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4.1 *Effects of Temperature and Humidity on Ecotoxicology of Chemicals*

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4.1.1 INTRODUCTION

Quantification of the influence of temperature and humidity on xenobiotics and environmentally significant chemicals in general will greatly help in prescribing safety limits for community and occupational exposure in countries in the tropical region. The formulation of guidelines aimed at the protection of ecosystems from pollutants could also be more effective. Since eco-epidemiological data pertaining to this subject are limited, inferences have to be drawn from simulated experimental studies and case reports. The basis of the present overview is the information available from literature on related temperature and humidity effects and on the toxicity and environmental fate of pollutants.

The physiological effects of thermal and vapour pressure stresses have been very well studied under a wide variety of conditions (Broulia *et al.*, 1960; Hertig, 1975; Hunt, 1979; Gill, 1980; Alpaugh, 1982). However, it is not clear whether exposure to higher temperatures, with and without high humidity, affects the toxicity of environmental chemicals. Similarly, the influence of ambient temperature on environmental toxicology is not understood, in spite of a large number of reports on the physiological effects of temperature stress (Bhatia *et al.*, 1975).

4.1.2 GENERAL CONSIDERATIONS

The effects of temperature and humidity on air quality and visibility have been well studied. Air parcels with high humidity and temperature, which represent conditions conducive to the formation of sulphates, tend to produce lower visibilities (Sloane, 1983). Increased levels of SO_2 and SPM (suspended particulate matter) are implicated in this phenomenon. There is more interference of visibility by pollutants in summer than in winter (Linak and Peterson, 1983). The data on monitoring of air quality in Indian cities under the national monitoring and GEMS (Global Environmental Monitoring System) programmes

indicated high particulate content (SPM) with high levels of SO_2 characteristic of both Bombay and Calcutta. Data are also available on the content of polycyclic hydrocarbons in samples of ambient air from Ahmedabad and Bombay. SPM could provide a matrix for adsorption of SO_2 and NO_x and for complex photochemical reactions, the nature of which needs elucidation in hot humid climates. Seasonal variations, with higher SPM in summer, could alter toxic effects. The release of terpenes into the air of a pine forest was found to be higher in summer than in winter (Yokuchi *et al.*, 1983). Depending on temperature and humidity, these organics interact with atmospheric chemical substances and affect ozone levels.

Seasonal variations in precipitation chemistry, with lower sulphur in winter, have been reported. Nitrate and H^+ were, however, unaffected (Pratt and Krupa, 1983). These results make the study of climatic influences in ecotoxicology a priority area in tropical countries.

The foliar uptake of pesticide aerosols as tested with labelled 2,4,5-T was higher at 37°C than at 21°C or 30°C. Similarly, humidity influences the rate of translocation. From a study of the influence of 32°C and 20°C and relative humidity of 100% and 40% it was found that washable residues of glyphosate on leaves of *Cynodon dactylon* after 48 hours were least at low temperatures and low humidity. The high- temperature, high-humidity effect was mostly through enhanced translocation (Hartley and Graham-Bryce, 1980). However, the response varied with different chemicals and features of the plant. The effect of humidity and temperature on pesticide uptake by roots has also been reviewed by the above authors. The pattern was similar to that shown by leaves with the addition that soil characteristics also play a part. It may be pointed out that very little systematic work is available in published literature on this subject.

Temperature also affects the stability of insecticides. Carbamates persist in cold water for longer periods than in warm water, due to less hydrolysis (Aly and ElDib, 1972). Some information and a few mathematical models are available regarding the influence of temperature and humidity on the fate of air pollutants, their transport, and secondary transformation. Similarly, these aspects have received attention in aquatic environments as well as soil matrices. Since they are beyond the scope of this section, they are not covered here. It should be pointed out, however, that from mathematical models it is possible to extrapolate to specific conditions relevant to a particular habitat.

