# DEVELOPMENT OF TOXICITY MODEL

By

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**Dissertation submitted in partial fulfilment of**
**the requirements for the**
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**CERTIFICATION OF APPROVAL**

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Approved by,

(Mr Azizul Bin Buang)

UNIVERSITI TEKNOLOGI PETRONAS

TRONOH, PERAK

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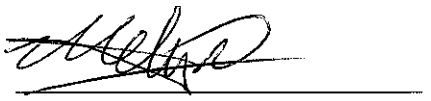
## CERTIFICATION OF ORIGINALITY

This is to certify that I am responsible for the work submitted in this project, that the original work is my own except as specified in the references and acknowledgements, and that the original work contained herein have not been undertaken or done by unspecified sources or persons.



Melvyn Lim Chee Liang

## ABSTRACT

Inherent safety has been recognized as a design approach useful to remove or reduce hazards at the source instead of controlling them with add-on protective barriers. Methods developed to date have largely been for the evaluating the safety of a proposed design. At the moment it seems that the best practice is not adopted quickly enough by the potential practitioners. The aim of this project is to develop a toxicity model that can be used in the design stages of chemical producing plants to predict the concentration at a given distance away from the point of release. The scope of this project is to create a toxicity model for ammonia. The model consists of two major parts, which are Gaussian Plume Equation and toxicity of ammonia. The information on these two parts need to be merged so user(s) can then find out the severity of the concentration of ammonia at a given distance away from the point of incident. Using Microsoft Excel as the program to run this toxicity model, the interface is arranged to make it as user-friendly as possible. Some of the important parameters to be keyed into the model are molecular weight, density, temperature, pressure, flow rate and distance of leak/rupture from ground level. Overall, this project can be used to evaluate whether it is feasible for a chemical producing plant to be built near housing areas. Further improvements are necessary to commercialize and integrate this project with other risk effects estimation.

## ACKNOWLEDGEMENTS

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# CHAPTER 1
# INTRODUCTION

## 1.1 BACKGROUND

Inherently safer design (ISD) concepts have been with us for over two decades since their elaboration by Kletz. Interest has really taken off globally since the early nineties after several major mishaps occurred during the eighties (Bhopal, Mexico city, Piper-alfa, Philips Petroleum, to name a few). Academic and industrial research personnel have been actively involved into devising inherently safer ways of production. Research funding has also been forthcoming for new developments as well as for demonstration projects.

While the Traditional approach aims to reduce the risk of a process by adding protective barriers to control the hazards, Inherent Safety aims to reduce or eliminate the hazards by modifying the design of the plant. Khan F. I. and Amyotte P. R. (2002) declared that "External safety is a cost intensive approach as the add-on control devices require continual staffing and maintenance as well as repetitive training and documentation upkeep throughout the life of the plant. It is favoured by management that considers safety and environmental activities as a need rather than a requirement, thus ignoring the use of basic principles of science in eliminating or reducing operational safety control measures."

Consequently, another method was developed in order to overcome this design strategy and it is known as inherent safety. Inherent safety involves the elimination or reduction of process hazards through the use of inherent properties of materials or processes and process equipment. Having been formalized approximately 35 years ago, full exploration of inherent safety ensure safe processing of chemicals and prevention of industrial accidents which in turn minimizing human, financial and material losses. Crawley F. (1995) and Lutz W. K. (1997) stated that an inherent

safety culture often achieves the lowest lifetime costs per unit mass of product in relation to safety and environmental concerns.

Inherent safety, sometimes referred as "primary prevention" is an approach to chemical accidents that is opposite to "secondary accident prevention" and mitigation. It is helpful also for pollution prevention. This approach to safety is based on the use of technologies and chemicals that reduce or eliminate the possibility of an accident. On the other hand, traditional safety relies on the reduction and mitigation of the consequences of an accident. This last approach alone is unable to avoid or reduce the risk of serious chemical accidents [Zwetsloot G.and Askounes-Ashford, 1999].

While the basic principle governing the inherent safety is generally accepted, this project presents the integration of Gaussian plume model with toxicity monograph of as a design tool for chemical plant design.

## 1.2 PROBLEM STATEMENT

The traditional approach can be very efficient and useful, but it presents some disadvantages. The initial cost of the plant could be lower, compared with the inherently safer options; however the installation of safety barriers represents an additional expense. The barriers must receive expensive maintenance [Lutz, W.K., 1997] and they can suffer a complete or partial dangerous-failure (undetected failure). Since the original hazard is still present, accidents can occur and its consequences could be worsted by the dangerous-failure mode of the barrier. Because the social, environmental and economical cost derived from every accident is not taken into account in the short-term economical analysis of a process, the initial lower cost of the plant is usually untrue.

Prevention is always better then control; however, process safety for the chemical industry has been traditionally applied as hazard control measures. The process safety approach based on control is known as "extrinsic" safety" while the process safety approach based on prevention is known as "inherent safety".

This dissertation establishes a conceptual framework for the analysis of inherent safety by means of a toxicity modelling. This model is proposed to be used in the design stage of a chemical plant – hence, a design tool.

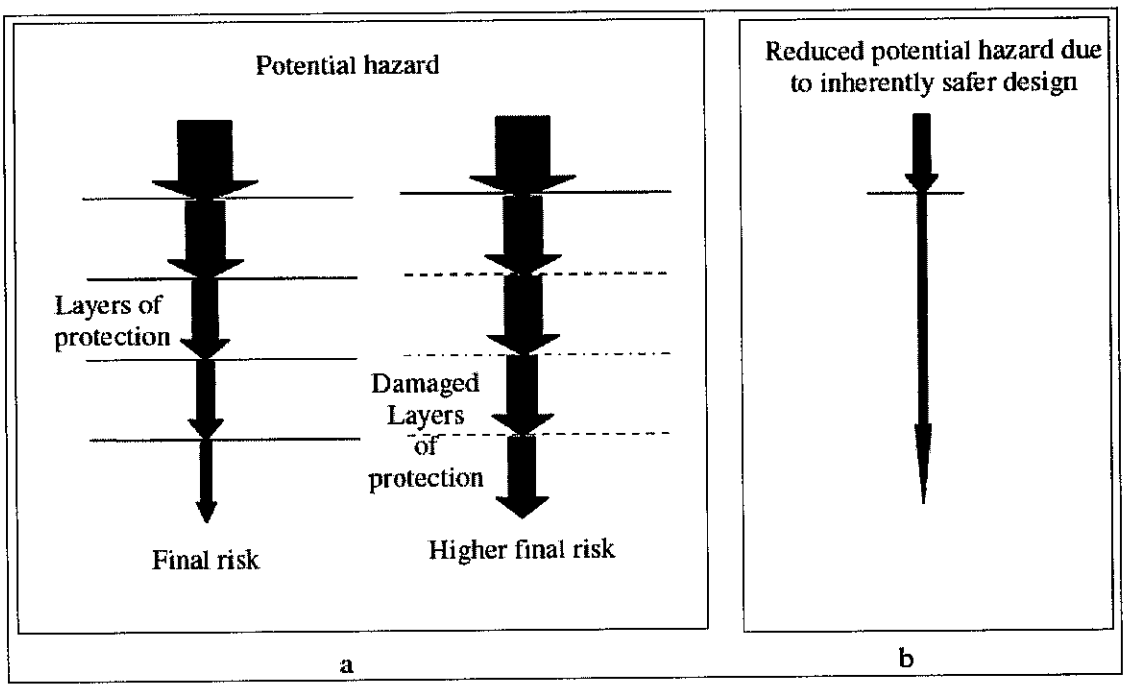



Figure 1: Difference between safety achieved through layers of protection (a) and inherently safety (b) [Hendershot, 1998]

In spite of the effort by many researchers and industrial people, there exists a general resistance to adopt and apply systematically the inherent safer design principles. Some of the most important problems that make difficult the application of inherent safety are: lack of awareness and knowledge about inherent safer technologies, conservatism in design and management. This depends also on the corporate attitude and objectives that are translated to cost/time pressure on projects need to meet legislative requirements, and other causes. There also has been inadequacy in tools/models such as Toxicity Model to assist in Inherent Safety Designs. Most of ISD are conceptual and theoretical. The lack of experience and knowledge (field and "real world plant") of the designers who are applying these principles and the lack of recognized methodology to review the agreement of different process alternatives

according to the Inherent Safety principles are other important obstacles to the implementation of this safety philosophy.

During the last years a number of techniques and tools for risk analysis and inherent safety quantification have been developed. However they present limitations because these tools:

- Have been developed to be applied to the earlier phases of the plant life-cycle or to a complete operating plant.
- Cover only specific elements of the aspects involved in the safety of a plant.
- Require subjective judgment and don't propose a technique to evaluate the effect of those personal evaluations.

Some disadvantages associated with these well known tools are:

- These procedures can only provide a partial idea about the safety present in a facility.
- The analytical statistical methodologies are rigorous but depend on data that in some cases are little more than guesses, since some events are too rare to allow the collection of statistically meaningful information [Bowles J.B. and Pelaez C.E., 1995].
- Combining data with a high degree of uncertainty will increase the uncertainty of the analysis information [Bowles J.B. and Pelaez C.E., 1995].

Besides the limitations associated to the previous methodologies, there exist and additional problem inherent to the mathematical foundation of the analysis. The chemical process plant is a facility composed by a large number of various elements and aspects whose relations are complex and include technical, human, environmental and economical factors.

## 1.3 OBJECTIVES AND SCOPE OF STUDY

The main objective of this project is to develop and improve a toxicity risk model in Microsoft Excel (ME) application. Then it is expected to integrate Gaussian Plume

Model with the Acute Toxicity Summary. ME platform will be used for the mathematical calculation and graph simulations. The student would have to simulate an ammonia manufacturing process (with sufficient operating parameters) and then correlate with the toxicity models (in ME applications) to evaluate the impact and consequences related to the toxic properties of the chemical presence in the process.

The entire research would start with the accumulation of information on the process of ammonia production, chemicals involved in the production and the physical and chemical properties. Concept of toxicity - Lethal concentration (LC), and toxic threshold limit values (TLV) will also be studied upon and all the chemicals identified for the ammonia production are correlated with them. Models on how to calculate the impact or consequences of being exposed to the toxic chemicals will be developed.

# CHAPTER 2
# LITERATURE REVIEW / THEORY

## 2.1 Introduction to Inherent Safety

Inherently Safer Design is a concept known since 1870, but it was not until a hundred years later when engineers considered it significantly. Despite the encouraging results obtained from past applications, there is a general resistance to adopt and systematically apply inherent safer design principles. The main purpose of Inherently Safer Design is quite different in comparison with the aim of the traditional concepts of Safety. While the former aims to eliminate or to reduce the hazards present in a process facility, the latter aims to control hazards and to reduce the consequences of a possible accident by using add-on barriers. Thus the hazard may still be present and "safety" depends upon the reliability of the protective barriers, which present other disadvantages such as high installation and maintenance costs [Lutz, 1997].

The best way of dealing with a hazard is to remove it completely. The provision of means to control the hazard is very much second solution. As Lees (1996) has said the aim should be to design the process and plant so that they are inherently safer.

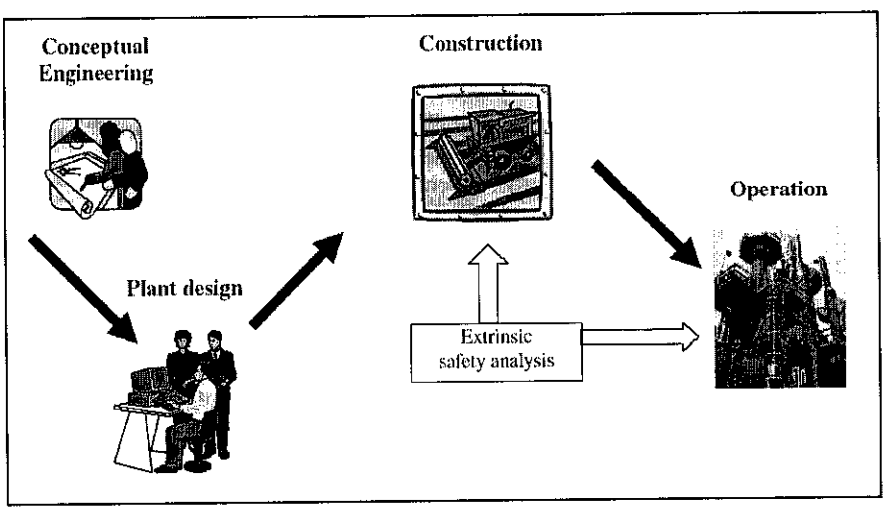



Figure 2: Current application of extrinsic safety methodologies during plant design

"Inherent" is defined by The American Heritage® Dictionary of the English Language, Fourth Edition as "Existing as an essential constituent or characteristic; intrinsic". Thus an inherently safer chemical process is safer because of its essential characteristics, those which belong to the process by its very nature. An inherently safer design is one that avoids hazards instead of controlling them, particularly by removing or reducing the amount of hazardous material in the plant or the number of hazardous operations.

Inherent safety has first widely expressed in the late 1970's by Trevor Kletz. The basic principles are common sense and include avoiding the use of hazardous materials, minimising the inventories of hazardous materials and aiming for simpler processes with more benign and moderate process alternatives (Kletz, 1984).

While the basic principle of inherently safer design is generally accepted, it is not always easy to put it into practice. Inherently safer design has been advocated since the explosion at Flixborough in 1974. Progress has been real but nevertheless the concept has not been adopted nearly as rapidly as quantitative risk assessment, introduced into the chemical industry only a few years earlier (Kletz, 1996).

It has been commented that methods developed to date have largely been for evaluating the safety of some proposed design. In the future safety experts expect to see a greater emphasis on the use of knowledge to synthesize a safe plant design in the first place. In their opinion the value of inherent safety has been recognised, but there is still room for better awareness and practice. The concern expressed by inherent safety experts is that best practice is not being adopted quickly enough by the potential practitioners (Preston, 1998).

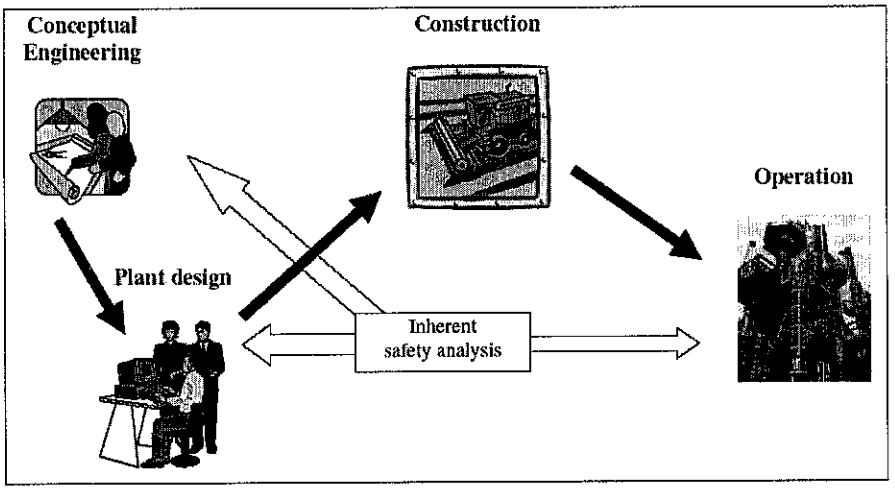



Figure 3: Targeted application of the proposed inherent safety

### 2.1.1 The Principles of Inherent Safety

*Minimize (intensification)*:

"What you don't have, can't leak". Small inventories of hazardous materials reduce the consequences of leaks. Inventories can often be reduced in almost all unit operations well as storage. This also brings reductions in cost, while less material needs smaller vessels, structures and foundations. Intensification strategy challenges the process designers to determine an optimum inventory of hazardous materials that compromises neither profitability nor the safety integrity of a process when the hazardous materials cannot be eliminates (Khan F. I. and Amyotte P. R., 2002). This strategy leads to the use of smaller and simpler equipment.

*Substitute (substitution)*:

If intensification is not possible, an alternative is substitution. It may be possible to replace flammable refrigerants and heat transfer with non-flammable ones, hazardous products with safer ones, and processes that use hazardous raw materials or intermediates with processes that do not. Using a safer material in place of a hazardous one decreases the need for added-on protective equipment and thus decreases plant cost and complexity. Substitution strives to eliminate

material with highly hazardous inherent characteristics (e.g. flammability, reactivity and toxicity).

