ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 5.2
IJAR 2015; 1(10): 494-500
www.allresearchjournal.com
Received: 30-07-2015
Accepted: 31-08-2015

Shalini Sharma
Assistant Professor, Graduate Government College,
Faridabad, Haryana, India

Correspondence
Shalini Sharma
Assistant Professor, Graduate Government College,
Faridabad, Haryana, India

Modeling and analysis of a biological population: effects of industrilization, toxicants emitted from external sources as well as formed by precursors

Shalini Sharma

Abstract
In this paper, a non-linear mathematical model is proposed and analyzed to study the effects of industrialization, toxicant which is emitted into the environment from various external sources at a constant rate and whose concentration is augmented due to transformation of a precursor produced by the species. It is shown that if the rate of emission of toxicant and rate of its transformation from precursor into the environment increases, the density of the biological population settles down to a lower equilibrium than its original carrying capacity and its magnitude decreases as the equilibrium level of concentration of toxicant and industrialization increases. It is pointed out that for very large emission and transformation rates of the toxicant from the above mentioned processes, the survival of the biological species is threatened.

Keywords: Modeling, biological population, effects of industrilization

1. Introduction

With rapid increase in industrialization and development the resource on which the biological population depends is highly affected. This type of industrialization may be augmented by human actions (precursors). The term "precursor" is generally used to represent an intermediate product produced by a living species (e.g. human population), which may get converted into a toxic substance in the environment harmful to itself as well as to other species living in the same habitat.The effects of human activities and patterns of resource use on the structure and composition of resource have been studiedby many investigators, Bormann and Likens (1979), Padoch and Vayda (1983) [12], Hammond (1990), Brown (1992), Garcia-Montiel and Scatena (1994) [4], Woodwell (1970) [22], McLaughlin (1985) [11], Hari *et al*. (1986), Woodman and Cowling (1987) [23], Schulze (1989) [15].

A typical case of toxic effect where airborne acid and various kind of dusts formed from precursors (e.g. gases, dust produced by industrial units) affect the human lung causing different types of diseases, Holma (1985), Folinsbee (1989).

Generally the biological species is affected by toxicants through different pathways including uptake of toxicants from the environment or through the external waste dumped. In the case of less toxic substances, when the plants are exposed to them for longer durations, these are uptaken through various means, the toxicant in the uptake phase interacts with tissues through physiological processes, which in turn makes it diseased. Various investigators have studied effects of toxicants on biological species using mathematical models, Hallam and Clark (1982) [5], Hallam *et al*. (1983 a, b) [6, 7], Hallam and De Luna (1984), De Luna and Hallam (1987) [1], Freedman and Shukla (1991) [2], Huaping and Ma (1991) [9], Shukla and Dubey (1996, 1997). In particular, Hallam and his co-workers (1982, 1983, 1984, 1987) proposed and analyzed mathematical models to study effects of toxicants on the biological populations when these are emitted in to the environment from external sources. Huaping and Ma (1991) [9] proposed a mathematical model to study effect of a toxicant/pollutant on two competing species populations. In these models, the simultaneous effects of industrialization, toxicant as well as precursor on growth rate and carrying capacity of the species have not been considered. However, Freedman and Shukla (1991) [2] proposed models to study effects of a toxicant on single species and predator prey systems by assuming that the intrinsic growth rate of species decreases as the uptake concentration of toxicant increases in the species population while its carrying capacity decreases with its environmental concentration. Shukla and Dubey (1996) [17] have also studied the effects of two toxicants on a biological species by using the same assumptions (see also Shukla and Dubey (1997) [18]. Shukla *et al*. (2001) [16] have further studied effect of a toxicant on the existence and survival of two competing species in a polluted environment.

It may be noted here that in these studies toxicants are emitted from external sources into the environment with constant rates affecting the species and the emission rates have no relation with species population density. In real situations, however, toxicants (pollutants) are emitted into the environment by various human actions (e.g. industrial, house hold, vehicle discharges, etc.) either directly or formed by its precursors which are population density dependent. Resigno, (1977) [14] has modeled and analyzed the effect of a toxicant, which is formed only by precursor produced by species and affecting itself directly. Keeping in view the above, in this paper, the survival of a biological species, such as plants, is modeled and analyzed by considering the effects of industrialization, toxicant, which is emitted in the environment with a constant rate as well as formed by precursor produced by the human population.