Aerial spraying of pesticides is carried out only in very limited areas. Most of the application is done manually using simple hand operated sprayers. A variety of chemicals are used and often the application is made repeatedly under conditions showing wide variations in temperature and humidity. Application of weedkillers to a plantation crop, like tea, presents its own problems related to downpour of rain in the tea-growing region. Data on residues have been documented to a certain extent, but practically nothing is known about the

dynamics of the chemicals in the ecosystem, especially the role of climate on biodegradation and biomagnification.

4.1.3 EFFECT OF TEMPERATURE AND HUMIDITY ON TOXICITY

Brown (1980) concluded that the relationship between ambient temperature and humidity and the toxic effects of pesticides in vertebrate animals is not linear. However, for man, it is presumed that there is an increased risk in hot climates due more to disinclination to wear protective garments than to the actual effects of higher intrinsic toxicity. The LD_{50} (intraperitoneal, mice) for parathion, carbaryl, and DDT at 1°C was 16.5, 263 and 750 mg/kg, respectively, whereas at 38°C the values were 11.3, 112, and 875. But at 27°C, the values were 29, 588, and 1175, showing the least toxicity at this temperature. Doull (1972) also suggested that ambient temperature may affect pesticide toxicity.

Cummings (1969) showed that penetration of N octylamine through skin is greater at higher temperatures. Craig *et al.* (1977) considered that the toxicity of cholinesterase inhibitors is higher in cold conditions, because depot formation is less at higher temperatures. In such cases, where temperature alters toxicity, Cornwall and Bull (1967) have suggested that body surface area rather than weight is the ideal basis for calculation of doses. Several organophosphates and carbamates show less *in vivo* inhibition of cholinesterase at higher ambient temperatures (Brown, 1980).

Even though very little systematic work has been done on pollutants one could draw analogy from the exhaustive work on drug toxicity versus temperature (Fuhrman and Fuhrman, 1961; Weihe, 1973). LD_{50} variations with temperature in animals could also be useful. Acute toxicity of these agents, which induces, among other signs, hyperthermy, increases with ambient temperature. Sympathomimetic amines, phenothiazines, and salicylate above and below a thermoneutral zone, show increased toxicity. Procaine and caffeine cause higher toxicity at higher temperatures, but toxicity is unchanged below thermoneutrality. The effects of atropine are more severe in hot humid zones. Thermal adaptation to environment (acclimatization) can also alter the toxic effects of drugs (Zbinden, 1973), suggesting the need for more detailed studies on the influence of geographical factors on toxicity.

One major effort in the above direction was the symposium 'Toxicology in the Tropics' at Ibadan, Nigeria (Smith and Bababunmi, 1980). The relatively high toxicity of chemotherapeutic agents used in large amounts for the treatment of parasitic diseases, under conditions of high humidity and temperature, was identified as a major problem in countries in the tropical belt. People in the tropics are more prone to toxicity by food toxins, like cyanogenetic glycosides, mycotoxins, nitrosamines, and lathyrogens.

People living in the humid tropical and semi-tropical countries have coexisted with a variety of myco- and phytotoxins. How far the traditional processing of

food by indigenous methods of fermentation (Koji fermentation in Japan/Korea/China; Idli/Kedli fermentation in Indonesia/Philippines/India) or dehydration modulate the properties and chemistry of these chemicals is not understood at the elementary level of investigation let alone as far as molecular mechanisms are concerned. One expects the half-lives of mycotoxins to be within time intervals which may not be consequential from the point of view of overall exposure. In contrast, some processing techniques might lead to adduct formations and acquisition of the property of recalcitrance. Ethnic variations in detoxification processes may also affect toxicity.