*Moderate (attenuation and limitation of effects)*:

Attenuation means carrying out a hazardous reaction under less hazardous conditions, or storing or transporting a hazardous material in a less hazardous form. It is sometimes the reverse of intensification, because less extreme reaction conditions may lead to a longer residence time. Limitation of effect is having effects of a failure limited. For instance equipment is designed so that it can leak only at a low rate that is easy to stop or control. Also limitations of effects should be done by equipment design or change in reaction conditions rather than by adding protective equipment. It is worth emphasizing that the overall objective of all moderation strategies is elimination or reduction of hazards.

*Simplify (simplification/error tolerance)*:

Simpler plants are inherently safer than complex plants, because they provide fewer opportunities for error and contain less equipment that can go wrong. Simpler plants are usually also cheaper and more user friendly. Equipment should tolerate poor installation or maintenance without failure. The construction materials should be resistant to corrosion and physical conditions. For most applications metal is safer than glass or plastic. Simplification involves designing process to eliminate irrelevant complexities that minimizing the opportunities for errors to occur for better layout of plant equipment and elimination of passive structures.

### 2.1.2 Inherent Safety in Preliminary Process/Plant Design

The possibility for affecting the inherent safety of a process decreases as the design proceeds and more and more engineering and financial decisions have been made (Fig. 4). It is much easier to affect the process configuration and inherent safety in the conceptual design phase than in the later phases of process design. For instance the

process route selection is made in the conceptual design and it is many times difficult and expensive to change the route later. Time and money is also saved when fewer expensive safety modifications are needed and fewer added-on safety equipment are included to the final process solution. In the early design phases the available information is limited to products, by-products and raw materials, capacity, main process equipment and a rough range of process conditions e.g. temperature and pressure. In the early phases of a plant design the changes will be most profitable, since nothing has been built or ordered yet and thus no expensive modifications are needed.

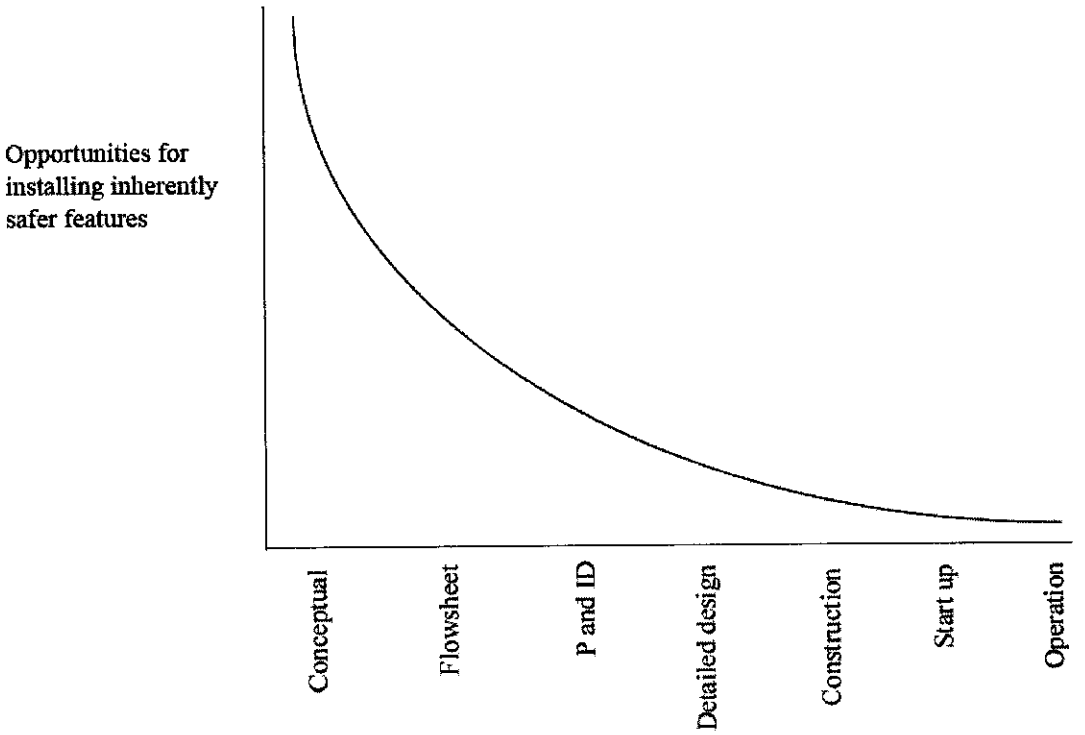



Figure 4: Inherently safer features become harder to install as a project progresses (Kletz, 1991)

### 2.1.3 Inherent Safety Indicators

Table 1: Inherent Safety Indicators (Khan F. I. and Amyotte P. R., 2002).

| Term | Description |
|---|---|
| 1. Inventory | The quantity of material in a process, wherein for potentially hazardous material a process becomes inherently less safe as the quantity of the material increase. |
| 2. Pressure | Indicator of hazard level of a process. High pressure indicates high potential energy as it provides the needed momentum for materials to escape at high velocities from confinement. |
| 3. Temperature | It is a necessary parameter for assessing the inherent safety of a process as molecules possess higher kinetic energy at higher temperature and vice versa. Systems operating at high temperature and pressure are more prone to fire and explosion hazards since the contents can easily flashed. |
| 4. Flammability | It is generally regarded that the flash point of a material is an appropriate property for the determination of flammability hazard. |
| 5. Reactivity | It is the ability of a material to react both itself and with other materials. |
| 6. Toxicity | It is a measure of the ability of a material to impair the health of living organism. Toxic material can be classified those that generate severe impact upon short exposure, and others can generate noticeable effect or permanent damage only on long-term. Thereby, minimizing their ability mitigate the severity of an incident. |

### 2.2 What is Toxicity?

Marshall (1987) and Well (1980) have described toxicity as a property of substance which destroys life or injures health when introduced into or absorbed by a living organism. The toxic hazard is a measure of the likelihood of such damage occurring.

It is determined by the frequency and duration of such exposure and the concentration of chemical in exposure.

The degree to which a substance or mixture of substances can harm humans or animals. Acute toxicity involves harmful effects in an organism through a single or short-term exposure. Chronic toxicity is the ability of a substance or mixture of substances to cause harmful effects over an extended period, usually upon repeated or continuous exposure sometimes lasting for the entire life of the exposed organism. Subchronic toxicity is the ability of the substance to cause effects for more than one year but less than the lifetime of the exposed organism.

According to Wells (1980) probably the most commonly used toxicity term is the Threshold Limit Value (TLV), which has been defined for five consecutive 8-hour working days. TLVs are based on different effects from irritation to a physiological damage. Especially in industrial context TLVs are the most usable toxicity values, while their aim is to protect employees at work. Threshold of odour is important when the TLV values are lower than the concentration of a substance needed for an odour to appear (Siegell, 1996).

Lethal Concentration is concentration of a potentially toxic substance in an environmental medium that causes death following a certain period of exposure (denoted by LC). LC is measured in milligram per kilogram of body weight (mg/kg). $LC_{50}$ is the concentration of substance in air to which exposure for 24 hours or less would cause the death of 50% of a test group.

### 2.2.1 Introduction to Human Toxicity

Toxic effects in human can occur due to exposure to a chemical substance through three main routes; inhalation, ingestion, and dermal contact, and the effects can be acute or chronic. While acute effects are produced by exposure to high doses of chemical for a short time, with immediate consequences for human health, chronic

effects occur due to exposure to lower doses for a long period of time and the symptoms can be latent or gradual [Lees, 1996]. In terms of toxicity, chemical substances can be classified according to their effects on the human organism. For instance, gases can be simple asphixiants when they only displace oxygen but are biologically inert (e.g. methane and carbon dioxide); chemical asphyxiants affect the absorption of oxygen by displacing it or destroying its transport mechanics (e.g. carbon monoxide, cyanide, hydrogen sulphide); irritants produce injury of the mucosa and, depending on their water solubility, will affect the upper or lower airways (e.g. ammonia affects upper airways while chlorine affects lower airways); asphyxiants and irritants affect the airways in both forms, by reducing the oxygenation and by injuring the respiratory tract. Other forms of toxicants may cause sensitization of specific organs such as respiratory system and liver; in other cases chemicals can carcinogenic, or have reproductive and teratogenic consequences.

The toxic effect of a chemical depends on several factors, such as its physicochemical properties, exposure time, dosage, and toxic mechanism. Therefore the evaluation of a chemical from toxicology viewpoint is a complex task and relevant information is usually incomplete or nonexistent [Lees, 1996].

When toxicity values are used for risk assessment it must be kept in mind that the values are based on the assumption that the organism is static; however, in emergency conditions people will be under stress and performing physical activity, increasing therefore the air intake, and the potential for injury may be more severe than expected.

## 2.3 Introduction to Gaussian Plume Equation

Air dispersion modelling has been evolving since before the 1930s. Over the last 15-25 years, strict environmental regulations and the availability of personal computers have fuelled an immense growth in the use of mathematical models to predict the dispersion of air pollution plumes. In most dispersion models, determining the

pollutant concentrations at ground-level receptors beneath an elevated, buoyant plume of dispersing pollutant-containing gas involves two major steps:

First, the height to which the plume rises at a given downwind distance from the plume source is calculated. The calculated plume rise is added to the height of the plume's source point to obtain the so-called "effective stack height", also known as the plume centreline height or simply the emission height.

Then, the ground-level pollutant concentration beneath the plume at the given downwind distance is predicted using the Gaussian dispersion equation.

### 2.3.1 The Gaussian Equation

The ISC short term model for stacks uses the steady-state Gaussian plume equation for a continuous elevated source. For each source and each hour, the origin of the source's coordinate system is placed at the ground surface at the base of the stack. The x axis is positive in the downwind direction, the y axis is crosswind (normal) to the x axis and the z axis extends vertically. The fixed receptor locations are converted to each source's coordinate system for each hourly concentration calculation. The hourly concentrations calculated for each source at each receptor are summed to obtain the total concentration produced at each receptor by the combined source emissions.

For a steady-state Gaussian plume, the hourly concentration at downwind distance x (meters) and crosswind distance y (meters) is given by:

$$\chi = \frac{QKVD}{2\pi u_s \sigma_y \sigma_z} \exp\left[-0.5\left(\frac{y}{\sigma_y}\right)^2\right] \qquad 2\text{-}1$$

Where:

Q = pollutant emission rate (mass per unit time)

K = a scaling coefficient to convert calculated concentrations to desired units (default value of 1 x $10^6$ for Q in g/s and concentration in $\mu g/m^3$)

V = vertical term

D = decay term

$F_y$,$F_z$ = standard deviation of lateral and vertical concentration distribution (m)

$u_s$ = mean wind speed (m/s) at release height

Equation (2-1) includes a Vertical Term (V), a Decay Term (D), and dispersion parameters ($F_y$ and $F_z$) as discussed below. It should be noted that the Vertical Term includes the effects of source elevation, receptor elevation, plume rise, limited mixing in the vertical, and the gravitational settling and dry deposition of particulates (with diameters greater than about 0.1 microns).

**2.3.2 Downwind and Crosswind Distances**

The ISC model uses either a polar or a Cartesian receptor network as specified by the user. The model allows for the use of both types of receptors and for multiple networks in a single run. All receptor points are converted to Cartesian (X, Y) coordinates prior to performing the dispersion calculations. In the polar coordinate system, the radial coordinate of the point (r, 2) is measured from the user-specified origin and the angular coordinate 2 is measured clockwise from the north. In the Cartesian coordinate system, the X axis is positive to the east of the user-specified origin and the Y axis is positive to the north. For either type of receptor network, the user must define the location of each source with respect to the origin of the grid using Cartesian coordinates. In the polar coordinate system, assuming the origin is at $X = X_o$, $Y = Y_o$, the X and Y coordinates of a receptor at the point (r, 2) are given by:

$$X(R) = r\sin\theta - X_o \quad 2\text{-}2$$

$$Y(R) = r\cos\theta - Y_o \quad 2\text{-}3$$

If the X and Y coordinates of the source are X(S) and Y(S), the downwind distance x to the receptor, along the direction of plume travel, is given by:

$$x = -(X(R) - X(S))\sin(WD) - (Y(R) - Y(S))\cos(WD) \quad 2\text{-}4$$

where WD is the direction from which the wind is blowing. The downwind distance is used in calculating the distance-dependent plume rise and the dispersion parameters. If any receptor is located within 1 meter of a point source or within 1 meter of the effective radius of a volume source, a warning message is printed and no concentrations are calculated for the source-receptor combination. The crosswind distance y to the receptor from the plume centreline is given by:

$$y = (X(R) - X(S))\cos(WD) - (Y(R) - Y(S))\sin(WD) \quad 2\text{-}5$$

The crosswind distance is used in Equation (2-1).

# CHAPTER 3

# METHODOLOGY

## 3. Methodology

### 3.1. Project Flowchart

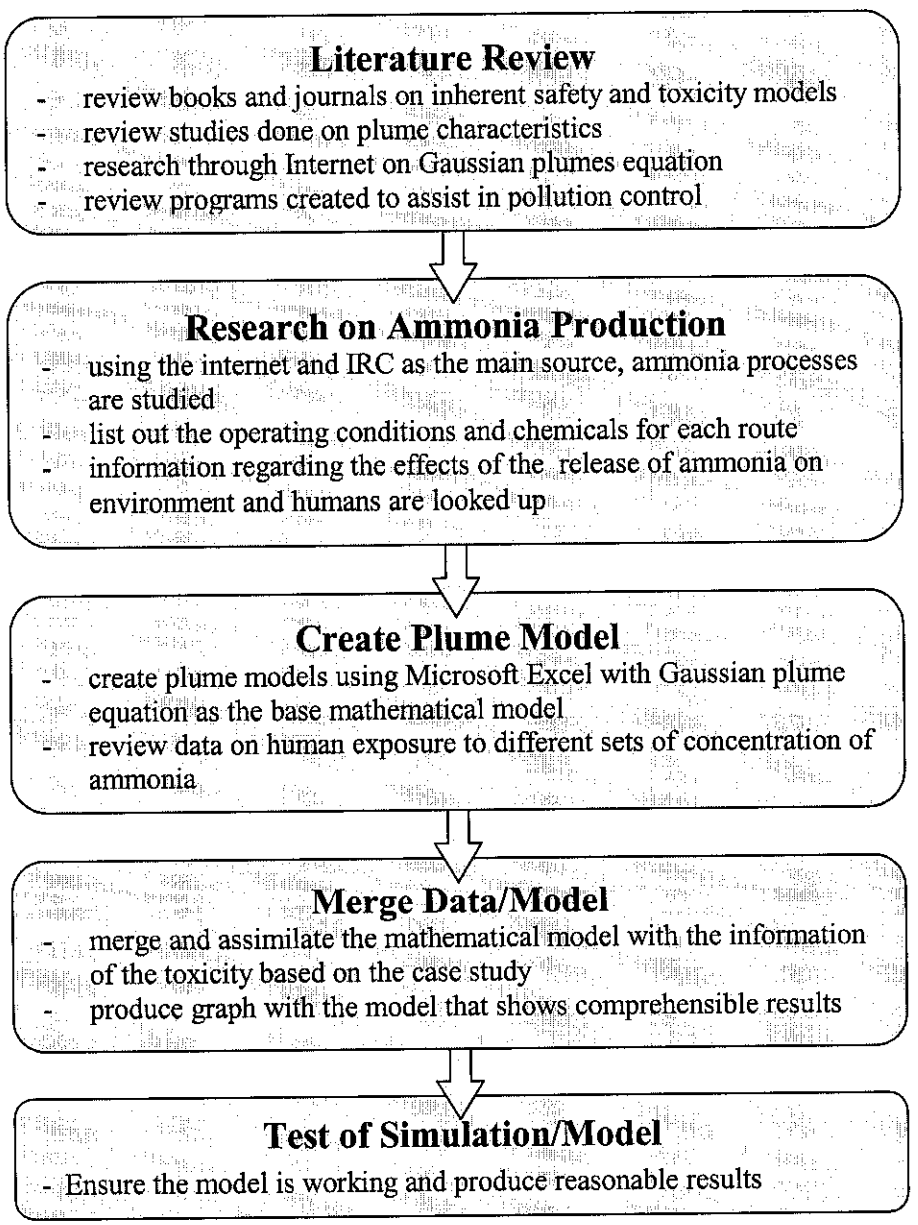



Figure 5: Flow Diagram for the Research Project

## 3.2. Project Procedure

This research project began with reviewing the concept of inherent safety. Research papers and journals from the likes of Kletz, Khan F. I., Amyotte P. R., Crawley F. and Lutz W. K. was reviewed. Other reviews on thesis and online research cover the topic of the Haber process, toxicity, plume models and air pollution control tools. From the literature review, sufficient information and knowledge on the related topic will enable the student to correlate the topic to the objectives of this research project.