In the modeling process, the following assumptions are made:

1. The density of biological species is assumed to be governed by a generalized logistic equation with variable intrinsic growth rate and carrying capacity.
2. The growth rate of the density of precursor is proportional to species population density and it decreases with its transformation into the environmental toxicant.
3. The concentration of toxicant in the environment decreases due to its assimilation (uptake, absorption, etc.) by species population, the amount being proportional to its density as well as its environmental concentration.
4. The environmental concentration of toxicant decreases the carrying capacity of the population density in the habitat.
5. The concentrations of toxicant in the environment as well as in its uptake phase decrease due to natural factors by an amount, which is proportional to its concentration in these phases.

2. Mathematical Model

We consider a biological population growing logistically in its habitat, which is affected by industrialization, toxicant produced by it, emitted into the environment by some external sources and the concentration of which is augmented due to transformation of a precursor produced by this population. It is assumed that the rate of increase of the precursor density is proportional to the density of the population producing it.

Keeping in view the considerations and assumptions as mentioned above the problem is assumed to be governed by the following differential equations

$$
\begin{aligned}
\frac{dN}{dt} &= rN - \frac{r_0 N^2}{K} + \beta_1 NB \\
\frac{dB}{dt} &= sB - \frac{s_{10} B^2}{L} - \beta_2 NB - s_1 IB - s_2 IB^2 \\
\frac{dI}{dt} &= \lambda_1 N + \mu IB - \theta_1 I \\
\frac{dP}{dt} &= \lambda N - \lambda_0 P - \theta P \qquad (1) \\
\frac{dT}{dt} &= Q + \pi_1 \theta P - \delta T - \alpha BT \\
& N(0) \geq 0,\ B(0) \geq 0,\ P(0) \geq 0,\ T(0) \geq 0,\ I(0) \geq 0,\ \text{and } 0 \leq \pi \leq 1
\end{aligned}
$$

Here, $N(t)$ is the density of biological species with growth rate r and carrying capacity K, $B(t)$ is the density of resource biomass density with growth rate s and carrying capacity L, $I(t)$ is the density of industrialization produced by the population, P is the density of precursor and $T(t)$ is the concentrations of toxicant/pollutant in the environment and in the species population respectively for any $t > 0$. In (1) Q is the cumulative emission rate of a toxicant into the environment from external sources. β_1 is the growth rate of N and β_2 is the depletion rate of B, the constant λ_1 is the growth rate coefficient of industrialization formed by population and θ_1 is the natural depletion rate coefficient of industrialization. The coefficient $\mu \geq 0$ is the growth rate of I, s_1 is the depletion rate coefficient of the biomass by industrialization and s_2 is the coefficient causing biomass depletion due to crowding of industrial activity. The constant λ is the growth rate coefficient of precursor formed by population, λ_0 is the natural depletion rate coefficient of precursor and θ is the fraction of the precursor, part of which is used in forming the same toxicant. The constant π_1 is the coefficient of augmentation of the

concentration of the toxicant being emitted in to the environment. The constant δ is the depletion rate coefficient of toxicants in the environment, α is the rate of depletion of pollutant in the environment.
In our model (1) the coefficient r and s represent the intrinsic growth rate constants of the consumer species and the resource species respectively. The constants K and L denote the carrying capacities of population and the resource respectively. Also, it can be seen that if industrialization is caused only by the population species then also the model is meaningful.

3. Equilibrium analysis

The given model (1) has two non-negative equilibria in $N-B-P-T-U$ space, namely $E_0 = \left(0,0,0,{}^{Q}\!/_{\delta},0\right)$, $E_1 = (0, B, 0, T, I)$ and $E^*(N^*, B^*, P^*, T^*, I^*)$. Existence of E_0 and E_1 is obvious. We show the existence of E^* as follows. Here N^*, B^*, P^*, T^* and I^* are the positive solutions of the algebraic equations.