That humidity and temperature affect the toxic potential of environmental pollutants like lead, parathion, and antimony has been suggested by Baetjer (1968). Some information is available regarding the effect of temperature variations on the toxic response of cold-blooded animals. Rehwoldt *et al.* (1972) reported that the toxicity of mercurous ion in fish increased threefold when water temperature rose from 15°C to 28°C. However, the toxic effects of Cu, Zn, Ni, Cd, and Cr remained unchanged with an increase in ambient temperature. In an exhaustive review, Cairns *et al.* (1975) suggested that temperature affects toxicants through altered metabolism, increased diffusion, altered oxygen levels, or actual interaction with toxic process. With ammonia and several chemicals, although not all, toxicity increases with temperature. Hg accumulation in fish is also greater at higher temperatures. With pesticides, the data do not show a regular pattern. Higher temperature enhanced endrin toxicity to fish while that of DDT was decreased. With detergents, a rise in temperature increases toxicity. Thus temperature influence depends on the nature of the toxicant, the organism, and on water quality.

Suskind (1977) reported that higher ambient temperatures enhance the percutaneous absorption of chemicals. In the case of miners, the effect of varied conditions of temperature on physiological processes, health, and productivity have been exhaustively studied (Wyndham, 1970), but how far higher ambient temperature has an impact on the effects of simultaneous chemical stress is not clear. Also, the ethnic variations in heat stress deserve more detailed study in tropical countries.

Casarett and Doull (1975) pointed out that any investigation on the interrelationship between temperature and toxic response should include the study of the effect of the toxic agent on temperature regulation as well as the study of the environmental temperature on drug response. Generally, it can be said that the response of a biological system to a drug would be decreased with a decrease in environmental temperature. This is not always true with toxicants. Parathion toxicity is increased in hyperthermia but malathion is potentiated by cold exposure.

Keplinger *et al.* (1959) studied the acute toxicity of 58 compounds in rats at different ambient temperatures. Warfarin was less toxic at 26°C than at 8°C or 36°C. DNOC and pentachlorophenol became less toxic as the temperature

became progressively lower. DDT had the same effect at 8°C and 26°C but was more toxic at 36°C. Strychnine was less toxic at 26°C than 8°C and 36°C, which elicited similar responses. LD_{50} of the rodenticide ANTU to rats was 1.9, 2.9, 4.0, and 1.2 mg/kg at 37, 48, 72, and 89°F (Meyer and Karel, 1948).

Variations in temperature and intensity, quality, and duration of light are well-known modulators in plant physiology. There is evidence to show that higher ambient temperature influences the response of plants to air pollutant toxicity (Heck and Dunning, 1967).

Climatic factors also affect toxic residue dissipation in plant tissues, as evident from the study on parathion in citrus orchards (Gunther *et al.*, 1977). With a temperature range of 50–100°F, it was found that residue in leaves dissipated faster at higher ambient temperatures. Similarly, leaf and soil residues in ethion sprayed grape crops decreased more rapidly at higher temperatures. Seasonal and diurnal variations in temperature also affected urinary *p*-nitrophenol in parathion sprayers. Higher temperatures enhance the excretion of dermally exposed parathion, in spite of higher absorption.

Another instance of temperature affecting toxicity is the recovery of grasshoppers exposed to DDT when the temperature rises from 20°C to 25°C. Also, ppb levels of DDT enhance the lower lethal temperature for certain fish, changing their cold resistance (Holdgate, 1979). The lethality of caffeine to rats maintained at room temperature of 84–89°F as compared to 74–79°F, caused hyperpyrexia in addition to the usual caffeine toxicity symptoms (Boyd, 1972).

Wolfe *et al.* (1961) observed temperature dependence in the toxicity of dinitro-*o*-cresol(DNOC) which also increases body temperature of exposed humans at ambient temperatures above 22°C and decreases it below 26°C (Hayes, 1963). The toxicity of alphachloralose to birds and rodents also showed variation with ambient temperature (Brown, 1980).

Many toxicological studies on fish indicate increased toxicity with temperature. Studies at the sublethal and lethal levels have shown that the rate of uptake for water-borne substances, such as lead, mercury, and zinc, will increase with temperature and that the time of death will be advanced (Somero *et al.*, 1977; MacLeod and Pessah, 1973). The bioconcentration factor or amount accumulated has been shown to increase with temperature for mercury and DDT (Cember *et al.*, 1978; Boudou *et al.*, 1980; Reinert *et al.*, 1974). In contrast, studies on other organic contaminants have shown toxicity to decrease with increasing temperatures (Kumaraguru and Beamish, 1981; Brown *et al.*, 1967). An assessment of the influence of temperature on contaminant toxicity to fish indicates that this response will increase over a wide temperature range; but there are some exceptions for different substances, particularly organic contaminants, which can vary among species.