After getting an overview of the whole project, specific research was carried out. Information closely related to the case study was reviewed via Internet and Information Resource Centre. Toxicity of ammonia and mathematical Gaussian plume model was specifically researched upon as information on both topics is vital for the success of this project. The topic on toxicity of ammonia was mainly on the impact towards human.

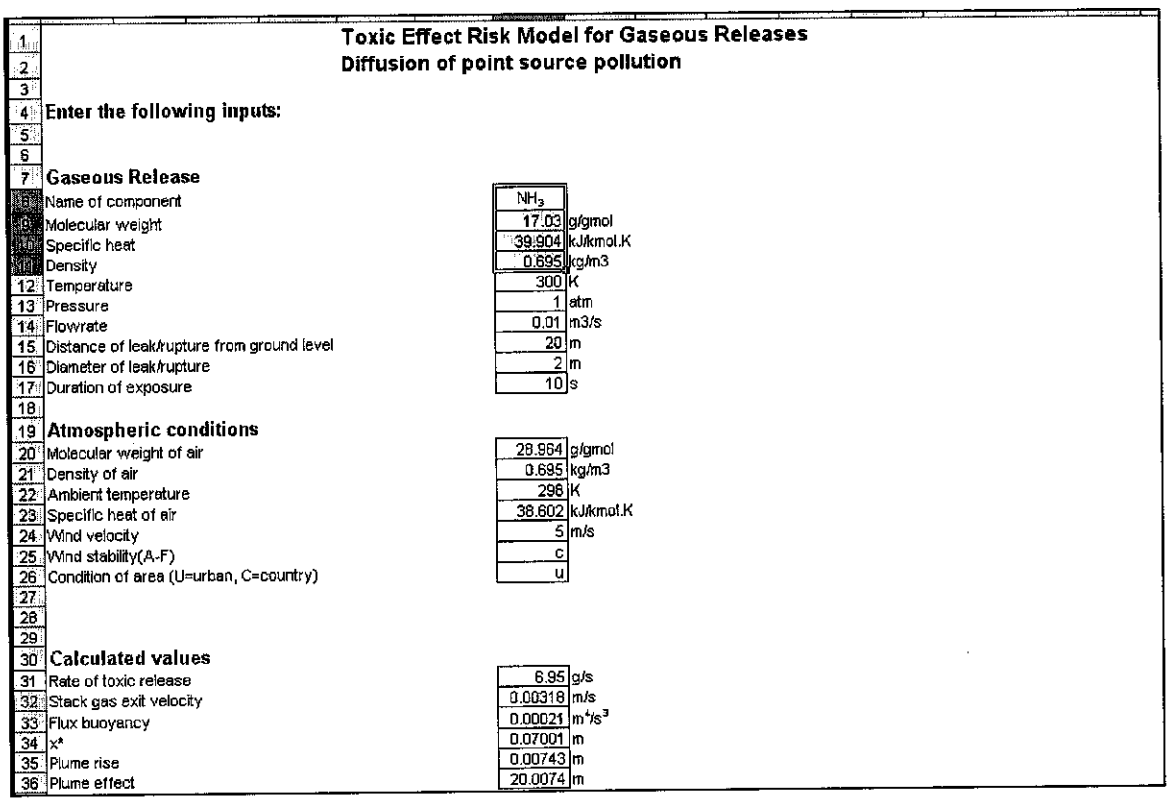
The next step was to further understand the plume mathematical model before proceeding in using Microsoft Excel to create the toxicity model with the mathematical model as the engine to estimate the plume rise and distribution. Some assumptions were made in order to simplify the model so that the plume equations could be used in the model and graphs on concentration vs. distance can be plotted using Microsoft Excel. Data on human exposure to different sets of concentration of ammonia was reviewed through.

After getting the mathematical model running on Microsoft Excel, comprehensible graphs were created from the model (Gaussian plume equation) in order to help user(s) in relating the concentration of ammonia at an interested distance from point of incident to the irritation scale. This can be done by referring the concentration calculated to the graphic representation of benchmark concentration determination. From the irritation vs. ammonia concentration graph, the user(s) will be able to comprehend the severity of the leak at a given distance from point of incident.

The final task is to test and apply the toxicity model to case study. During this stage, the model is tested for any error whether in terms of the calculation, equation or graph. The results should be reasonable and the concentration of toxic release graph should be of the characteristics of a normal Gaussian plume graph.

## 3.3. Procedure in Using the Model

1. This toxicity model is able to work on any airborne toxicants. However, since the case study in this project is on ammonia gas, the information in the highlighted boxes in Figure 6 is filled in according to ammonia gas.

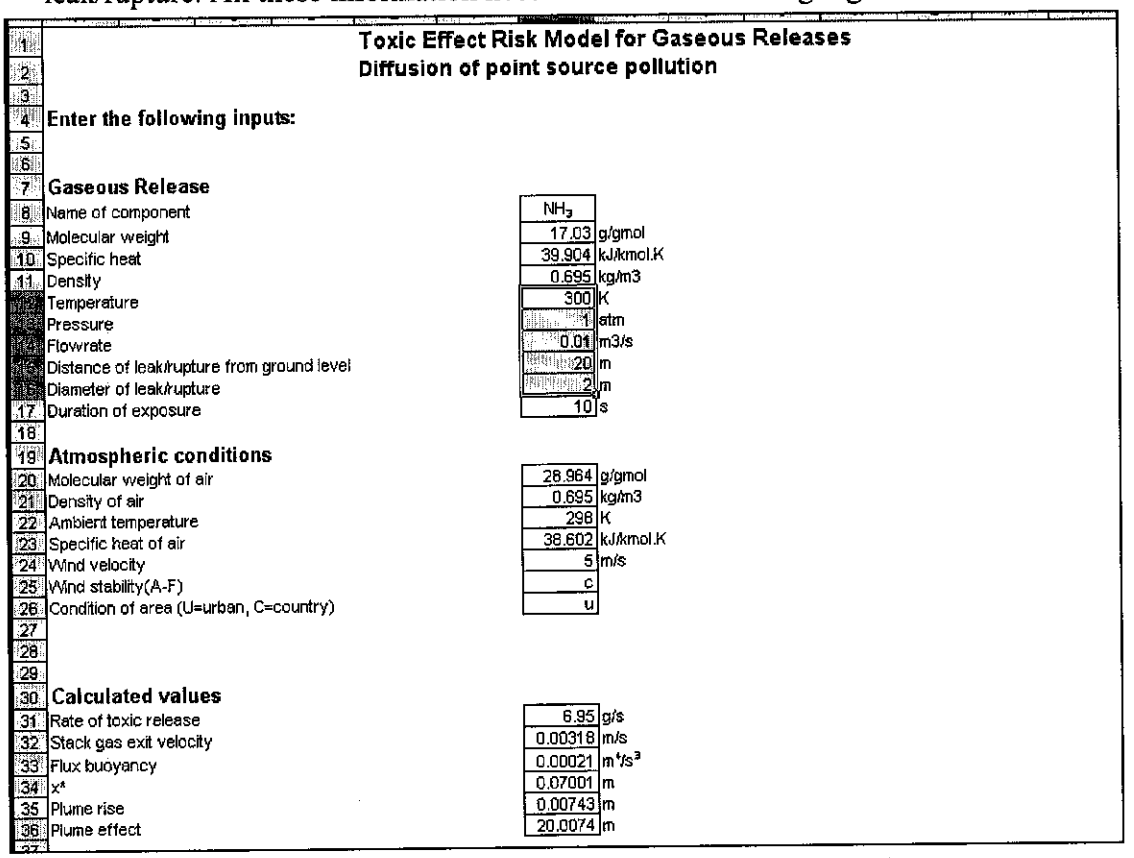


| | | | |
|---|---|---|---|
| | **Toxic Effect Risk Model for Gaseous Releases** | | |
| | **Diffusion of point source pollution** | | |
| | **Enter the following inputs:** | | |
| | **Gaseous Release** | | |
| | Name of component | $NH_3$ | |
| | Molecular weight | 17.03 | g/gmol |
| | Specific heat | 39.904 | kJ/kmol.K |
| | Density | 0.695 | kg/m3 |
| | Temperature | 300 | K |
| | Pressure | 1 | atm |
| | Flowrate | 0.01 | m3/s |
| | Distance of leak/rupture from ground level | 20 | m |
| | Diameter of leak/rupture | 2 | m |
| | Duration of exposure | 10 | s |
| | **Atmospheric conditions** | | |
| | Molecular weight of air | 28.964 | g/gmol |
| | Density of air | 0.695 | kg/m3 |
| | Ambient temperature | 296 | K |
| | Specific heat of air | 38.602 | kJ/kmol.K |
| | Wind velocity | 5 | m/s |
| | Wind stability(A-F) | c | |
| | Condition of area (U=urban, C=country) | u | |
| | **Calculated values** | | |
| | Rate of toxic release | 6.95 | g/s |
| | Stack gas exit velocity | 0.00318 | m/s |
| | Flux buoyancy | 0.00021 | $m^4/s^3$ |
| | x* | 0.07001 | m |
| | Plume rise | 0.00743 | m |
| | Plume effect | 20.0074 | m |

Figure 6: Entering Data Related to Ammonia Gas

It is important to note that the correct molecular weight and density for ammonia gas is filled in as a change in value will affect the final result of the concentration calculated.

2. After filling in the fixed values for ammonia gas, a unique combination of conditions can then be tested, simulating the condition of a specific case of accidental ammonia gas release. Values that needs to be filled covers the temperature of the ammonia gas that is released, the pressure at point of release, the flow rate, distance of leak/rupture from ground level, and diameter of the leak/rupture. All these information need to be filled in the highlighted boxes.



**Toxic Effect Risk Model for Gaseous Releases**
**Diffusion of point source pollution**

**Enter the following inputs:**

| | | |
|---|---|---|
| **Gaseous Release** | | |
| Name of component | $NH_3$ | |
| Molecular weight | 17.03 | g/gmol |
| Specific heat | 39.904 | kJ/kmol.K |
| Density | 0.695 | kg/m3 |
| Temperature | 300 | K |
| Pressure | 1 | atm |
| Flowrate | 0.01 | m3/s |
| Distance of leak/rupture from ground level | 20 | m |
| Diameter of leak/rupture | 2 | m |
| Duration of exposure | 10 | s |
| **Atmospheric conditions** | | |
| Molecular weight of air | 28.964 | g/gmol |
| Density of air | 0.695 | kg/m3 |
| Ambient temperature | 298 | K |
| Specific heat of air | 38.602 | kJ/kmol.K |
| Wind velocity | 5 | m/s |
| Wind stability(A-F) | c | |
| Condition of area (U=urban, C=country) | u | |
| **Calculated values** | | |
| Rate of toxic release | 6.95 | g/s |
| Stack gas exit velocity | 0.00318 | m/s |
| Flux buoyancy | 0.00021 | $m^4/s^3$ |
| $x^*$ | 0.07001 | m |
| Plume rise | 0.00743 | m |
| Plume effect | 20.0074 | m |

Figure 7: Entering Data Related to the Condition of Leak

3. The part for Atmospheric conditions needs to be filled as well. What values that will affect the values of the final calculated concentration are the ambient temperature, wind velocity, wind stability and condition of area. Ambient temperature should always be set to 298K (25°C). As for the wind velocity, the normal wind velocity in Malaysia ranges from 2.78 m/s to 5.56 m/s. For wind stability, both charts as shown in Figure 9 and Figure 10 are used as guide to choose from the value of A to F. In Malaysia, the wind stability is under C. Lastly, the condition of area also needs to be considered. This is because wind

profile varies with geographical conditions. If the study is to be done for a plant in the urban area, U will be chosen, and vice versa.

**Toxic Effect Risk Model for Gaseous Releases**
**Diffusion of point source pollution**

**Enter the following inputs:**

| | | |
|---|---|---|
| **Gaseous Release** | | |
| Name of component | $NH_3$ | |
| Molecular weight | 17.03 | g/gmol |
| Specific heat | 39.904 | kJ/kmol.K |
| Density | 0.695 | kg/m3 |
| Temperature | 300 | K |
| Pressure | 1 | atm |
| Flowrate | 0.01 | m3/s |
| Distance of leak/rupture from ground level | 20 | m |
| Diameter of leak/rupture | 2 | m |
| Duration of exposure | 10 | s |
| **Atmospheric conditions** | | |
| Molecular weight of air | 28.964 | g/gmol |
| Density of air | 0.695 | kg/m3 |
| Ambient temperature | 293 | K |
| Specific heat of air | 38.602 | kJ/kmol.K |
| Wind velocity | 5 | m/s |
| Wind stability(A-F) | C | |
| Condition of area (U=urban, C=country) | U | |
| **Calculated values** | | |
| Rate of toxic release | 6.95 | g/s |
| Stack gas exit velocity | 0.00318 | m/s |
| Flux buoyancy | 0.00021 | $m^4/s^3$ |
| x* | 0.07001 | m |
| Plume rise | 0.00743 | m |
| Plume effect | 20.0074 | m |

Figure 8: Entering Data Related to the Atmospheric Conditions

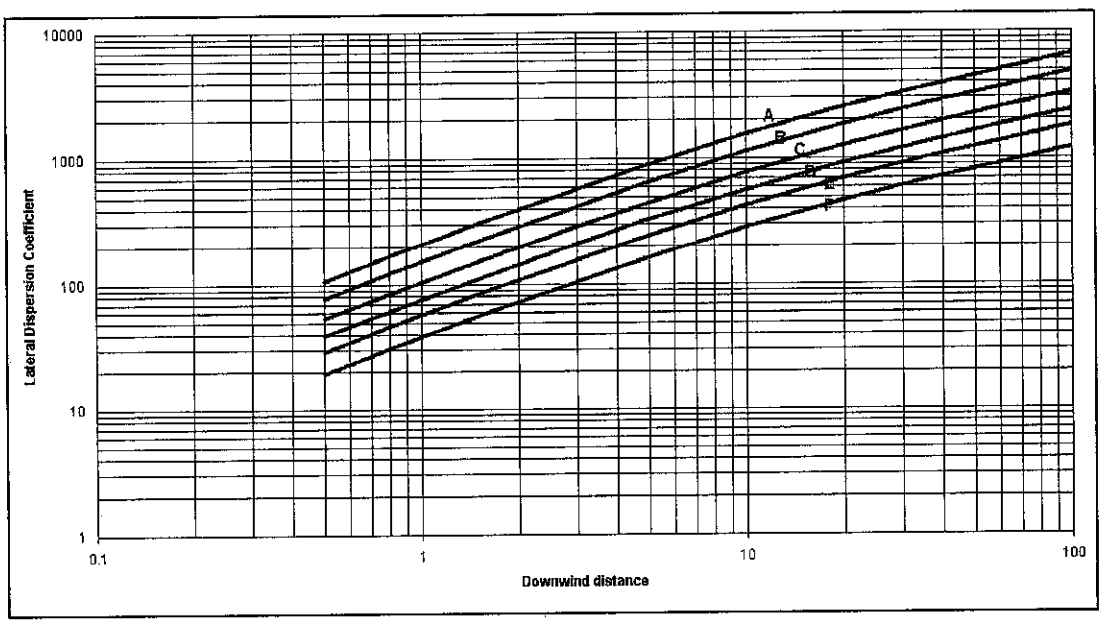



Figure 9: Lateral Dispersion Coefficient vs. Downwind Distance (Sigma Y)

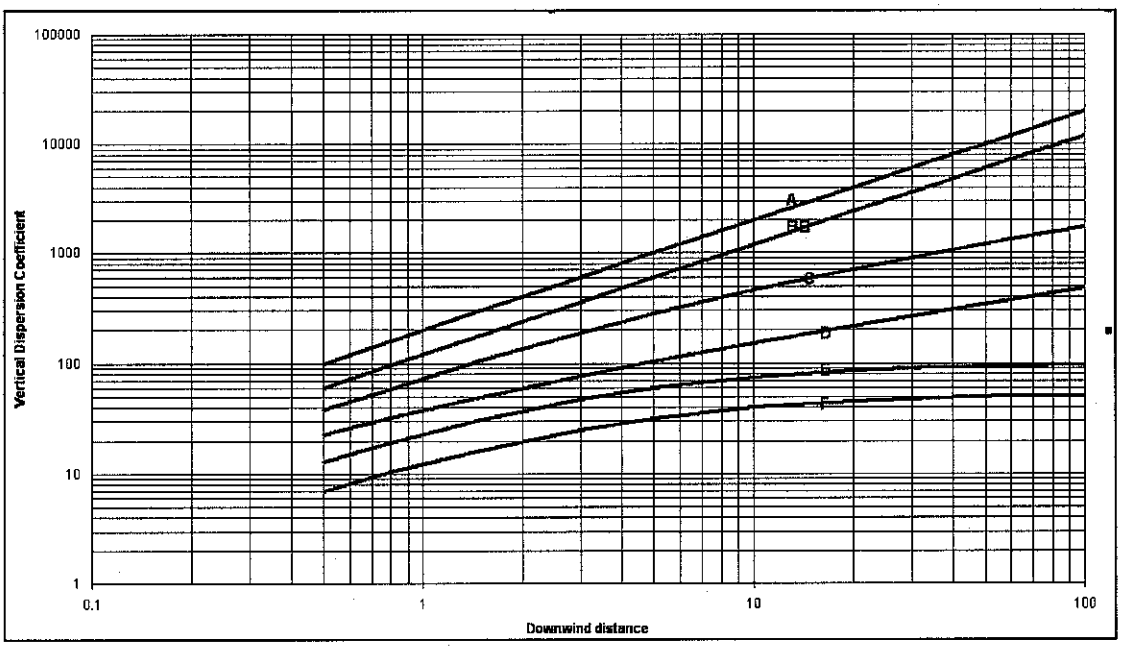



Figure 10: Vertical Dispersion Coefficient vs. Downwind Distance (Sigma Z)

4. After keying in all the required values, the model is able to calculate the concentration in $mg/m^3$ and also in ppm. Figure 11 shows the trend line of the concentration plotted against distance from point of incident. Besides that, the model will also calculate the concentration in terms of log ppm, which will be used to check the irritation level.

