M. S. (1978). Clinical observations and biochemical studies of humans exposed to leptophos. *Chemosphere*, **7**, 283–290.

Hill, E. F., and Hoffman, D. J. (1984). Avian models for toxicity testing. *J. Am. Coll. Toxicol.*, **3** (6), 357–376.

Hodges, L. (1976). *Environmental Pollution*, 2nd edn, pp. 230–236. Holt, Rinehart and Winston, New York.

Ingram D. L., and Mount, L. E. (1975). *Man and Animals in Hot Environments*. Springer-Verlag, New York.

Irving, L. (1964). Maintenance of warmth in arctic animals. *Symp. Zool. Soc., London*, 13 : 1. Cited in: Yousef, M. K., (1985) *Stress Physiology in Livestock*, vol. II (chap. 10). CRC Press Inc., Boca Raton, FL.

Kenaga, E. E. (1978). Test organisms and methods useful for early assessment of acute toxicity of chemicals. *Environ. Sci. Technol.* **12**, 1322–1329.

Maguire, C. C., and Williams, B. A. (1987). Cold stress and acute organophosphorus exposure: interaction effects on juvenile northern bobwhite *Arch. Environ. Contam. Toxicol.* **16**, 477–481.

Metcalf, R. L. (1982). Historical perspective of organophosphorus ester-induced delayed neurotoxicity. *Neurotoxicol.*, **3** (4), 269–284.
National Research Council (1972). *Lead: Airborne Lead in Perspective*. National Academy of Sciences, Washington, D.C.
National Research Council (1979). *Interactions of Mycotoxins in Animal Production*. National Academy of Sciences, Washington, D.C.
National Research Council (1980). *Mineral Tolerance of Domestic Animals*. National Academy of Sciences, Washington, D.C.
National Research Council (1981). *Effect of Environment on Nutrient Requirements of Domestic Animals*. National Academy of Sciences, Washington, D.C.
Neathery, M. W., and Miller, W. J. (1975). Metabolism and toxicity of cadmium, mercury and lead in animals: a review. *J. Dairy Sci.*, **58** (12), 1767–1781.
Ordonez, J. V., Carrillo, J. A., Miranda, C. M., and Yale, J. L. (1966). Organic mercury identified as the cause of poisoning in humans and hogs. *Science*, **172**, 65–67.
Osweiler, G. D., Carson, T. L., Buck, W. B., and Van Gelder, G. A. (1985). *Clinical and Diagnostic Veterinary Toxicology*, 3rd edn. Kendall/Hunt Publishing Co., Dubuque, IA.
Rattner, B. A., Sileo, L., and Scanes, C. G. (1982). Hormonal responses and tolerance to cold of female quail following parathion ingestion. *Pestic. Biochem. Physiol.*, **18**, 132–138.
Ruckebusch, Y., Toutain, P. L., and Koritz, G. D. (1983). *Veterinary Pharmacology and Toxicology*. AVI Publishing Co., Westport, CT.
Schmidt-Nielsen, K. (1979). *Animal Physiology: Adaptation and Environment*, 2nd edn. Cambridge University Press, New York.
Selby, L. A., Case, A. A., Dorn, C. R., and Wagstaff, D. J. (1974). Public health hazards associated with arsenic poisoning in cattle. *J. Am. Vet. Med. Ass.*, **165**, 1010.
Shull, L. R., and Cheeke, P. R. (1983). Effects of synthetic and natural toxicants on livestock. *J. Anim. Sci.*, **57** (2), 330–354.
Smith, J. E. (1982). Mycotoxins and poultry management. *World's Poult. Sci. J.*, **38** (3), 201–212.
Walker, C. H. (1983). Pesticides and birds—mechanisms of selective toxicity. *Agric. Ecosystems and Environ.*, **9**, 211–226.
Yousef, M. K. (1982). *Animal Production in the Tropics*. Praeger Publishers, New York.
Yousef, M. K. (1985a). *Stress Physiology in Livestock*, vol. I. CRC Press Inc., Boca Raton, FL.
Yousef, M. K. (1985b). *Stress Physiology in Livestock*, vol. II. CRC Press Inc., Boca Raton, FL.