Acute toxicities of environmental pollutants, such as organic solvents, heavy metals, and agricultural chemicals, are known to be aggravated at higher or lower environmental temperatures (Nomiyama *et al.*, 1980a). Environmental

temperature has been known to modify acute toxicities of environmental pollutants. Parathion, lead, and 1,1,1-trichloroethane have been reported to be more toxic under high environmental temperatures (Baetjer and Smith, 1956; Baetjer *et al.*, 1960; Horiguchi and Horiguchi, 1966; Horiguchi *et al.*, 1979). Acute toxicities of benzene, trichloroethylene, mercuric chloride, cadmium chloride, fratol, methyl parathion, and dieldrin increased markedly under low environmental temperatures and were also enhanced at a high temperature (Nomiyama and Nomiyama, 1976; Nomiyama *et al.*, 1980a). However, acute toxicities of toluene, copper sulphate, and chromium trioxide were enhanced at a high temperature of 38°C. Toxicity of beryllium was also enhanced under environmental temperatures of 8°C and 38°C. Higher temperatures, furthermore, aggravated cadmium induced testicular haemorrhage (Matsui and Nomiyama, 1979). Acute toxicity of methyl mercury was aggravated under the environmental temperature above 80°F. The acute toxicity of methyl mercury at 38°C was higher than at 22°C (Nomiyama *et al.*, 1980b). Yamanouchi *et al.* (1967) reported that the body temperature of mice, which were fixed on a board, decreased in a cold room at 10°C but that the body temperature of five mice kept in one cage remained unchanged because they warmed each other. Yamaguchi *et al.* (1984) studied the effects of environmental temperatures on the toxicity of methyl mercury in rats and observed that high temperatures can result in increased mortality and neurotoxicity, and even low temperatures may result in some increases in mortality in comparison to room temperature. Neurotoxicity due to mercury poisoning can be responsible for further dysfunction due to its effect on food and ingestion during heat and cold stress.

The environmental temperature can influence the actions of drugs and chemicals in warm-blooded animals (Farris and Griffith, 1949).

A considerable amount of work has been done in the USSR on the effect of ambient temperature, humidity, and other factors (Filov *et al.*, 1978). Temperature affects toxicity, through impaired heat regulation, water loss, respiratory/circulatory disturbances, basal metabolism changes, and altered individual reactions. Increase or decrease in toxicity with temperature varies with the chemical nature of the toxin. The toxicity of narcotics, nitrogen oxides, mercury, petroleum solvents, trichlorfor, methyl styrene, CO, thiotic poisons, etc. is affected by higher temperature. Ambient temperature may affect toxicokinetics also.

Filov *et al.* (1978) include a 20 page table in Russian, 'Combined action of industrial poisons and elevated ambient temperature'. Generally, higher temperature enhances toxicity and accelerates onset. The exception was silicosis, which is alleviated in animals at higher temperature. Aniline inhalation was more toxic at temperatures above 35°C in rats but not in dogs. The effect of higher temperature on CO toxicity was different for guinea pigs, rats, and rabbits. Temperature effect was more marked in chronic toxicity.

The combined effects of heat and toxicant can be taken as a mutual aggravation syndrome. Such effects have to be taken into account while fixing

MAC (Maximum Allowable Concentration) values. It was suggested that MAC for pesticides should be reduced by a factor of 5–10 in hot climates. Also, the temperature range of minimum toxicity varies for different substances and can be worked out. Type I narcotics are generally hydrophilic and their toxicity decreases at higher temperature, while the reverse is the case with hydrophobic type II narcotics.

According to Jahnke (1957), there is no drug whose actions in man are more influenced by climatic conditions than atropine.