$$N = K\left[\frac{r+\beta_1 B}{r_0}\right]$$

$$B = L\left[\frac{r-k\alpha T}{r_0}\right]$$

$$I = \frac{\lambda_1 N}{(\theta_1 - \mu B)}$$

$$P = \frac{\lambda N}{(\lambda_0 + \theta)}$$

$$T = \frac{\left(Q + \pi_1\theta\left(\frac{\lambda N}{(\lambda_0+\theta)}\right)\right)(\phi + \nu B)}{f(B)} = g(B)$$

where, $\theta > \mu B^*$

Rewriting 2nd equation of the model using above notations we have

$$\frac{dB}{dt} = (s - \beta_2 N - s_1 I)B - \left(\frac{s_{10}}{L} + s_2 I\right)B^2$$

In this equation $(s - \beta_2 N - s_1 I)$ denotes the intrinsic growth rate of $\frac{dB}{dt}$, which would increase only if $(s - \beta_2 N - s_1 I) > 0$ for $B \geq 0$ and $I \geq 0$.
For $B = 0$ we then have

$$s - \frac{\beta_2 rK}{r_0} - s_1 I(B=0) > 0$$

To prove the existence of E^*, let us take

$$F(B) = s_{10}B + s_2 ILB - sL + \beta_2 NL + s_1 IL$$

Then $F(0) = -L\left[s - \frac{\beta_2 rK}{r_0} - s_1 I(B=0)\right] < 0$,

Also,

$$F\left(\frac{sL}{s_{10}}\right) = L\left[\frac{ss_2}{s_{10}} I\left(\frac{sL}{s_{10}}\right) + \beta_2 N\left(\frac{sL}{s_{10}}\right) + s_1 I\left(\frac{sL}{s_{10}}\right)\right] > 0$$

Therefore, their exists a root B^* in the interval $0 < B^* < \left(\frac{sL}{s_{10}}\right)$ which is obtained by solving, $F(B^*) = 0$.

For B^* to be unique, we must have $F'(B) > 0$, where $0 < B^* < \left(\frac{sL}{s_{10}}\right)$

$$F'(B) = s_{10} + s_2 IL + s_2 BL \frac{dI}{dB} + \beta_2 L \frac{dN}{dB} + s_1 L \frac{dI}{dB}$$

Or

$$F'(B) = s_{10} + L\left[s_2 I + s_2 B \frac{dI}{dB} + \beta_2 \frac{dN}{dB} + s_1 \frac{dI}{dB}\right]$$

It is noted that at $B = B^*$, $F'(B^*) > 0$.

Thus, the condition for unique and positive B^* is $F'(B) > 0$. Once B^* is determined N^* and I^* can be found from equations.

These show that the biological population density decreases as the concentration of the toxicant in the environment increases.

4. Stability analysis

Theorem: - Let the following inequalities hold-

$$\lambda_1^2 < \frac{r_0(\theta_1 - \mu B^*)}{K}$$

$$\lambda^2 < 2\frac{r_0(\lambda_0 + \theta)}{K}$$

$$(\alpha T^*)^2 < \frac{\beta_1}{\beta_2}\left(\frac{s_{10}}{L} + s_2 I^*\right)(\delta + \alpha B^*)$$

Where, $\theta_1 > \mu B^*$

Then E^* is locally asymptotically stable.

Proof: - Using the following Liapunov's function for the linearlized system (1).

$N = N^* + n$, $B = B^* + b$, $I = I^* + i$ $P = P^* + p$ and $T = T^* + t$

Where, n, b and i are small perturbations around E^*,

We get

$$\dot{n} = \frac{-r_0 N^*}{K} n + \beta_1 N^* b$$

$$\dot{b} = -\beta_2 B^* n - \left(\frac{s_{10} B^*}{L} + s_2 I^* B^*\right) b - \left(s_1 B^* + s_2 B^{*2}\right) i$$

$$\dot{i} = \lambda_1 n + \mu I^* b - (\theta_1 - \mu B^*) i$$

$$\dot{p} = \lambda n - (\lambda_0 + \theta) p$$

$$\dot{t} = -\alpha T^* b + \pi_1 \theta p - (\delta + \alpha B^*) t$$

where, $\theta_1 > \mu B^*$

To study the local stability of E^*, we consider the following positive definite function,

$$V = \frac{1}{2}\frac{C_1 n^2}{N^*} + \frac{1}{2}\frac{C_2 b^2}{B^*} + \frac{1}{2} C_3 i^2 + \frac{1}{2} C_4 p^2 + \frac{1}{2} C_5 t^2$$