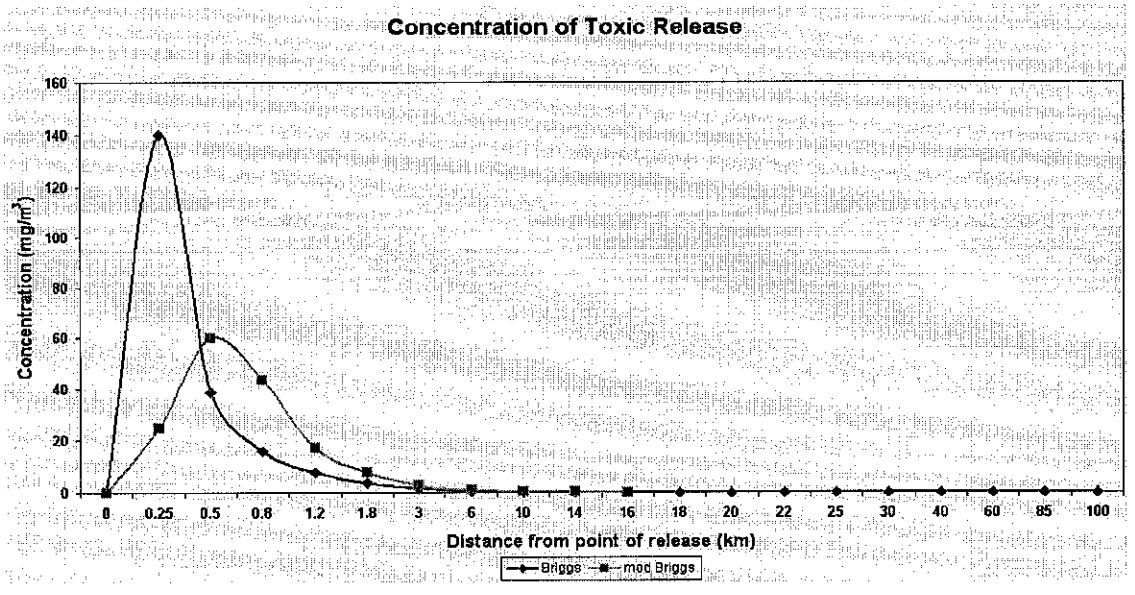


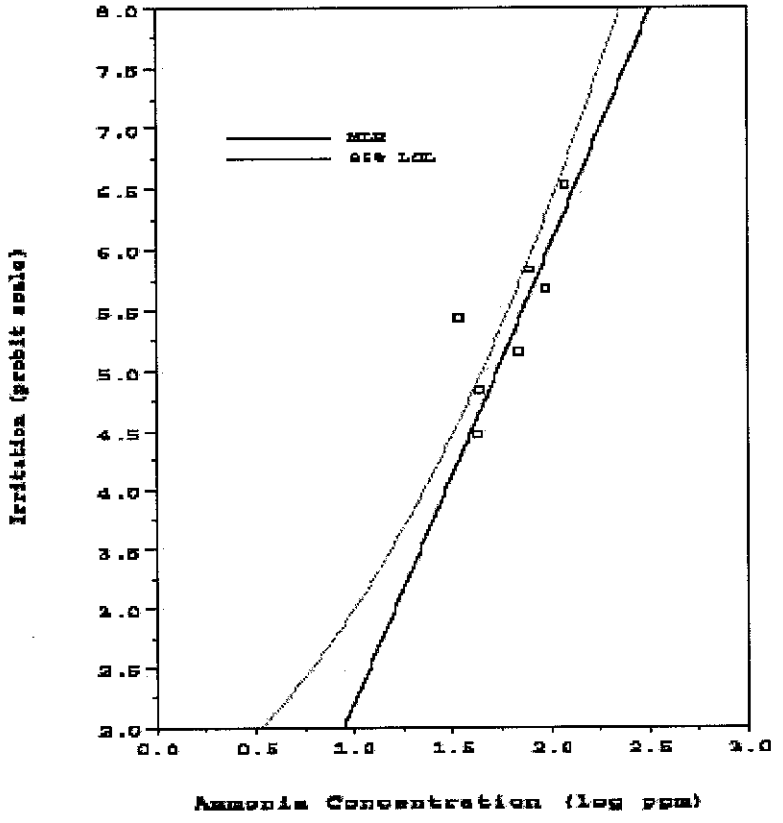

Figure 11: Concentration of Toxic Release

Estimated Concentration of Ground-Level Pollution (mg/m3) on Plume Centerline at Selected Distances (km) from Source

| Downwind distance (km) | Estimated concentration (mg/m3) | Estimated concentration (ppm) | Concentration (log ppm) |
|---|---|---|---|
| 0 | 0 | 0 | NA |
| 0.25 | 140.066 | 202.587 | 2.30661 |
| 0.5 | 38.8383 | 56.1744 | 1.74954 |
| 0.8 | 16.1071 | 23.2987 | 1.36729 |
| 1.2 | 7.61318 | 11.0114 | 1.04184 |
| 1.8 | 3.65473 | 5.28607 | 0.72313 |
| 3 | 1.48948 | 2.15433 | 0.33331 |
| 6 | 0.46311 | 0.66983 | -0.17404 |
| 10 | 0.2022 | 0.29245 | -0.53395 |
| 14 | 0.11853 | 0.17143 | -0.76591 |
| 16 | 0.09609 | 0.13898 | -0.85705 |
| 18 | 0.07992 | 0.1156 | -0.93706 |
| 20 | 0.06782 | 0.09809 | -1.00836 |
| 22 | 0.05849 | 0.0846 | -1.07285 |
| 25 | 0.04789 | 0.06941 | -1.1586 |
| 30 | 0.03623 | 0.0524 | -1.28069 |
| 40 | 0.0233 | 0.0337 | -1.47231 |
| 50 | 0.01256 | 0.01817 | -1.74075 |
| 85 | 0.0074 | 0.01071 | -1.97022 |
| 100 | 0.00579 | 0.00837 | -2.07702 |

Figure 12: Estimated Concentration of Ground-Level Pollution on Plume Centreline at Selected Distances (km) from Source

5. Lastly, the concentration (in log ppm) is used and referred to Figure 13. From there, the probit scales can be determined and whether the value falls under 95% LCL (lower confidence limit) or MLE (maximum likelihood estimate). Mortality can also be determined based on the probit given.




| % Mortality | Probits |
|---|---|
| 10% | 3.72 |
| 20 | 4.16 |
| 30 | 4.48 |
| 40 | 4.75 |
| **50** | **5.00** |
| 60 | 5.25 |
| 70 | 5.52 |
| 80 | 5.84 |
| 90 | 6.28 |

Figure 13: Irritation vs. Ammonia Concentration

# CHAPTER 4
# RESULTS AND DISCUSSION

Chemical substances are everywhere; they are present in the air that we breathe. Some are safe to mine, manufacture or use, others are harmful. The environment is full of chemical substances, some of which are harmful. Harmful interactions (to the organism or system being targeted, or to the organism affected incidentally or affected accidentally by the chemical) are those which result in loss of homeostasis, leading to debility, damage or death. The harmful interactions may be studied at many levels, from molecular interactions to interactions affecting the overall ability of earth to support present-day life.

The brief exposure to high lethal concentrations of ammonia particularly affects the upper parts of the lungs. At lower levels of concentration there is little doubt that people can acquire tolerance towards ammonia so that a level which is obnoxious or even intolerable to some people may pass unnoticed by others who work habitually in such levels. On the other hand, chronic residual disability and disease have been recorded after single high level exposures to ammonia. Literature reports that ammonia is a primary respiratory irritant in both animals and man. Low concentrations produce irritations and/or inflammation of the upper respiratory tract, nausea, salivation and coughing.

Ammonia is highly water soluble such that it is absorbed rapidly by the mucous coating of the upper respiratory system. Taking all factors into account, it appears reasonable to assume that animal data on the acute lethal effects of ammonia is applicable to man. Indeed, if a bodyweight factor does exist; such a stance will introduce a margin of safety. There is no reason to suppose that the smaller animals are less sensitive than man to the effects of ammonia or that man will respond to a potentially lethal dose in a different manner from animals. The conclusion from research done is that the average value applicable to man is 11, 500 ppm for the 50% lethal concentration at 30 minutes.

From researching on the information of ammonia (being the case study of this Toxicity Model), the information of ammonia is as follows:

### 4.1 Ammonia

*(Anhydrous ammonia; aqueous ammonia)*

#### 4.1.1 Chronic Toxicity Summary

| | |
|---|---|
| *Inhalation reference exposure level* | **200 mg/m3 (300 ppb)** |
| *Critical effect(s)* | Pulmonary function tests or subjective symptomatology in workers |
| *Hazard index target(s)* | Respiratory system |

#### 4.1.2 Physical and Chemical Properties (HSDB, 1994; 1999)

| | |
|---|---|
| *Description* | Colourless gas |
| *Molecular formula* | NH3 |
| *Molecular weight* | 17.03 g/mol |
| *Density* | 0.7710 g/L @ 0°C |
| *Boiling point* | -33.35° C |
| *Vapour pressure* | 7510 torr @ 25°C |
| *Solubility* | Soluble in water, alcohol, and ether |
| *Conversion factor* | 1 ppm = 0.71 mg/m3 |

#### 4.1.3 Major Uses or Sources

This strongly alkaline chemical is widely used in industry as a feed stock for nitrogen-based chemicals such as fertilizers, plastics and explosives (ATSDR, 1990). Ammonia is also used as a refrigerant. The general public is exposed by off-gassing

from cleaning solutions containing aqueous ammonia. Household ammonia solutions contain 5-10% ammonia in water while industrial strength can be up to 28%.

### 4.1.4 Effects of Human Exposures

Comparisons were made between 52 workers and 31 control subjects in a soda ash plant for pulmonary function and eye, skin and respiratory symptomatology (Holness *et al.*, 1989). The pulmonary function tests included FVC (forced vital capacity – the total amount of air the subject can expel during a forced expiration), FEV1 (forced expiratory volume in one second), FEF50 (forced expiratory flow rate at 50% of the FVC) and FEF75 (forced expiratory flow rate at 75% of the FVC). Age, height, and pack-years smoked were treated as covariates for the comparisons. The workers were exposed on average for 12.2 years to mean (time-weighted average) ammonia concentrations of 9.2 ppm (6.4 mg/m3) ± 1.4 ppm, while controls were exposed to 0.3 ppm (0.21 mg/m3) ± 0.1 ppm. No differences in any endpoints (respiratory or cutaneous symptoms, sense of smell, baseline lung function, or change in lung function over a work shift at the beginning and end of a workweek) were reported between the exposed and control groups.

Groups of human volunteers were exposed to 25, 50, or 100 ppm (0, 17.8, 35.5, or 71 mg/m3) ammonia 5 days/week for 2, 4, or 6 hours/day, respectively, for 6 weeks (Ferguson *et al.*, 1977). Another group of 2 volunteers was exposed to 50 ppm ammonia for 6 hours/day for 6 weeks.

Table 2: Groups of Human Volunteers and the Amount of Time of Exposure

| Group | Exposure | Week 1 | Week 2 | Week 3 | Week 4 | Week 5 | Week 6 |
|---|---|---|---|---|---|---|---|
| A | ppm $NH_3$ | 25 | 50 | 100 | 25 | 50 | 100 |
| | hours | 2 | 4 | 6 | 2 | 4 | 6 |
| B | ppm $NH_3$ | 50 | 50 | 50 | 50 | 50 | 50 |
| | hours | 6 | 6 | 6 | 6 | 6 | 6 |
| C | ppm $NH_3$ | 100 | 50 | 25 | 25 | 50 | 100 |
| | hours | 6 | 4 | 2 | 6 | 4 | 2 |

Pulmonary function tests (respiration rate, FVC and FEV1) were measured in addition to subjective complaints of irritation of the eyes and respiratory tract. The difficulty experienced in performing simple cognitive tasks was also measured, as was pulse rate. There were reports of transient irritation of the nose and throat at 50 or 100 ppm. Acclimation to eye, nose, and throat irritation was seen after two to three weeks (in addition to the short-term subjective adaptation). No significant differences between subjects or controls on common biological indicators, in physical exams, or in performance of normal job duties were found. After acclimation, continuous exposure to 100 ppm, with occasional excursions to 200 ppm, was easily tolerated and had no observed effect on general health.

### 4.1.5 Effects of Animal Exposures

Rats were continuously exposed to ammonia at 0, 25, 50, 150, or 250 ppm (0, 18, 36, 107, or 179 mg/m3) ammonia for 7 days prior to intratracheal inoculation with *Mycoplasma pulmonis*, and from 28 to 42 days following *M. pulmonis* exposure (Broderson *et al.*, 1976). All exposures to ammonia resulted in significantly increased severity of rhinitis, otitis media, tracheitis, and pneumonia characteristic of *M. pulmonis* infection, therefore 25 ppm was a LOAEL (Lowest Observed Adverse Effect Level) in this subchronic study. LOAEL is the lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects on people or animals. Exposure to 250 ppm ammonia alone resulted in nasal lesions (epithelial thickening and hyperplasia) which were not like those seen in *M. pulmonis*-infected rats. The growth of bacteria in the lungs and nasal passages, and the concentration of serum immunoglobulin were significantly increased in rats exposed to 100 ppm (71 mg/m3) ammonia over that seen in control rats (Schoeb *et al.*, 1982).

Guinea pigs (10/group) and mice (20/group) were continuously exposed to 20 ppm (14.2 mg/m3) ammonia for up to 6 weeks (Anderson *et al.*, 1964). Separate groups of 6 guinea pigs and 21 chickens were exposed to 50 ppm and 20 ppm ammonia for up to 6 and 12 weeks, respectively. All species displayed pulmonary edema, congestion,

and haemorrhage after 6 weeks exposure, whereas no effects were seen after only 2 weeks. Guinea pigs exposed to 50 ppm ammonia for 6 weeks exhibited enlarged and congested spleens, congested livers and lungs, and pulmonary edema. Chickens exposed to 200 ppm for 17-21 days showed liver congestion and slight clouding of the cornea. Anderson and associates also showed that a 72-hour exposure to 20 ppm ammonia significantly increased the infection rate of chickens exposed to Newcastle disease virus, while the same effect was observed in chickens exposed to 50 ppm for just 48 hours.