4.1.4 EFFECT OF TEMPERATURE ON PLANTS

Indirect injury may occur at a temperature just below the point of denaturation (Langridge, 1963). As the temperature increases, the reaction rates and metabolic activity increase proportionately. As temperatures approach 30°C, the metabolic rate can be very high. Cellular damage at high temperatures may also result from the formation of toxic substances in certain cells exposed to a high localized temperature. The toxic material may subsequently be translocated to other parts of the plant and cause widespread injury (Yarwood, 1961).

Beevers and Cooper (1964) grew rye plants under various temperature regimes. They found that plants growing continuously at 12°C had a higher carbohydrate and nitrogen content than similar plants grown in warmer regimes. The relatively high carbohydrate level was considered to result from the slower degradation of carbohydrates caused by decreased respiration at lower temperatures. The young stem developing early in the growing season may grow best at one temperature while later growth stages and reproduction require different temperatures (Went, 1953).

4.1.5 HUMIDITY

High ambient humidity causes swelling of the stratum corneum so that penetration of chemicals through skin is enhanced (Suskind, 1977). Lindquist *et al.* (1982) reported that the interconversion of nitrogen oxides in the atmosphere is influenced also by humidity and temperature, which may be significant in photochemical smog.

Without moisture in the atmosphere, there would be no corrosion of materials, even in the most heavily polluted air (MacCormick and Holzworth, 1976). In the formation of secondary pollutants, like sulphate, which are more toxic, moisture plays a role (Wagman *et al.*, 1967).

Humidity also affects toxicity by forming more irritant products from the pollutants, for example nitric acid from NO_2, and HCl or Cl_2 from labile chlorinated organics.

The effects of exposure to cold and hot occupational environments have been exhaustively reviewed by Horvath (1979). Relative humidity and soil moisture

have been noted to exert a marked effect upon the sensitivity of plants to phytotoxic air pollutants, plants grown under drought conditions being less sensitive (Heck *et al.*, 1965).

Environmental conditions causing full opening of stomata cause more severe air pollution phytotoxicity. A mixture of SO_2 and O_3 was more toxic in high humidity than low (Carlson, 1979). At 55–90% relative humidity, rates of photosynthesis by maple and white ash leaves were reduced by 67% and 58% in 1 day, by 50 pphm O_3 and 50 pphm SO_2. The corresponding figures for 20–50% humidity were 26% and 60%. Subsequent periods of fumigation were also different. Thus different plants may vary in their response.

The influence of humidity on nicotine toxicity on i.p. (intraperitoneal exposure indicates factors other than absorption (Brown, 1980).

4.1.6 COMBINED EFFECTS

It is well known that higher temperature and humidity have a role in enhancing the release and affecting the transport of aeroallergens and other viable particulates (Jacobson and Morris, 1976). This is of great concern for health in tropical countries due to the abundance of sources of biological pollution and human, plant, and animal pathogens.

Baetjer (1968) has reviewed the effect of climatic factors on toxicity of chemicals. Since the bulk of lead poisoning in children takes place in summer, lead toxicity of rats was studied after ip/iv (intraperitoneal or intravenous) injections. Death rate was higher at 95°F than at 72°F. In the absence of sweat glands, humidity was indirectly tested by withdrawal of water, which increased mortality. Lowering temperature at night reduced the effects. Similar results were also obtained with mice. Urinary lead decreased at higher temperatures indicating higher retention. The temperature effect on Pb was independent of its effect on normal physiology. With parathion also, death rate was higher and survival time shorter in mice at 96°F than 73°F. The effect of temperature variation was even more rapid than with lead. On the basis of these data and of those on antimony and benzol, it has been concluded that high environmental temperature generally increases susceptibility to toxic chemicals. In the case of inhaled chemicals, environmental temperature and humidity could affect the ciliomucous clearance mechanism, thereby enhancing toxicity. This was established from the clearance of intratracheally injected ^{131}I in chicks exposed to variation from 40°F to 95°F and 3 mm to 39 mm Hg vapour pressure. Higher temperature may also stimulate cutaneous blood flow and also enhance skin reaction to irritant chemicals. Excessive sweating, when not evaporated due to high ambient humidity, could lead to hydration of skin thereby increasing penetration of chemicals. Sweat may also dissolve many chemicals settled on skin. Further, high temperature may enhance the vapour pressure of chemicals,

increasing the risk of exposure. Thus higher temperature and humidity could enhance chemical toxicity (Baetjer, 1968).