Which on differentiation gives

$$\dot{V} = \frac{C_1 n\dot{n}}{N^*} + \frac{C_2 b\dot{b}}{B^*} + C_3 i\dot{i} + C_4 p\dot{p} + C_5 t\dot{t}$$

On substituting the values of $\dot{n}$, $\dot{b}$ and $\dot{i}$ and we get.

$$\dot{V} = \frac{-C_1 r_0 n^2}{K} - C_2\left(\frac{s_{10}}{L} + s_2 I^*\right)b^2 - C_3\left(\theta_1 - \mu B^*\right)i^2 - C_4\left(\lambda_0 + \theta\right)p^2 - C_5\left(\delta + \alpha B^*\right)t^2 + \left(C_1\beta_1 - C_2\beta_2\right)nb$$
$$+ \left(C_3 \mu I^* - C_2\left(s_1 + s_2 B^*\right)\right)bi + C_3 \lambda np - C_5 \alpha T^* bt + C_5 \pi_1 \theta pt$$

As $\theta_1 > \mu B^*$

Choosing the values of the as

$$C_1 = 1,\; C_2 = \frac{\beta_1}{\beta_2},\; C_3 = \frac{\beta_1\left(s_1 + s_2 B^*\right)}{\beta_2 \mu I^*}$$

The equation reduces to,

$$\dot{V} = \frac{-rn^2}{K} - \frac{\beta_1}{\beta_2}\left(\frac{s_{10}}{L} + s_2 I^*\right)b^2 - C_3\left(\theta_1 - \mu B^*\right)i^2 - C_4\left(\lambda_0 + \theta\right)p^2 - C_5\left(\delta + \alpha B^*\right) + C_3 \lambda_1 ni$$
$$+ C_4 \lambda np - C_5 \alpha T^* bt + C_5 \pi_1 \theta pt$$

For to be $\dot{V}$ negative definite the following conditions must be satisfied,

$$\lambda_1^2 < \frac{r_0\left(\theta_1 - \mu B^*\right)}{K}$$
$$\lambda^2 < 2\,\frac{r_0\left(\lambda_0 + \theta\right)}{K}$$
$$\left(\alpha T^*\right)^2 < \frac{\beta_1}{\beta_2}\left(\frac{s_{10}}{L} + s_2 I^*\right)\left(\delta + \alpha B^*\right)$$

Lemma: The set

$$A = \left\{ (N, B, I, P, T) : 0 \le N \le N_m, 0 \le N \le N_m, 0 \le (I + P + T) \le \frac{Q + \frac{K}{r}\left(r + \beta_1 \frac{sL}{s_{10}}\right)}{\phi_1} \right\}$$

Where, $\lambda_1 = \min\left(\lambda_0, \theta, \pi_1\right)$ attracts all solutions initiating in the positive orthant.

Proof: From the model we have,

$$\frac{dB}{dt} = sB - \frac{s_{10} B^2}{L} - \beta_2 NB - s_1 IB - s_2 IB^2$$
$$\le sB - \frac{s_{10} B^2}{L}$$
$$0 \le B \le \frac{sL}{s_{10}} = B_m$$

Also,

$$\frac{dN}{dt} \le rN - \frac{r_0 N^2}{K} + \beta_1 N \frac{sL}{s_{10}}$$
$$\le \left(r + \beta_1 \frac{sL}{s_{10}}\right)N - \frac{r_0 N^2}{K}$$
$$0 \le N \le \frac{K}{r}\left(r + \beta_1 \frac{sL}{s_{10}}\right) = N_m$$

Further, $\frac{dI}{dt} + \frac{dP}{dt} + \frac{dT}{dt} = \lambda N - \left(\lambda_0 + \theta + \pi_1 \theta\right)P + Q + \left(\delta + \alpha B\right)T - \left(\theta - \mu B\right)I$

$$\leq Q + \lambda N_m - \lambda_1 P + (\delta + \alpha B)T - (\theta - \mu B)I \quad \text{Where, } \lambda_1 = \lambda_0 + \theta + \pi_1 \theta$$
$$\leq Q + \lambda N_m - \lambda_1 P + (\delta + \alpha B)T - (\theta - \mu B)I$$
$$\leq Q + \frac{K}{r}\left(r + \beta_1 \frac{sL}{s_{10}} \right) - \phi (I + P + T)$$
$$\Rightarrow 0 \leq (I + P + T) \leq \frac{Q + \frac{K}{r}\left(r + \beta_1 \frac{sL}{s_{10}} \right)}{\phi_1}$$