Coon *et al.* (1970) exposed groups of rats (as well as guinea pigs, rabbits, dogs, and monkeys) continuously to ammonia concentrations ranging from 40 to 470 mg/m3. There were no signs of toxicity in 15 rats exposed continuously to 40 mg/m3 for 114 days or in 48 rats exposed continuously to 127 mg/m3 for 90 days. Among 49 rats exposed continuously to 262 mg/m3 for 90 days, 25% had mild nasal discharge. At 455 mg/m3 50 of 51 rats died. Thus 127 mg/m3 (179 ppm) is a subchronic NOAEL (No Observable

Adverse Effect Level) for upper respiratory effects in rats. NOAEL is the highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals. Coon *et al.* (1970) also found no lung effects in 15 guinea pigs exposed continuously to 40 mg/m3 (28 ppm) ammonia for 114 days.

### 4.1.6 Derivation of Chronic Reference Exposure Level

| | |
|---|---|
| *Study* | Holness *et al.*, 1989 (supported by Broderson *et al.*, 1976) |
| *Study population* | 52 workers; 31 controls |
| *Exposure method* | Occupational inhalation |
| *Critical effects* | Pulmonary function, eye, skin, and respiratory symptoms of irritation |
| *LOAEL* | 25 ppm (Broderson *et al.*, 1976) (rats) |
| *NOAEL* | 9.2 ppm (Holness *et al.*, 1989) |
| *Exposure continuity* | 8 hours/day (10 $m^3$/day occupational inhalation rate), 5 days/week |
| *Exposure duration* | 12.2 years |
| *Average occupational exposure* | 3 ppm for NOAEL group (9.2 x 10/20 x 5/7) |
| *Human equivalent concentration* | 3 ppm for NOAEL group |
| *LOAEL uncertainty factor* | 1 |
| *Subchronic uncertainty factor* | 1 |
| *Interspecies uncertainty factor* | 1 |
| *Intraspecies uncertainty factor* | 10 |
| *Cumulative uncertainty factor* | 10 |
| *Inhalation reference exposure level* | 0.3 ppm ( 300 ppb; 0.2 $mg/m^3$; 200 $\mu g/m^3$) |

The Holness *et al.* (1989) study was selected because it was a chronic human study and was published in a respected, peer-reviewed journal. It is also the only chronic study available. The USEPA (1995) based its RfC of 100 mg/m3 on the same study but included a Modifying Factor (MF) of 3 for database deficiencies.

For comparison with the proposed REL of 200 mg/m3 based on human data, we estimated RELs from 2 animal studies. (1) Anderson *et al.* (1964) exposed guinea pigs continuously to 50 ppm (35 mg/m3) ammonia for 6 weeks and observed pulmonary edema. Use of an RGDR of 0.86 and a cumulative uncertainty factor of 3000 (10 for use of a LOAEL, 10 for subchronic, 3 for interspecies, and 10 for intraspecies) resulted in a REL of 10 mg/m3. Staff notes that the nearly maximal total uncertainty factor of 3000 was used in this estimation. (2) Coon *et al.* (1970) exposed rats continuously to 127 mg/m3 ammonia for 90 days and saw no signs of toxicity. Use of an RGDR(ET) of 0.16 for nasal effects (observed in rats exposed to higher levels of ammonia in Broderson *et al.* (1976)) and a cumulative uncertainty factor of

100 (3 for subchronic, 3 for interspecies, and 10 for intraspecies) resulted in a REL of 200 mg/m3.

### 4.1.7 Data Strengths and Limitations for Development of the REL

Significant strengths in the ammonia REL include (1) the availability of long-term human inhalation exposure data (Holness *et al.*, 1989), (2) the demonstration of consistent effects in experimentally exposed human volunteers following short-term exposures (Ferguson *et al.*, 1977), and (3) reasonable consistency with animal data (Coon *et al.*, 1970).

Major areas of uncertainty are (1) the lack of a NOAEL and LOAEL in a single study, (2) a lack of animal data with chronic exposure and histopathological analyses, and (3) difficulties in estimated human occupational exposures. The overall database for this common chemical is limited.

Based on the theoretical values from the acute reference exposure levels for ammonia, a graph of Irritation vs Ammonia Concentration (log ppm) is plotted. Hence, this graph will then be used to correlate with the data calculated from the Gaussian Plume Dispersion Model. This will enable one to see the extent of the severity of the exposure at a certain distance from the point of incident.

## 4.2 Sample Calculation Using the Toxicity Model

For example, and accident with a leakage with a diameter of 3m on an ammonia tank, giving a flowrate of $0.05m^3/s$, shows a graph as below. Referring to the concentration of toxic release vs distance from point of release, the maximum concentration is 484.9 $mg/m^3$ of ammonia (935.132 ppm) at 0.25 km away from the point of release. Hence, log 935.132 will give a value of 2.97. The value of Irritation for maximum likelihood estimate corresponding to 5% lethality is way over 8.0 while the value of Irritation 95% LCL is also way over 2.75

**Toxic Effect Risk Model for Gaseous Releases**
**Diffusion of point source pollution**

**Enter the following inputs:**

| | | | |
|---|---|---|---|
| **Gaseous Release** | | | |
| Name of component | | $NH_3$ | |
| Molecular weight | | 17.03 | g/gmol |
| Specific heat | | 39.904 | kJ/kmol.K |
| Density | | 0.695 | kg/m3 |
| Temperature | | 400 | K |
| Pressure | | 1 | atm |
| Flowrate | | 0.05 | m3/s |
| Distance of leak/rupture from ground level | | 20 | m |
| Diameter of leak/rupture | | 2 | m |
| Duration of exposure | | 10 | s |
| **Atmospheric conditions** | | | |
| Molecular weight of air | | 28.964 | g/gmol |
| Density of air | | 0.695 | kg/m3 |
| Ambient temperature | | 298 | K |
| Specific heat of air | | 38.602 | kJ/kmol.K |
| Wind velocity | | 4 | m/s |
| Wind stability(A-F) | | c | |
| Condition of area (U=urban, C=country) | | u | |
| **Calculated values** | | | |
| Rate of toxic release | | 34.75 | g/s |
| Stack gas exit velocity | | 0.015915 | m/s |
| Flux buoyancy | | 0.039813 | $m^4/s^3$ |
| x* | | 1.867009 | m |
| Plume rise | | 0.477397 | m |
| Plume effect | | 20.4774 | m |

Model / plume.xls / Sheet1 / Chart2 / Chart1 / Sigma-y / Sigma-z /

Figure 14: Toxicity Model Interface

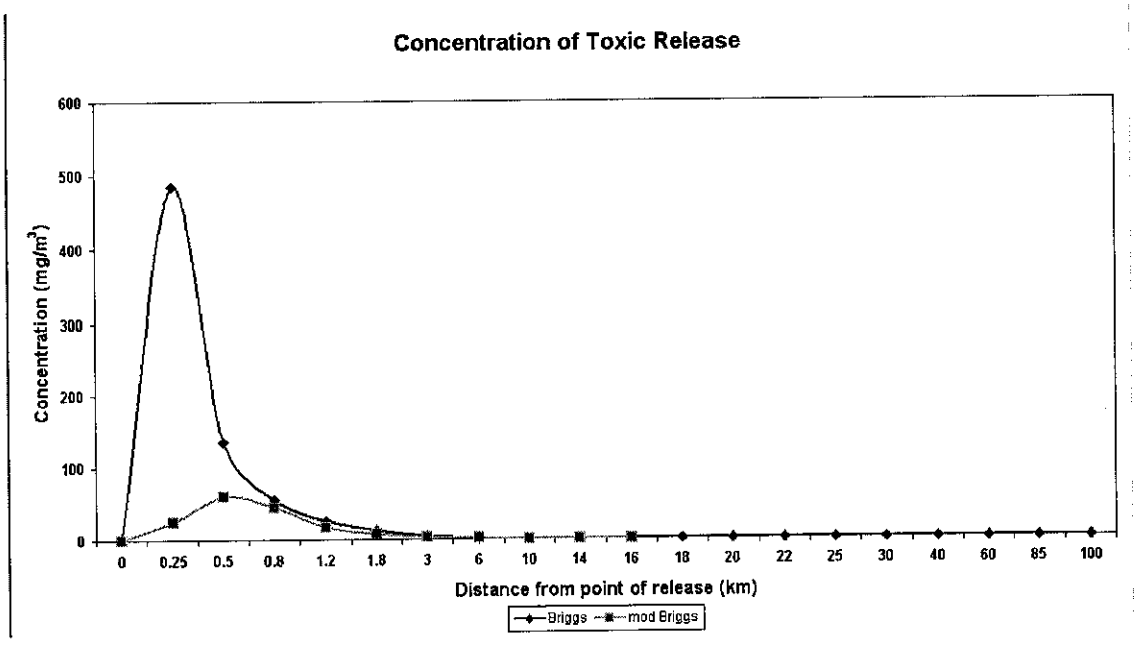



Figure 15: Concentration of Toxic Release

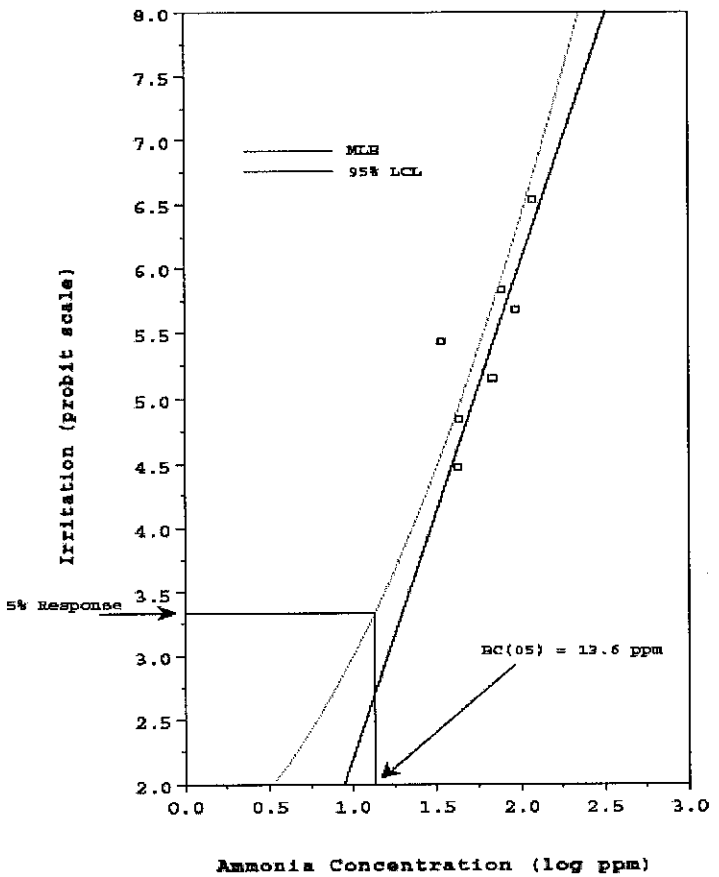



Figure 16: Irritation vs. Ammonia Concentration

This shows that the model is able to give a figure of estimate of the extent of irritation based on the certain inputs of parameters such as diameter of leakage, flowrate of the ammonia from the leak, and also the interested distance from point of release.

### 4.3 Assumptions and Constraints

A host of assumptions and constraints are required to derive the Gaussian dispersion equation for modelling a continuous, buoyant plume from a single point-source in flat terrain. The most important assumptions and constraints are related to:

- The accuracy of predicting the plume rise since that affects the emission height used in the Gaussian dispersion equation.
- The accuracy of the dispersion coefficients (i.e., the vertical and horizontal standard deviations of the emission distribution) used in the Gaussian dispersion equation.

- The assumption of the averaging time period represented by the calculated ground-level pollutant concentrations as determined by the dispersion coefficients used in the Gaussian equation.
- Wind speed and wind direction are constant from the source point to the receptor (for a wind speed of 2 m/s and a distance of 10 km, 80 minutes of constant conditions would be needed).
- Atmospheric turbulence is also constant throughout the plume travel distance.
- The entire plume is conserved, meaning: no deposition or washout of plume components; components reaching the ground are reflected back into the plume; no components are absorbed by bodies of water or by vegetation; and components are not chemically transformed. [Some of the more complex dispersion models do adjust for deposition and chemical transformation. However, such adjustments are separate from the basic Gaussian dispersion equation.]
- Only vertical and crosswind dispersion occurs (i.e., no downwind dispersion). The dispersion pattern is probabilistic and can be described exactly by Gaussian distribution.
- The plume expands in a conical fashion as it travels downward, whereas the ideal "coning plume" is only one of many observed plume behaviours.
- Terrain conditions can be accommodated by using one set of dispersion coefficients for rural terrain and another set for urban terrain. The basic Gaussian dispersion equation is not intended to handle terrain regimes such as valleys, mountains or shorelines.

# CHAPTER 5
# CONCLUSION

Inherent safety is a concept derived from the learning that no matter how well hazards are controlled by extrinsic measures and protective barriers, there is always a possibility for abnormal events that can degenerate into industrial chemical incidents. Kletz's famous expression "what you don't have cannot leak" became the motto of inherent safety because it summarizes the very essence of the approach: hazard elimination rather than control. Because the idea sounds as a very reasonable approach, the application of the inherent safety principle has proven to be complex and difficult to analyze because of the complexity of the chemical industry and the several aspects that must be taken into account.

In Conclusion, this research and modelling project has reached its objective. Hence, this tool can further assist the process of designing an inherently safe chemical plant. The model is able to provide interested party estimates of the concentration of the toxic gas (in this case ammonia) at any distance from the site of incident. Toxicity Model is a tool used during the designing stage of a chemical plant to ensure that the plant can be inherently safe with regards to toxicity. A further development in some of the parameters in the Gaussian plume equation to reduce assumptions is highly recommended. This will help increase the accuracy of the results. For further improvement of the model, one should create a database for acute reference exposure levels for airborne toxicants. In addition, this model should integrate with Inherent Safety Index to cover a wider scope of inherent safety.

Overall, the project has a great potential in becoming a commercial tool that present the integration of Gaussian Plume Dispersion Model with acute reference exposure levels for airborne toxicants, as one of the element for Inherent Safety (IS) in Chemical Process Plant Design (CPPD). Future work is required in order to expand the model, and perhaps upgrade the model so it can be a design tool as well as a monitoring tool.

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## APPENDICES

1. Gaussian Plume Model
2. Fundamentals of Environmental Toxicology

**The Gaussian Equation**

The ISC short term model for stacks uses the steady-state Gaussian plume equation for a continuous elevated source. For each source and each hour, the origin of the source's coordinate system is placed at the ground surface at the base of the stack. The x axis is positive in the downwind direction, the y axis is crosswind (normal) to the x axis and the z axis extends vertically. The fixed receptor locations are converted to each source's coordinate system for each hourly concentration calculation. The hourly concentrations calculated for each source at each receptor are summed to obtain the total concentration produced at each receptor by the combined source emissions.

For a steady-state Gaussian plume, the hourly concentration at downwind distance x (meters) and crosswind distance y (meters) is given by:

$$\chi = \frac{QKVD}{2\pi u_s \sigma_y \sigma_z} \exp\left[-0.5\left(\frac{y}{\sigma_y}\right)^2\right] \quad 1\text{-}1$$

Where:

Q = pollutant emission rate (mass per unit time)

K = a scaling coefficient to convert calculated concentrations to desired units (default value of 1 x $10^6$ for Q in g/s and concentration in $\mu g/m^3$)

V = vertical term

D = decay term

$F_y$,$F_z$ = standard deviation of lateral and vertical concentration distribution (m)

$u_s$ = mean wind speed (m/s) at release height

Equation (1-1) includes a Vertical Term (V), a Decay Term (D), and dispersion parameters ($F_y$ and $F_z$) as discussed below. It should be noted that the Vertical Term includes the effects of source elevation, receptor elevation, plume rise, limited mixing in the vertical,

and the gravitational settling and dry deposition of particulates (with diameters greater than about 0.1 microns).