By measuring urinary *p*-nitrophenol in humans exposed to parathion, Funkes *et al.* (1963) showed that dermal absorption increased directly with ambient temperature, from 14.4 to 40.5°C. Other cases of temperature and toxicity of pesticides are listed by Brown (1980).

Crayfish can withstand mercury in water better at lower than higher temperatures due to less intake and to altered metabolic activity (Heit and Fingerman, 1977).

4.1.7 THEORETICAL CONSIDERATIONS

In the absence of adequate experimental studies, the mechanisms governing the alteration in toxic response as a function of temperature and humidity can best be understood from conceptual logic based on theoretical considerations. In most cases higher temperature enhances and accelerates toxicity. Filov *et al.* (1978) considered that the temperature component of the environment acted indirectly, modifying the functional state of the organism. Water loss, impaired thermoregulation, and higher breathing and blood flow may lead to larger amounts of toxicant entering and being transported to target loci. The mutual aggravation syndrome with temperature and toxicants, implies that MAC for pesticides in hot climates should be 10–20% of that elsewhere. Temperature controls the physico-chemical properties of the toxicants. Ballard (1974) has considered the theoretical aspects of temperature variation on toxicokinetics and stated that temperature could also weaken protein binding of toxicants. How far other views based on experimental animal studies are applicable to ecotoxicology is not clear. Similarly, high humidity can enhance irritancy of chemicals, such as nitrogen and sulphur oxides, forming the acids.

The influence of thermal stress on ecosystems has been well studied, mostly from the pollution by heated effluents (Laws, 1981). When toxicant stress is superimposed, the combined effect could vary. The median tolerance level of fish to copper is known to be lower at higher temperatures (EPA, 1971). The response could be attributed to faster metabolic rate, toxicant mobilization, and lesser oxygen availability at higher temperatures so that toxicity of a particular dose becomes more serious (Metelev *et al.*, 1971). The toxicity of cyanide, metals, and phenol becomes faster and more drastic at higher temperatures. The bioaccumulation of mercury in fish is also enhanced by higher ambient temperatures (Reinert *et al.*, 1974; Cember *et al.*, 1978; Kumaraguru and Beamish, 1981). The enhanced toxicity to fish of a xenobiotic at higher temperature is also indicated by a behavioural response with SO_2 at higher temperatures (Burton *et al.*, 1978). Alterations in biotransformation by liver of polychlorinated benzene and naphthoflavon exposed fish were caused by variations in temperature (Foertin *et al.*, 1983). As in the case of fauna, the

toxicity of mercury to algae was also found to be higher at elevated temperatures (Huisman *et al.*, 1980). Also, in the case of insects, there are a few reports suggesting greater toxicity at higher temperature, DDT being a notable exception (Sun, 1963).

4.1.8 QUANTITATIVE STRUCTURE ACTIVITY RELATIONSHIP (QSAR)

The QSAR approach has been found to be a useful tool in environmental toxicology, as well as in occupational toxicology and pharmacology (Kaiser, 1983). Since the octanol-water partition coefficient, which is an important parameter in QSAR ecotoxicology, is likely to be influenced by temperature, once the effect of temperature of one compound is understood it may be possible to calculate it for others. Similarly, by suitable models, it may be possible to extrapolate effects from one temperature to another. Recently, the QSAR approach has been applied in predicting responses in environmental toxicology (Birge and Cassidy, 1983; Black *et al.* 1983). In cold-blooded animals the influence of ambient temperature variation on toxicokinetics can be extrapolated from experimental studies more directly than in warm-blooded animals. As such, some of the models suggested for aquatic species (Hermens *et al.*, 1985; Lipnick *et al.*, 1985; Bobra *et al.*, 1985) can be modified to include the temperature component.