Theorem: In addition to the above assumptions that growth rate r and s and the carrying capacity are assumed to be constant. Let the functions s and L satisfy in A. Then if the following conditions hold

$$\lambda_1^2 < \frac{r_0(\theta_1 - \mu B^*)}{K}$$
$$\lambda^2 < 2\frac{r_0(\lambda_0 + \theta)}{K}$$
$$(\alpha T^*)^2 < \frac{\beta_1}{\beta_2}\left(\frac{s_{10}}{L} + s_2 I^* \right)(\delta + \alpha B^*)$$

Then E^* is non linearly asymptotically stable in A.

Proof: Consider the following positive definite function around E^*.

$$W(N, B, I, P, T) = C_1\left(N - N^* - N^* \ln \frac{N}{N^*} \right) + C_2\left(B - B^* - B^* \ln \frac{B}{B^*} \right) + \frac{1}{2}C_3(I - I^*)^2$$
$$+ \frac{1}{2}C_4(P - P^*)^2 + \frac{1}{2}C_5(T - T^*)^2$$

Differentiating with respect to t

$$\dot{W}(N, B, I, P, T) = C_1\left(\frac{N - N^*}{N} \right)\frac{dN}{dt} + C_2\left(\frac{B - B^*}{B} \right)\frac{dB}{dt} + C_3(I - I^*)\frac{dI}{dt}$$
$$+ C_4(P - P^*)\frac{dP}{dt} + C_5(T - T^*)\frac{dT}{dt}$$

Substituting from the above equations and doing manipulations,

$$\dot{W} = \frac{-C_1 r}{K}(N - N^*)^2 - C_2\left(\frac{s_{10}}{L} + s_2 I \right)(B - B^*)^2 - C_3(\theta_1 - \mu B)(I - I^*)^2 - C_4(\lambda_0 + \theta)(P - P^*)^2$$
$$- C_5(\delta + \alpha B)(T - T^*)^2 + (C_1\beta_1 - C_2\beta_2)(N - N^*)(B - B^*)$$
$$+ [C_3 \mu I^* - C_2(s_1 + s_2 B^*)](B - B^*)(I - I^*) + C_4\lambda(N - N^*)(P - P^*) - C_5\alpha T(B - B^*)(T - T^*)$$
$$+ C_5\pi_1\theta(T - T^*)(P - P^*)$$

Choosing the values of the as

$$C_1 = 1,\ C_2 = \frac{\beta_1}{\beta_2},\ C_3 = \frac{\beta_1(s_1 + s_2 B^*)}{\beta_2 \mu I^*}$$

The equation reduces to

$$\dot{W} = \frac{-C_1 r}{K}(N - N^*)^2 - C_2\left(\frac{s_{10}}{L} + s_2 I \right)(B - B^*)^2 - C_3(\theta_1 - \mu B)(I - I^*)^2 - C_4(\lambda_0 + \theta)(P - P^*)^2$$
$$- C_5(\delta + \alpha B)(T - T^*)^2 + C_4\lambda(N - N^*)(P - P^*) - C_5\alpha T(B - B^*)(T - T^*) - C_5\pi_1\theta(P - P^*)(T - T^*)$$

For $\dot{W}$ to be negative definite the following conditions must be satisfied,

$$\lambda_1^2 < \frac{r_0\left(\theta_1 - \mu B^*\right)}{K}$$

$$\lambda^2 < 2\frac{r_0\left(\lambda_0 + \theta\right)}{K}$$

$$\left(\alpha T^*\right)^2 < \frac{\beta_1}{\beta_2}\left(\frac{s_{10}}{L} + s_2 I^*\right)\left(\delta + \alpha B^*\right)$$

Which is same as done in above theorem.

The above theorems imply that under some conditions the equilibrium level of population and resource biomass density decreases as the emission rate of the toxicant from the external source increases and precursor is introduced in the environment. It is also noted that this decrease is enhanced as the rate of transformation of the precursor produced by the biological population increases. Further it is pointed out that if the amount of toxicant in the environment goes on increasing, the survival of species may be threatened.