**Downwind and Crosswind Distances**

The ISC model uses either a polar or a Cartesian receptor network as specified by the user. The model allows for the use of both types of receptors and for multiple networks in a single run. All receptor points are converted to Cartesian (X, Y) coordinates prior to performing the dispersion calculations. In the polar coordinate system, the radial coordinate of the point (r, 2) is measured from the user-specified origin and the angular coordinate 2 is measured clockwise from the north. In the Cartesian coordinate system, the X axis is positive to the east of the user-specified origin and the Y axis is positive to the north. For either type of receptor network, the user must define the location of each source with respect to the origin of the grid using Cartesian coordinates. In the polar coordinate system, assuming the origin is at $X = X_o$, $Y = Y_o$, the X and Y coordinates of a receptor at the point (r, 2) are given by:

$$X(R) = r\sin\theta - X_o \qquad 1\text{-}2$$

$$Y(R) = r\cos\theta - Y_o \qquad 1\text{-}3$$

If the X and Y coordinates of the source are X(S) and Y(S), the downwind distance x to the receptor, along the direction of plume travel, is given by:

$$x = -(X(R) - X(S))\sin(WD) - (Y(R) - Y(S))\cos(WD) \qquad 1\text{-}4$$

where WD is the direction <u>from</u> which the wind is blowing. The downwind distance is used in calculating the distance-dependent plume rise (see Section 1.1.4) and the dispersion parameters (see Section 1.1.5). If any receptor is located within 1 meter of a point source or within 1 meter of the effective radius of a volume source, a warning message is printed and no concentrations are calculated for the source-receptor combination. The crosswind distance y to the receptor from the plume centerline is given by:

$$y = (X(R) - X(S))\cos(WD) - (Y(R) - Y(S))\sin(WD) \qquad 1\text{-}5$$

The crosswind distance is used in Equation (1-1).

**Wind Speed Profile**

The wind power law is used to adjust the observed wind speed, $u_{ref}$, from a reference measurement height, $z_{ref}$, to the stack or release height, $h_s$. The stack height wind speed, $u_s$, is used in the Gaussian plume equation (Equation 1-1), and in the plume rise formulas described in Section 1.1.4. The power law equation is of the form:

$$u_s = u_{ref}\left(\frac{h_s}{z_{ref}}\right)^p \qquad 1\text{-}6$$

where p is the wind profile exponent. Values of p may be provided by the user as a function of stability category and wind speed class. Default values are as follows:

| *Stability Category* | *Rural Exponent* | *Urban Exponent* |
|---|---|---|
| A | 0.07 | 0.15 |
| B | 0.07 | 0.15 |
| C | 0.10 | 0.20 |
| D | 0.15 | 0.25 |
| E | 0.35 | 0.30 |
| F | 0.55 | 0.30 |

The stack height wind speed, $u_s$, is not allowed to be less than 1.0 m/s.

**THE DISPERSION PARAMETERS**

***Point Source Dispersion Parameters.***

Equations that approximately fit the Pasquill-Gifford curves (Turner, 1970) are used to calculate $F_y$ and $F_z$ (in meters) for the rural mode. The equations used to calculate $F_y$ are of the form:

$$\sigma_y = 465.11628(x)\tan(TH) \qquad 1\text{-}32$$

where:

$$TH = 0.017453293[c - d\ln(x)] \qquad 1\text{-}33$$

In Equations (1-32) and (1-33) the downwind distance x is in kilometers, and the coefficients c and d are listed in Table 1. The equation used to calculate $F_z$ is of the form:

$$\sigma_z = ax^b \qquad 1\text{-}34$$

where the downwind distance x is in kilometers and $F_z$ is in meters. The coefficients a and b are given in Table 2. Tables 3 and 4 show the equations used to determine $F_y$ and $F_z$ for the urban option. These expressions were determined by Briggs as reported by Gifford (1976) and represent a best fit to urban vertical diffusion data reported by McElroy and Pooler (1968). While the Briggs functions are assumed to be valid for downwind distances less than 100m, the user is cautioned that concentrations at receptors less than 100m from a source may be suspect.

**TABLE 1**

**Parameters used to calculate pasquill-gifford.**

| Pasquill Category | Stability | $F_y = 465.11628\ (x)\tan(TH)$<br>$TH = 0.017453293\ [c - d\ln(x)]$ | |
|---|---|---|---|
| | | c | d |
| A | | 24.1670 | 2.5334 |
| B | | 18.3330 | 1.8096 |
| C | | 12.5000 | 1.0857 |
| D | | 8.3330 | 0.72382 |
| E | | 6.2500 | 0.54287 |
| F | | 4.1667 | 0.36191 |

where $F_y$ is in meters and x is in kilometers

**TABLE** **2**

**Parameters used to calculate pasquill-gifford**

| Pasquill Stability Category | x (km) | $F_z(\text{meters}) = ax^b$ (x in km) a | b |
|---|---|---|---|
| | <.10 | 122.800 | 0.94470 |
| | 0.10 - 0.15 | 158.080 | 1.05420 |
| | 0.16 - 0.20 | 170.220 | 1.09320 |
| | 0.21 - 0.25 | 179.520 | 1.12620 |
| A* | 0.26 - 0.30 | 217.410 | 1.26440 |
| | 0.31 - 0.40 | 258.890 | 1.40940 |
| | 0.41 - 0.50 | 346.750 | 1.72830 |
| | 0.51 - 3.11 | 453.850 | 2.11660 |
| | >3.11 | ** | ** |
| | <.20 | 90.673 | 0.93198 |
| B* | 0.21 - 0.40 | 98.483 | 0.98332 |
| | >0.40 | 109.300 | 1.09710 |
| C* | All | 61.141 | 0.91465 |

| | | | |
|---|---|---|---|
| | .30 | 34.459 | 0.86974 |
| | 0.31 - 1.00 | 32.093 | 0.81066 |
| | 1.01 - 3.00 | 32.093 | 0.64403 |
| D | 3.01 - 10.00 | 33.504 | 0.60486 |
| | 10.01 - 30.00 | 36.650 | 0.56589 |
| | >30.00 | 44.053 | 0.51179 |
| | <.10 | 24.260 | 0.83660 |
| | 0.10 - 0.30 | 23.331 | 0.81956 |
| | 0.31 - 1.00 | 21.628 | 0.75660 |
| | 1.01 - 2.00 | 21.628 | 0.63077 |
| E | 2.01 - 4.00 | 22.534 | 0.57154 |
| | 4.01 - 10.00 | 24.703 | 0.50527 |
| | 10.01 - 20.00 | 26.970 | 0.46713 |
| | 20.01 - 40.00 | 35.420 | 0.37615 |
| | >40.00 | 47.618 | 0.29592 |
| | <.20 | 15.209 | 0.81558 |
| | 0.21 - 0.70 | 14.457 | 0.78407 |
| | 0.71 - 1.00 | 13.953 | 0.68465 |
| | 1.01 - 2.00 | 13.953 | 0.63227 |
| | 2.01 - 3.00 | 14.823 | 0.54503 |
| F | 3.01 - 7.00 | 16.187 | 0.46490 |
| | 7.01 - 15.00 | 17.836 | 0.41507 |
| | 15.01 - 30.00 | 22.651 | 0.32681 |
| | 30.01 - 60.00 | 27.074 | 0.27436 |
| | >60.00 | 34.219 | 0.21716 |

* If the calculated value of $F_z$ exceed 5000 m, $F_z$ is set to 5000 m.

** $F_z$ is equal to 5000 m.

**TABLE 3**

| Pasquill Stability Category | $F_y$(meters)* |
|---|---|
| A | $0.32 \times (1.0 + 0.0004 x)^{-1/2}$ |
| B | $0.32 \times (1.0 + 0.0004 x)^{-1/2}$ |
| C | $0.22 \times (1.0 + 0.0004 x)^{-1/2}$ |
| D | $0.16 \times (1.0 + 0.0004 x)^{-1/2}$ |
| E | $0.11 \times (1.0 + 0.0004 x)^{-1/2}$ |
| F | $0.11 \times (1.0 + 0.0004 x)^{-1/2}$ |

* Where x is in meters

**TABLE 4**

| Pasquill Stability Category | $F_z$(meters)* |
|---|---|
| A | $0.24 \times (1.0 + 0.001 x)^{1/2}$ |
| B | $0.24 \times (1.0 + 0.001 x)^{1/2}$ |
| C | $0.20 x$ |
| D | $0.14 \times (1.0 + 0.0003 x)^{-1/2}$ |
| E | $0.08 \times (1.0 + 0.0015 x)^{-1/2}$ |
| F | $0.08 \times (1.0 + 0.0015 x)^{-1/2}$ |

* Where x is in meters.

***Lateral and Vertical Virtual Distances***

The equations in Tables 1 through 4 define the dispersion parameters for an ideal point source. However, volume sources have initial lateral and vertical dimensions. Also, as discussed below, building wake effects can enhance the initial growth of stack plumes. In these cases, lateral ($x_y$) and vertical ($x_z$) virtual distances are added by the ISC models to the actual downwind distance x for the $F_y$ and $F_z$ calculations. The lateral virtual distance in kilometers for the rural mode is given by:

$$x_y = \left( \frac{\sigma_{yo}}{p} \right)^{1/q} \qquad 1\text{-}35$$

where the stability-dependent coefficients p and q are given in Table 1-5 and $F_{yo}$ is the standard deviation in meters of the lateral concentration distribution at the source. Similarly, the vertical virtual distance in kilometers for the rural mode is given by:

$$x_z = \left( \frac{\sigma_{zo}}{a} \right)^{1/b} \qquad 1\text{-}36$$

where the coefficients a and b are obtained form Table 1-2 and $F_{zo}$ is the standard deviation in meters of the vertical concentration distribution at the source. It is important to note that the ISC model programs check to ensure that the $x_z$ used to calculate $F_z$ at $(x + x_z)$ in the rural mode is the $x_z$ calculated using the coefficients a and b that correspond to the distance category specified by the quantity $(x + x_z)$.

To determine virtual distances for the urban mode, the functions displayed in Tables 3 and 4 are solved for x. The solutions are quadratic formulas for the lateral virtual distances; and for vertical virtual distances the solutions are cubic equations for stability classes A and B, a linear equation for stability class C, and quadratic equations for stability classes D, E, and F. The cubic equations are solved by iteration using Newton's method.

**TABLE 5**

**FUNC {x_y~=~LEFT ( {{F_{yo}} over p} right ) ^{``1/q}}**

| **Pasquill Stability Category** | **p** | **q** |
|---|---|---|

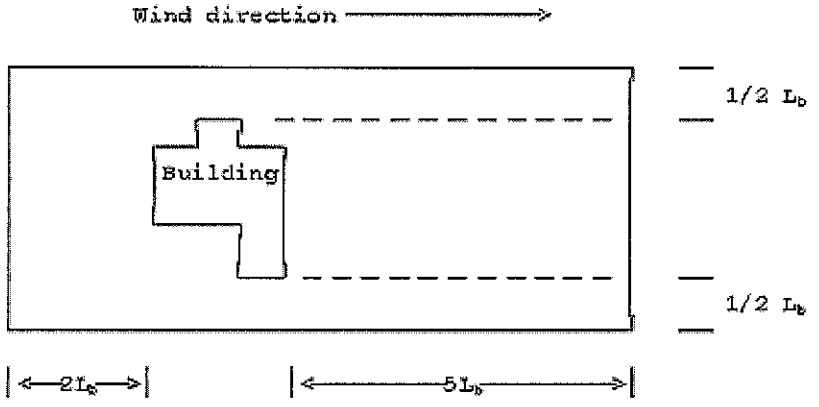
| | | |
|---|---|---|
| A | 209.14 | 0.890 |
| B | 154.46 | 0.902 |
| C | 103.26 | 0.917 |
| D | 68.26 | 0.919 |
| E | 51.06 | 0.921 |
| F | 33.92 | 0.919 |

***Procedures Used to Account for the Effects of Building Wakes on Effluent Dispersion***

The procedures used by the ISC models to account for the effects of the aerodynamic wakes and eddies produced by plant buildings and structures on plume dispersion originally followed the suggestions of Huber (1977) and Snyder (1976). Their suggestions are principally based on the results of wind-tunnel experiments using a model building with a crosswind dimension double that of the building height. The atmospheric turbulence simulated in the wind-tunnel experiments was intermediate between the turbulence intensity associated with the slightly unstable Pasquill C category and the turbulence intensity associated with the neutral D category. Thus, the data reported by Huber and Snyder reflect a specific stability, building shape and building orientation with respect to the mean wind direction. It follows that the ISC wake-effects evaluation procedures may not be strictly applicable to all situations. The ISC models also provide for the revised treatment of building wake effects for certain sources, which uses modified plume rise algorithms, following the suggestions of Schulman and Hanna (1986). This treatment is largely based on the work of Scire and Schulman (1980). When the stack height is less than the building height plus half the lesser of the building height or width, the methods of Schulman and Scire are followed. Otherwise, the methods of Huber and Snyder are followed. In the ISC models, direction-specific building dimensions may be used with either the Huber-Snyder or Schulman-Scire downwash algorithms.

The wake-effects evaluation procedures may be applied by the user to any stack on or adjacent to a building. For regulatory application, a building is considered sufficiently close to a stack to cause wake effects when the distance between the stack and the nearest

part of the building is less than or equal to five times the lesser of the height or the projected width of the building. For downwash analyses with direction-specific building dimensions, wake effects are assumed to occur if the stack is within a rectangle composed of two lines perpendicular to the wind direction, one at $5L_b$ downwind of the building and the other at $2L_b$ upwind of the building, and by two lines parallel to the wind direction, each at $0.5L_b$ away from each side of the building, as shown below:




$L_b$ is the lesser of the height and projected width of the building for the particular direction sector. For additional guidance on determining whether a more complex building configuration is likely to cause wake effects, the reader is referred to the Guideline for Determination of Good Engineering Practice Stack Height (Technical Support Document for the Stack Height Regulations) - Revised (EPA, 1985). In the following sections, the Huber and Snyder building downwash method is described followed by a description of the Schulman and Scire building downwash method.

***Huber and Snyder building downwash procedures***

The first step in the wake-effects evaluation procedures used by the ISC model programs is to calculate the gradual plume rise due to momentum alone at a distance of two building heights using Equation (1-23) or Equation (1-25). If the plume height, $h_e$, given by the sum of the stack height (with no stack-tip downwash adjustment) and the momentum rise is greater than either 2.5 building heights ($2.5\ h_b$) or the sum of the building height and 1.5 times the building width ($h_b + 1.5\ h_w$), the plume is assumed to be unaffected by the building wake. Otherwise the plume is assumed to be affected by the building wake.

The ISC model programs account for the effects of building wakes by modifying both $F_y$ and $F_z$ for plumes with plume height to building height ratios less than or equal to 1.2 and by modifying only $F_z$ for plumes from stacks with plume height to building height ratios greater than 1.2 (but less than 2.5). The plume height used in the plume height to stack

height ratios is the same plume height used to determine if the plume is affected by the building wake. The ISC models define buildings as squat ($h_w$ $ $h_b$) or tall ($h_w < h_b$). The ISC models include a general procedure for modifying $F_z$ and $F_y$ at distances greater than or equal to $3h_b$ for squat buildings or $3h_w$ for tall buildings. The air flow in the building cavity region is both highly turbulent and generally recirculating. The ISC models are not appropriate for estimating concentrations within such regions. The ISC assumption that this recirculating cavity region extends to a downwind distance of $3h_b$ for a squat building or $3h_w$ for a tall building is most appropriate for a building whose width is not much greater than its height. The ISC user is cautioned that, for other types of buildings, receptors located at downwind distances of $3h_b$ (squat buildings) or $3h_w$ (tall buildings) may be within the recirculating region.

The modified $F_z$ equation for a squat building is given by:

$$\sigma_z' = 0.7h_b + 0.067(x-3h_b) \quad \text{for} \quad 3h_b \le x < 10h_b$$

or 1-37

$$= \sigma_z(x + x_z) \quad \text{for} \quad x \ge 10h_b$$

where the building height $h_b$ is in meters. For a tall building, Huber (1977) suggests that the width scale $h_w$ replace $h_b$ in Equation (1-37). The modified $F_z$ equation for a tall building is then given by:

$$\sigma_z' = 0.7h_w + 0.067(x-3h_w) \quad \text{for} \quad 3h_w \le x < 10h_w$$

or 1-38

$$= \sigma_z(x + x_z) \quad \text{for} \quad x \ge 10h_w$$

where $h_w$ is in meters. It is important to note that $F_z'$ is not permitted to be less than the point source value given in Tables 1-2 or 1-4, a condition that may occur.

The vertical virtual distance, $x_z$, is added to the actual downwind distance x at downwind distances beyond $10h_b$ for squat buildings or beyond $10h_w$ for tall buildings, in order to account for the enhanced initial plume growth caused by the building wake. The virtual distance is calculated from solutions to the equations for rural or urban sigmas provided earlier.