4.1.9 TEMPERATURE, CARBON DIOXIDE AND PHOTOSYNTHESIS

Considerable information is available on the possible climatic changes caused by increasing levels of CO_2 due to uncontrolled fossil fuel burning (Clark, 1982). How far increased temperature affects the phytotoxicity of xenobiotics is not fully understood. In the case of acid deposition hotter and wetter climates promote soil acidification and oxygen deficiency leading to root injury (Ulrich, 1983).

One finding of significance could be the differences in the effect of temperature on the CO_2 assimilation capacity of C_3 and C_4 plants. At usual levels of CO_2, C_4 plants are less affected than C_3 plants by higher temperature. But on a threefold increase in CO_2, the pattern becomes similar (Cooper, 1982). It may be that at higher temperatures there is better tolerance to higher CO_2 concentrations, or vice versa.

4.1.10 TEMPERATURE AND ECOTOXICOLOGY OF POLLUTANTS

The effects of elevated temperatures on ecosystems and individual species have been fairly well understood (Connell and Miller, 1984). The decrease in dissolved oxygen, apart from direct effect on species, could also influence their response to toxicants. The biophysical aspects of the influence of temperature on various

species have been discussed by Gates (1981). Even though the influence of high temperature on insect physiology is well understood, data relating to pesticidal response are limited. However, such information is vital in biological monitoring of pollutants and their effects. The solubility of several organochlorine insecticides in water increases with ambient water temperature, leading to greater uptake and possibly higher toxicity to aquatic biota (Phillips, 1980). Studies with DDT and mosquito fish, and rainbow trout support this. Similarly, the uptake of several toxic metals by different higher and lower aquatic fauna was found to be increased with temperature. Cairns *et al.* (1975) have reviewed the information on the effect of temperature on the toxicity of various pollutants to aquatic biota. Whereas *Daphnia magna* had similar responses to phenol, chlorobenzene, diethanol, amine, and ethylene glycol at 20°C and 24°C, *Ceriodaphnia* was distinctly more sensitive at the higher temperature (Cowgill *et al.*, 1985). Cooney *et al.* (1983) also observed temperature to influence toxicity in lower fauna.

4.1.11 ENVIRONMENTAL SIGNIFICANCE

The influence of ambient temperature and humidity on the toxicity of an environmental pollutant may have diverse practical significance. Hot and humid climates favour breeding of insects, parasites, rodents, and noxious weeds so that an effective strategy for control is needed. This has to include any superimposed effect of climate on toxicity to both target and non-target species, while designing the nature, dose, mode, and time of applying biocides. The effect of climatic extremes on toxic response may vary from species to species, at least quantitatively if not qualitatively. In that case, if any non-target species is likely to be specifically sensitive, such situations could be anticipated and avoided. Further, if higher temperature and humidity influence the safety of a plant protection chemical, a severe situation could arise during situations of drought. In the context of polyclonal natural forest ecosystems, the interrelationship between climate and xenobiotics has to be such that the natural balance is not altered. The same situation could also apply to other ecosystems when under chemical stress. Where the non-target species is mostly grown in monoculture, as in commercial agriculture, sylviculture, or pisciculture, ideal situations can be developed using seasonal variation as a means to reduce sensitivity to the chemical. The pattern of use of pesticides, fertilizers, and growth parameters can be guided by data on climatic influence. Since there is a paucity of experimental and field studies on this, it is a priority area for ecotoxicologists.

4.1.12 CONCLUDING REMARKS

Epidemiological and experimental data are very limited on any superimposed effect of high temperature or humidity, as prevalent in tropical climates, on

the toxicity of occupational and environmental xenobiotics. From available information it is clear that there are chances of increased risk. Therefore, in-depth research in this direction is needed for the assessment and abatement of problems of environmental toxicology in tropical countries.

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