5. Conclusions

In this paper, a mathematical model has been proposed and analyzed to study the effect of a industrialization, toxicant and precursor produced by human population, on a biological species, such as plants, trees etc. toxicant/pollutant emitted into the environment by an external source and its concentration is augmented due to transformation of a precursor. The existence of non-trivial equilibrium has been proved and the sufficient conditions for its stability behavior have been determined. It has been shown that the population settles down to an equilibrium level, which is much lower than its initial (toxicant independent) carrying capacity, the magnitude of which decreases as the emission rate of the toxicant from external source increases. This decrease is further enhanced if the rate of formation of toxicant from precursor produced by the species increases.

It is noted that for large concentration of the toxicant produced in the environment, the possibility of survival of biological population may be threatened.

References

1. DeLuna JK, Hallam TG. Effect of toxicants on population: a qualitative approach IV. Resource-consumer-toxicant models. Ecol. Modelling 1987; 35:249-273.
2. Freedman HI, Shukla JB. Models for the effect of toxicant in single species and predator-prey systems. J Math. Biol. 1991; 30:15-30.
3. Freedman HI, So JWH. Global stability and persistence of simple food chains Math. Biosciences. 1985; 76:69-86.
4. Garcia-Montiel DC, Scatena FN. The effect of human activity on the structure and composition of a tropical forest in Puerto Rico. Forest Ecol. Management. 1994; 63:57-78.
5. Hallam TG, Clark CE. Nonautonomous logistic equation as models of population in a deteriorating environment. J. Theor. Biol. 1982; 93:303-311.
6. Hallam TG, Clark CE, Jordan GS. Effects of toxicants on populations: a qualitative approach II, First order Kinetics, J. Math. Biol., 1983a; 18:25-37.
7. Hallam TG, Clark CE, Lassiter RR. Effects of toxicants on populations: a qualitative approach I. Equilibrium environmental exposure, Ecol. Model. 1983 b; 18:291-304.
8. Hallam TG, De Luna JT. Effects of toxicants on population: A qualitative approach III. Environmental and food chain pathways, J Theor. Biol. 1982; 109:411-429.
9. Huaping L, Zhein M. The threshold of survival for system of two species in a polluted environment, J. Math. Biol., 1991; 30:49-62.
10. LaSalle J, Lefschetz S. Stability by Liapunov's Direct Method with Applications, Academic Press, New York, London, 1961.
11. McLaughlin SB. Effects of air pollution on forests, J. of Air Pollut. Control Assoc. 1985; 35:512-534.
12. Padoch C, Vayada AP. Pattern of resource use and human settlement in tropical forest. In: Golley F.B. (ed), Tropical Rain Forest Ecosystems, Elsevier, New York, 1983, 301-313.
13. Patin SA. Pollution and the biological resource of the ocean, Butterworth Scientific, London, 1982.
14. Rescigno A. The struggle for life- V. One species living in a limited environment, Bull. Math. Biol. 1977; 39:479-485.
15. Schulze ED. Air pollution and forest decline in a Spruce (Picaabies) forest, Science. 1989; 224:776-783.
16. Shukla JB, Agarwal A, Dubey B, Sinha P. Existence and survival of two competing species in a polluted environment: A mathematical model, Journal of biological Systems. 2001; 9(2):89-103.
17. Shukla A, Shukla JB, Dubey B. Effect of Environmentally degraded soil on crop yeild: the role of conservation. Ecological Modelling. 1996; 86:235-240.
18. Shukla JB, Dubey B. Modelling the depletion and conservation of forestry resource: Effects of population and pollution J. Math Bio. 1997a, 35.
19. Shukla JB, Dubey B, Freedman HI. Effect of changing habitat on survival of species. Ecol. Model. 1997b; 87:205-216.
20. Shukla JB, Freedman HI, Pal VN, Misra OP, Agarwal M, Shukla A. Degradation and subsequent regeneration of a forestry resource: A mathematical model. Ecol. Model. 1989; 44:219-229.
21. Srinivasu PDN. Control of environmental pollution to conserve a population, Non Linear Analysis, RWA 2002; 3:397-411.
22. Woodwell GM. Effects of pollution on the structure and physiology of ecosystems, Science, 1970; 168:429-431.
23. Woodman JN, Cowling EB. Airborne chemicals and forest health, Environ. Sci. Technol., 1987; 21:120-126.