As an example for the rural options, Equations (1-34) and (1-37) can be combined to derive the vertical virtual distance $x_z$ for a squat building. First, it follows from Equation

(1-37) that the enhanced $F_z$ is equal to $1.2h_b$ at a downwind distance of $10h_b$ in meters or $0.01h_b$ in kilometers. Thus, $x_z$ for a squat building is obtained from Equation (1-34) as follows:

$$\sigma_z \{0.01h_b\} = 1.2h_b = a(0.01h_b + x_z)^b \qquad 1\text{-}39$$

$$x_z = \left(\frac{1.2h_b}{a}\right)^{1/b} - 0.01h_b \qquad 1\text{-}40$$

where the stability-dependent constants a and b are given in Table 2. Similarly, the vertical virtual distance for tall buildings is given by:

$$x_z = \left(\frac{1.2h_w}{a}\right)^{1/b} - 0.01h_w \qquad 1\text{-}41$$

For the urban option, $x_z$ is calculated from solutions to the equations in Table 4 for $F_z = 1.2h_b$ or $F_z = 1.2\ h_w$ for tall or squat buildings, respectively.

For a squat building with a building width to building height ratio ($h_w/h_b$) less than or equal to 5, the modified $F_y$ equation is given by:

$$\sigma_y' = 0.35h_w + 0.067(x-3h_b) \quad \text{for } 3h_b \le x < 10h_b$$

$$\text{or} \qquad 1\text{-}42$$

$$= \sigma_y(x + x_y) \quad \text{for } x \ge 10h_b$$

The lateral virtual distance is then calculated for this value of $F_y$.

For a building that is much wider than it is tall ($h_w/h_b$ greater than 5), the presently available data are insufficient to provide general equations for $F_y$. For a stack located toward the center of such a building (i.e., away form either end), only the height scale is considered to be significant. The modified $F_y$ equation for a very squat building is then given by:

$$\sigma_y' = 0.35h_b + 0.067(x-3h_b) \quad \text{for } 3h_b \le x < 10h_b$$

$$\text{or} \qquad 1\text{-}43$$

$$= \sigma_y(x + x_y) \quad \text{for } x \ge 10h_b$$

For $h_w/h_b$ greater than 5, and a stack located laterally within about 2.5 $h_b$ of the end of the building, lateral plume spread is affected by the flow around the end of the building. With

end effects, the enhancement in the initial lateral spread is assumed not to exceed that given by Equation (1-42) with $h_w$ replaced by 5 $h_b$. The modified $F_y$ equation is given by:

$$\sigma_y' = 1.75h_b + 0.067(x-3h_b) \quad \text{for} \quad 3h_b \le x < 10h_b$$

or

$$= \sigma_y(x + x_y) \quad \text{for} \quad x \ge 10h_b \qquad \text{1-44}$$

The upper and lower bounds of the concentrations that can be expected to occur near a building are determined respectively using Equations (1-43) and (1-44). The user must specify whether Equation (1-43) or Equation (1-44) is to be used in the model calculations. In the absence of user instructions, the ISC models use Equation (1-43) if the building width to building height ratio $h_w/h_b$ exceeds 5.

Although Equation (1-43) provides the highest concentration estimates for squat buildings with building width to building height ratios ($h_w/h_b$) greater than 5, the equation is applicable only to a stack located near the center of the building when the wind direction is perpendicular to the long side of the building (i.e., when the air flow over the portion of the building containing the source is two dimensional). Thus, Equation (1-44) generally is more appropriate then Equation (1-43). It is believed that Equations (1-43) and (1-44) provide reasonable limits on the extent of the lateral enhancement of dispersion and that these equations are adequate until additional data are available to evaluate the flow near very wide buildings.

The modified $F_y$ equation for a tall building is given by:

$$\sigma_y' = 0.35h_w + 0.067(x-3h_w) \quad \text{for} \quad 3h_w \le x < 10h_w$$

or

$$= \sigma_y(x + x_y) \quad \text{for} \quad x \ge 10h_w \qquad \text{1-45}$$

The ISC models print a message and do not calculate concentrations for any source-receptor combination where the source-receptor separation is less than 1 meter, and also for distances less than 3 $h_b$ for a squat building or 3 $h_w$ for a tall building under building wake effects. It should be noted that, for certain combinations of stability and building height and/or width, the vertical and/or lateral plume dimensions indicated for a point source by the dispersion curves at a downwind distance of ten building heights or widths can exceed the values given by Equation (1-37) or (1-38) and by Equation (1-42) or (1-43). Consequently, the ISC models do not permit the virtual distances $x_y$ and $x_z$ to be less than zero.

***Schulman and Scire refined building downwash procedures***

The procedures for treating building wake effects include the use of the Schulman and Scire downwash method. The building wake procedures only use the Schulman and Scire method when the physical stack height is less than $h_b + 0.5\ L_B$, where $h_b$ is the building height and $L_B$ is the lesser of the building height or width. In regulatory applications, the maximum projected width is used. The features of the Schulman and Scire method are: (1) reduced plume rise due to initial plume dilution, (2) enhanced vertical plume spread as a linear function of the effective plume height, and (3) specification of building dimensions as a function of wind direction. The reduced plume rise equations were previously described in Section 1.1.4.11.

When the Schulman and Scire method is used, the ISC dispersion models specify a linear decay factor, to be included in the $F_z$'s calculated using Equations (1-37) and (1-38), as follows:

$$\sigma_z'' = A\sigma_z' \qquad 1\text{-}46$$

where $F_z'$ is from either Equation (1-37) or (1-38) and A is the linear decay factor determined as follows:

$$\begin{aligned} A &= 1 && \text{if } h_e \le h_b \\ A &= \frac{h_b - h_e}{2L_B} + 1 && \text{if } h_b < h_e \le h_b + 2L_B \qquad 1\text{-}47 \\ A &= 0 && \text{if } h_e > h_b + 2L_B \end{aligned}$$

where the plume height, $h_e$, is the height due to gradual momentum rise at 2 $h_b$ used to check for wake effects. The effect of the linear decay factor is illustrated in Figure 1-1. For Schulman-Scire downwash cases, the linear decay term is also used in calculating the vertical virtual distances with Equations (1-40) to (1-41).

When the Schulman and Scire building downwash method is used the ISC models require direction specific building heights and projected widths for the downwash calculations. The ISC models also accept direction specific building dimensions for Huber-Snyder downwash cases. The user inputs the building height and projected widths of the building tier associated with the greatest height of wake effects for each ten degrees of wind direction. These building heights and projected widths are the same as are used for GEP stack height calculations. The user is referred to EPA (1986) for calculating the appropriate

building heights and projected widths for each direction. Figure 1-2 shows an example of a two tiered building with different tiers controlling the height that is appropriate for use for different wind directions. For an east or west wind the lower tier defines the appropriate height and width, while for a north or south wind the upper tier defines the appropriate values for height and width.

***Procedures Used to Account for Buoyancy-Induced Dispersion.***

The method of Pasquill (1976) is used to account for the initial dispersion of plumes caused by turbulent motion of the plume and turbulent entrainment of ambient air. With this method, the effective vertical dispersion $F_{ze}$ is calculated as follows:

$$\sigma_{ze} = \left[\sigma_z^2 + \left(\frac{\Delta h}{3.5}\right)^2\right]^{1/2} \qquad 1\text{-}48$$

where $F_z$ is the vertical dispersion due to ambient turbulence and )h is the plume rise due to momentum and/or buoyancy. The lateral plume spread is parameterized using a similar expression:

$$\sigma_{ye} = \left[\sigma_y^2 + \left(\frac{\Delta h}{3.5}\right)^2\right]^{1/2} \qquad 1\text{-}49$$

where $F_y$ is the lateral dispersion due to ambient turbulence. It should be noted that )h is the distance-dependent plume rise if the receptor is located between the source and the distance to final rise, and final plume rise if the receptor is located beyond the distance to final rise. Thus, if the user elects to use final plume rise at all receptors the distance-dependent plume rise is used in the calculation of buoyancy-induced dispersion and the final plume rise is used in the concentration equations. It should also be noted that buoyancy-induced dispersion is not used when the Schulman-Scire downwash option is in effect.

**The Vertical Term**

- The Vertical Term Without Dry Deposition
- The Vertical Term in Elevated Simple Terrain
- The Vertical Term With Dry Deposition

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The Vertical Term (V), which is included in Equation (1-1), accounts for the vertical distribution of the Gaussian plume. It includes the effects of source elevation, receptor

elevation, plume rise (Section 1.1.4), limited mixing in the vertical, and the gravitational settling and dry deposition of particulates. In addition to the plume height, receptor height and mixing height, the computation of the Vertical Term requires the vertical dispersion parameter ($_z$) described in Section 1.1.5.

***The Vertical Term Without Dry Deposition.***

In general, the effects on ambient concentrations of gravitational settling and dry deposition can be neglected for gaseous pollutants and small particulates (less than about 0.1 microns in diameter). The Vertical Term without deposition effects is then given by:

$$V = \exp\left[-0.5\left(\frac{z_r - h_e}{\sigma_z}\right)^2\right] + \exp\left[-0.5\left(\frac{z_r + h_e}{\sigma_z}\right)^2\right]$$

$$+ \sum_{i=1}^{m}\left\{\exp\left[-0.5\left(\frac{H_1}{\sigma_z}\right)^2\right] + \exp\left[-0.5\left(\frac{H_2}{\sigma_z}\right)^2\right]\right. \quad \text{1-50}$$

$$\left. + \exp\left[-0.5\left(\frac{H_3}{\sigma_z}\right)^2\right] + \exp\left[-0.5\left(\frac{H_4}{\sigma_z}\right)^2\right]\right\}$$

where:

$h_e = h_s + \Delta h$

$H_1 = z_r - (2iz_i - h_e)$

$H_2 = z_r + (2iz_i - h_e)$

$H_3 = z_r - (2iz_i + h_e)$

$H_4 = z_r + (2iz_i + h_e)$

$z_r$ = receptor height above ground (flagpole) (m)

$z_i$ = mixing height (m)

The infinite series term in Equation (1-50) accounts for the effects of the restriction on vertical plume growth at the top of the mixing layer. As shown by Figure 1-3, the method of image sources is used to account for multiple reflections of the plume from the ground surface and at the top of the mixed layer. It should be noted that, if the effective stack height, $h_e$, exceeds the mixing height, $z_i$, the plume is assumed to fully penetrate the elevated inversion and the ground-level concentration is set equal to zero.

Equation (1-50) assumes that the mixing height in rural and urban areas is known for all stability categories. As explained below, the meteorological preprocessor program uses mixing heights derived from twice-daily mixing heights calculated using the Holzworth (1972) procedures. The ISC models currently assume unlimited vertical mixing under stable conditions, and therefore delete the infinite series term in Equation (1-50) for the E and F stability categories.

The Vertical Term defined by Equation (1-50) changes the form of the vertical concentration distribution from Gaussian to rectangular (i.e., a uniform concentration within the surface mixing layer) at long downwind distances. Consequently, in order to reduce computational time without a loss of accuracy, Equation (1-50) is changed to the form:

$$V = \frac{\sqrt{2\pi}\sigma_z}{z_i} \qquad 1\text{-}51$$

at downwind distances where the $_z/z_i$ ratio is greater than or equal to 1.6.

The meteorological preprocessor program, RAMMET, used by the ISC Short Term model uses an interpolation scheme to assign hourly rural and urban mixing heights on the basis of the early morning and afternoon mixing heights calculated using the Holzworth (1972) procedures. The procedures used to interpolate hourly mixing heights in urban and rural areas are illustrated in Figure 1-4, where:

$H_m\{max\}$ = maximum mixing height on a given day
$H_m\{min\}$ = minimum mixing height on a given day
MN = midnight
SR = sunrise
SS = sunset

The interpolation procedures are functions of the stability category for the hour before sunrise. If the hour before sunrise is neutral, the mixing heights that apply are indicated by the dashed lines labeled neutral in Figure 1-4. If the hour before sunrise is stable, the mixing heights that apply are indicated by the dashed lines labeled stable. It should be

pointed out that there is a discontinuity in the rural mixing height at sunrise if the preceding hour is stable. As explained above, because of uncertainties about the applicability of Holzworth mixing heights during periods of E and F stability, the ISC models ignore the interpolated mixing heights for E and F stability, and treat such cases as having unlimited vertical mixing.

***The Vertical Term in Elevated Simple Terrain.***

The ISC models make the following assumption about plume behavior in elevated simple terrain (i.e., terrain that exceeds the stack base elevation but is below the release height):

- The plume axis remains at the plume stabilization height above mean sea level as it passes over elevated or depressed terrain.
- The mixing height is terrain following.
- The wind speed is a function of height above the surface (see Equation (1-6)).

Thus, a modified plume stabilization height $h_e'$ is substituted for the effective stack height $h_e$ in the Vertical Term given by Equation (1-50). For example, the effective plume stabilization height at the point x, y is given by:

$$h_e' = h_e + z_s - z|_{(x,y)} \quad 1\text{-}52$$

where:

$z_s$ = height above mean sea level of the base of the stack (m)

$z_{(x,y)}$ = height above mean sea level of terrain at the receptor location (x,y) (m)

It should also be noted that, as recommended by EPA, the ISC models "truncate" terrain at stack height as follows: if the terrain height z - $z_s$ exceeds the source release height, $h_s$, the elevation of the receptor is automatically "chopped off" at the physical release height. The user is cautioned that concentrations at these complex terrain receptors are subject to considerable uncertainty. Figure 1-5 illustrates the terrain-adjustment procedures used by the ISC models for simple elevated terrain. The vertical term used with the complex terrain algorithms in ISC is described in Section 1.5.6.

***The Vertical Term With Dry Deposition.***

Particulates are brought to the surface through the combined processes of turbulent diffusion and gravitational settling. Once near the surface, they may be removed from the atmosphere and deposited on the surface. This removal is modeled in terms of a deposition velocity ($v_d$), which is described in Section 1.3.1, by assuming that the deposition flux of

material to the surface is equal to the product $v_{dd}$, where $_d$ is the airborne concentration just above the surface. As the plume of airborne particulates is transported downwind, such deposition near the surface reduces the concentration of particulates in the plume, and thereby alters the vertical distribution of the remaining particulates. Furthermore, the larger particles will also move steadily nearer the surface at a rate equal to their gravitational settling velocity ($v_g$). As a result, the plume centerline height is reduced, and the vertical concentration distribution is no longer Gaussian.

A corrected source-depletion model developed by Horst (1983) is used to obtain a "vertical term" that incorporates both the gravitational settling of the plume and the removal of plume mass at the surface. These effects are incorporated as modifications to the Gaussian plume equation. First, gravitational settling is assumed to result in a "tilted plume", so that the effective plume height ($h_e$) in Equation

(1-50) is replaced by

$$h_{ed} = h_e - h_v = h_e - \frac{x}{u_s} v_g \quad 1\text{-}53$$

where $h_v = (x/u_s)v_g$ is the adjustment of the plume height due to gravitational settling. Then, a new vertical term ($V_d$) that includes the effects of dry deposition is defined as:

$$V_d(x, z, h_{ed}) = V(x, z, h_{ed})\, F_Q(x)\, P(x, z) \quad 1\text{-}54$$

$V(x,z,h_{ed})$ is the vertical term in the absence of any deposition--it is just Equation (1-50), with the tilted plume approximation. $F_Q(x)$ is the fraction of material that remains in the plume at the downwind distance x (i.e., the mass that has not yet been deposited on the surface). This factor may be thought of as a source depletion factor, a ratio of the "current" mass emission rate to the original mass emission rate. P(x,z) is a vertical profile adjustment factor, which modifies the reflected Gaussian distribution of Equation

(1-50), so that the effects of dry deposition on near-surface concentrations can be simulated.

For large travel-times, $h_{ed}$ in Equation (1-53) can become less than zero. However, the tilted plume approximation is not a valid approach in this region. Therefore, a minimum value of zero is imposed on $h_{ed}$. In effect, this limits the settling of the plume centerline, although the deposition velocity continues to account for gravitational settling near the surface. The effect of gravitational settling beyond the plume touchdown point (where $h_{ed} = 0$) is to modify the vertical structure of the plume, which is accounted for by modifying the vertical dispersion parameter ($_z$).

The process of adjusting the vertical profile to reflect loss of plume mass near the surface is illustrated in Figures 1-6 and 1-7. At a distance far enough downwind that the plume size in the vertical has grown larger than the height of the plume, significant corrections to the concentration profile may be needed to represent the removal of material from the plume due to deposition. Figure 1-6 displays a depletion factor $F_Q$, and the corresponding profile correction factor P(z) for a distance at which $_z$ is 1.5 times the plume height. The depletion factor is constant with height, whereas the profile correction shows that most of the material is lost from the lower portion of the plume. Figure 1-7 compares the vertical profile of concentration both with and without deposition and the corresponding depletion of material from the plume. The depleted plume profile is computed using Equation (1-54). Both $F_Q(x)$ and P(x,z) depend on the size and density of the particles being modeled, because this effects the total deposition velocity (See Section 1.3.2). Therefore, for a given source of particulates, ISC allows multiple particle-size categories to be defined, with the maximum number of particle size categories controlled by a parameter statement in the model code (see Volume I). The user must provide the mass-mean particle diameter (microns), the particle density ($g/cm^3$), and the mass fraction () for each category being modeled. If we denote the value of $F_Q(x)$ and P(x,z) for the $n^{th}$ particle-size category by $F_{Qn}(x)$ and $P_n(x,z)$ and substitute these in Equation (1-54), we see that a different value for the vertical term is obtained for each particle-size category, denoted as $V_{dn}$. Therefore, the total vertical term is given by the sum of the terms for each particle-size category, weighted by the respective mass-fractions:

$$V_d(x, z, h_{ed}) = \sum_{n=1}^{N} \varphi_n V_{dn}(x, z, h_{ed}) \quad 1\text{-}55$$

$F_Q(x)$ is a function of the total deposition velocity ($v_d$), $V(x,z_d,h_{ed})$, and $P(x,z_d)$:

$$F_Q(x) = EXP\left[ - \int_0^x v_d \, V(x', z_d, h_{ed}) \, P(x', z_d) \, dx' \right] \quad 1\text{-}56$$

where $z_d$ is a height near the surface at which the deposition flux is calculated. The deposition reference height is calculated as the maximum of 1.0 meters and $20z_0$. This equation reflects the fact that the material removed from the plume by deposition is just the integral of the deposition flux over the distance that the plume has traveled. In ISC, this integral is evaluated numerically. For sources modeled in elevated or complex terrain, the user can input a terrain grid to the model, which is used to determine the terrain elevation at various distances along the plume path during the evaluation of the integral. If a terrain

grid is not input by the user, then the model will linearly interpolate between the source elevation and the receptor elevation.

The profile correction factor P(x,z) is given by

$$x, z) = E(x, z_d)\left[1 + \frac{v_d - v_g}{v_g}\left(1 - EXP\left[-v_g\, R(z, z_d)\right]\right)\right]$$

$$<, z_d) = \left[1 + \frac{v_d - v_g}{v_g}\int_0^{\infty} \frac{V(x, z', o)}{\sqrt{2\pi}\sigma_z}\left(1 - EXP\left[-v_g R(z', z_d)\right]\right) dz'\right]^{-1} \quad \text{1-57a}$$

where $R(z,z_d)$ is an atmospheric resistance to vertical transport that is derived from Briggs' formulas for $_z$ (Gifford, 1976). When the product $v_gR(z,z_d)$ is of order 0.1 or less, the exponential function is approximated (for small argument) to simplify P(x,z):

$$P(x, z) \approx P(x, z_d)\left[1 + (v_d - v_g)\, R(z, z_d)\right]$$

$$P(x, z_d) \approx \left[1 + (v_d - v_g)\int_0^{\infty} \frac{V(x, z', o)}{\sqrt{2\pi}\sigma_z} R(z', z_d)\, dz'\right]^{-1} \quad \text{1-57b}$$

This simplification is important, since the integral in Equation (1-57a) is evaluated numerically, whereas that in Equation (1-57b) is computed using analytical approximations.

The resistance $R(z,z_d)$ is obtained for the following functional forms of $_z$ defined by Briggs:

$$\sigma_z = ax/(1 + bx)^{1/2}$$

$$R(z, z_d) = \sqrt{\frac{2}{\pi}}\frac{1}{au}\left[\ln(z/z_d) + \frac{b}{a}\sqrt{\frac{\pi}{2}}(z - z_d)\right] \quad \text{1-58}$$

For this last form, the x(z) and $x(z_d)$ must be solved for z and $z_d$ (respectively) by finding the root of the implicit relation

$$\sqrt{\frac{\pi}{2}}\, z = a\, x\, \sqrt{1 + bx} \quad \text{1-59}$$

The corresponding functions for $P(x,z_d)$ for the special case of Equation (1-57) are given by:

Case 1:
Rural: stability A, B
Urban: stability C

$$\sigma_z = ax$$

$$P^{-1}(x, z_d) = 1 + \frac{v_d - v_g}{ua}\sqrt{\frac{2}{\pi}}\left[\ln\left(\sqrt{2}\,\sigma_z / z_d\right) - 1\right]$$

Case 2:
Rural: stability C, D
Urban: stability D, E, F

$$\sigma_z = ax/(1 + bx)^{1/2}$$

$$P^{-1}(x, z_d) = 1 + \frac{v_d - v_g}{ua}\sqrt{\frac{2}{\pi}}\left[\ln\left(\sqrt{2}\sigma_z / z_d\right) - 1 + \frac{b}{a}\left(\sigma_z - \sqrt{\frac{\pi}{2}}\, z_d\right)\right]$$

Case 3:
Rural: stability E, F

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$$\sigma_z = ax/(1 + bx)$$

$$P^{-1}(x, z_d) = 1 + \frac{v_d - v_g}{ua}\sqrt{\frac{2}{\pi}}\left[\ln\left(\sqrt{2}\,\sigma_z / z_d\right) - 1 + \frac{2b}{a}\left(\sigma_z - \sqrt{\frac{\pi}{2}}\, z_d\right) + \frac{3b^2}{2a^2}\,\frac{\pi}{2}\left(\sigma_z^2 - z_d^2\right)\right]$$

Case 4:
Urban: stability A, B

$$\sigma_z = ax(1 + bx)^{1/2}$$

$$P^{-1}(x, z_d) = 1 + \frac{v_d - v_g}{ua}\sqrt{\frac{2}{\pi}}\left[\ln\left(\sqrt{2}\,\sigma_{z1} / z_d\right) - 1 + \ln\left(1 + k\, z_d / \beta - \sqrt{\frac{2}{\pi}}\, k\, \sigma_{z2} / \beta\right)\right]$$

$$k = \frac{2b}{a}\sqrt{\frac{\pi}{2}},$$

For the last form, , and

$$\sigma_{z1} = \sigma_z\left(1 - .0006\,\sigma_z\right)^2 \qquad \sigma_z \le 300\text{m}$$

$$\sigma_{z1} = 0.6724\,\sigma_z \qquad \sigma_z > 300\text{m}$$

and

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$$\sigma_{z2} = \sigma_{z1} \qquad \sigma_{z1} \le 1000\text{m}$$

$$\sigma_{z2} = \sqrt{1000\,\sigma_{z1}} \qquad \sigma_{z1} > 1000\text{m}$$

The added complexity of this last form arises because a simple analytical solution to Equation (1-57) could not be obtained for the urban class A and B. The integral in $P(x,z_d)$ for $_z = ax(1 + bx)^{1/2}$ listed above matches a numerical solution to within about 2% for $z_d$ = 1 m.

When vertical mixing is limited by $z_i$, the profile correction factor $P(x,z_d)$ involves an integral from 0 to $z_i$, rather than from 0 to infinity. Furthermore, V contains terms that simulate reflection from $z = z_i$ as well as $z = 0$ so that the profile correction factor, $P(x,z_d)$, becomes a function of mixing height, i.e, $P(x,z_d,z_i)$. In the well-mixed limit, $P(x,z_d,z_i)$ has the same form as $P(x,z_d)$ in Equation (1-60) but $_z$ is replaced by a constant times $z_i$:

$$\ln\left(\sqrt{2}\,\sigma_z/z_d\right) \rightarrow \ln\left(z_i/z_d\right)$$

$$\sigma_z - \sqrt{\frac{\pi}{2}}\,z_d \rightarrow \left(\sqrt{\frac{\pi}{8}}\,z_i - \sqrt{\frac{\pi}{2}}\,z_d\right) \qquad \text{1-62}$$

$$\sigma_z^2 - z_d^2 \rightarrow \sqrt{\frac{2}{\pi}}\left(\frac{1}{3}z_i^2 - z_d^2\right) = \left(\sqrt{\frac{2}{\pi}}\,\frac{z_i^2}{3} - z_d^2\right)$$

Therefore a limit is placed on each term involving $_z$ in Equation (1-60) so that each term does not exceed the corresponding term in $z_i$. Similarly, since the leading order term in $P(x,z_d)$ for $_z = ax(1 + bx)^{1/2}$ corresponds to the $\ln\left(\sqrt{2}\,\sigma_z/z_d\right)$ term in Equation (1-62), $_z$ is capped at $z_i/\sqrt{2}$ for this $P(x,z_d)$ as well. Note that these caps to $_z$ in Equation (1-60) are broadly consistent with the condition on the use of the well-mixed limit on V in Equation (1-51) which uses a ratio $_z/z_i = 1.6$. In Equation (1-62), the corresponding ratios are $_z/z_i =$ 1.4, 1.6, and 1.9.

In many applications, the removal of material from the plume may be extremely small, so that $F_Q(x)$ and $P(x,z)$ are virtually unity. When this happens, the vertical term is virtually unchanged ($V_d = V$, see Equation (1-54)). The deposition flux can then be approximated as $v_d$ rather than $v_{dd}$. The plume depletion calculations are optional, so that the added expense of computing $F_Q(x)$ and $P(x,z)$ can be avoided. Not considering the effects of dry depletion results in conservative estimates of both concentration and deposition, since material deposited on the surface is not removed from the plume.

**The Decay Term (D)**

The Decay Term in Equation (1-1) is a simple method of accounting for pollutant removal by physical or chemical processes. It is of the form:

$$D = \exp\left(-\psi \frac{x}{u_s}\right) \quad \text{for } \psi > 0$$

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or

$$= 1 \quad \text{for } \psi = 0$$

where:

$\psi$ = the decay coefficient ($s^{-1}$) (a value of zero means decay is not considered)

$x$ = downwind distance (m)

For example, if $T_{1/2}$ is the pollutant half life in seconds, the user can obtain R from the relationship:

$$\psi = \frac{0.693}{T_{1/2}} \qquad 1\text{-}64$$

The default value for R is zero. That is, decay is not considered in the model calculations unless R is specified. However, a decay half life of 4 hours (R = 0.0000481 $s^{-1}$) is automatically assigned for $SO_2$ when modeled in the urban mode.

**August 31, 2005**
**Lecture 3: Nature of Toxicity (Measuring Toxicity, Expressing Toxicity, Factors Influencing Toxicity)**

## I. What Is Toxicity

A. Two variables are most important in determining the likelihood that exposure to a toxicant will result in an adverse response: dose (amount of exposure) and time (frequency and duration of exposure).

B. Based on these variables, the following definition has been proposed: Toxicity "is the accumulation of injury over short or long periods of times that renders an organism incapable of functioning within the limits of adaptation or other forms of recovery." (Rozman et al. 2001, Dose, time, and other factors influencing toxicity. p. 7 in Handbook of Pesticide Toxicology, vol. 1, R. Krieger (ed.). Academic Press)

1. Note that this definition focuses on the organism, but toxicity adversely affecting many members of a population will eventually result in effects at higher levels of organization.

## II. Measuring Toxicity

A. We have lofty goals of protecting ecosystems, but measuring hazards at this level and scale are not easily done. Rather, we tend to measure hazards at lower levels, and than attempt to extrapolate effects on individuals or populations to higher levels of organization.

1. Thus, to even begin to assess risk of adverse effects in ecosystems, it is necessary to understand how we measure adverse effects or toxicity at lower levels of organization.

a. An example of this "downsizing" of our focus area relative to our goal of protecting ecosystems is illustrated in the following table (which comes from Suter and Barnthouse 1993, p. 25 in Ecological Risk Assessment, G. Suter (ed.), Lewis Publishers).

Table 1. Scenario I--The policy goal (i.e., risk management objective) is no unacceptable loss of fisheries in a southern lake when a herbicide is used for weed control. The hazard in this scenario is adverse effects on fish populations. The table provides examples of assessment endpoints, possible indicators of effects on those assessment endpoints, and possible endpoints for measurements of those indicators.

| Assessment Endpoints | Indicators of Effects | Measurement Endpoints |
|---|---|---|
| Probability of >10% reduction in game fish production | Laboratory toxicity to fish | Fathead minnow LC50; Larval bass concentration/mortality function |
| | Laboratory toxicity to food-chain organisms | Daphnia magna LC50; Selenastrum capricornum (algal species) EC10 |
| | Field toxicity to fish | % mortality of caged bass |
| | Population abundance in treated lakes | Catch per unit effort; Size/age ratios by age classes |

Table 2. Scenario II—The policy goal is no unacceptable reductions in avian populations. The hazard is bird kills following application of an agricultural insecticide.

| Assessment Endpoints | Indicators of Effects | Measurement Endpoints |
|---|---|---|
| **Proportion of raptors killed within the region of use** | Laboratory toxicity to prey | Rat LD50;<br>Japanese quail dietary LC50 |
| | Laboratory toxicity to raptors | Sparrow hawk dietary concentration/response function;<br>Japanese quail dietary LC50 |
| | Avian field toxicity | Number of prey carcasses per hectare;<br>Number of dead or moribund raptors per hectare |
| | | |
| **Increase in the rates of decline of declining bird populations within the region of use** | Avian laboratory toxicity | Japanese quail dietary LC50;<br>Starling dietary LC50 |
| | Avian field toxicity | Number of bird carcasses per hectare by species |
| | Trends in populations of declining birds | Rates of decline in areas of use as proportions of reference areas |

2. For the most part, we will be talking about effects on individuals, although we need some population of these individuals to estimate toxicity.
   a. Indeed, when measuring toxicity, we must use as many individuals as possible to understand the distribution of response within the population.
      1. In other words, we want to know about the heterogeneity of the response within the population.

**B. Endpoints**

1. To measure toxicity, we must observe some specific endpoint. Think of an endpoint as the direct or indirect biochemical, cellular, physiological, or behavioral response following exposure to a toxicant.
2. In the above tables, the most used endpoint would be lethality or mortality, as represented by the measure called the LC50 (lethal concentration to 50% of the test population) or the LD50 (lethal dose to 50% of the test population)
   a. Note the difference between concentration and dose;
      1. The use of concentration refers to an environmental residues in some volume or mass of matrix that an organism is exposed to;
      2. In contrast, expression of exposure as a dose refers to the known mass (or total amount) of xenobiotic to which an organism is exposed;
         a. However, in mammalian toxicology, dose is often normalized to a reference point like body weight; dose relative to body weight is called dosage, which is a convenient expression for comparing exposure across different organisms or different age/sex classes of a single species.

   b. The absorbed dose is the amount of toxicant that is actually absorbed into the body, whether it is through the skin or the lungs or via absorption from the intestine.
   c. In toxicity studies wherein the amount of toxicant is expressed as a concentration, for example, as so many ppb in water, the dose can be estimated by examining the toxicant residues in the whole body at different times after exposure.
      1. If the toxicant was in the diet or in drinking water, dose could be estimated by monitoring the consumption of food or water (i.e., kg or L consumed during the observation period).

3. Other lower level or individual endpoints could be biochemical, genetic, cellular, physiological, morphological, functional, or behavioral. Indeed, any mechanism of toxic action can be the basis for using an endpoint as a qualitative or quantitative measure of toxicity.
4. Elucidating endpoints is part of the Hazard Identification process. However, not all endpoints are necessarily injurious, and some may be indicative of an interaction with a toxicant but without physiological (or biological) relevance.
5. Short descriptions of examples of endpoints applicable to individuals follow (the following list is based on a general reading of published environmental toxicology studies and is only an overview, not an exhaustive treatment):
   a. <u>Biochemical and Genetic Endpoints</u>
      1. Enzyme-toxicant interactions
         a. Induction of enzyme activity
         b. Inhibition of enzyme activity
      2. Receptor-toxicant interactions
         a. Inhibition of ability of receptor to bind with its normal biochemical substrate
         b. Increase in receptor activity by mimicking the normal biochemical substrate
      3. Unusually high or low blood titers of hormones
         a. Males exhibiting unusually high levels of female hormones like vitellogenin (e.g., in fish)
      4. DNA interactions
         a. Binding with DNA, causing mutations
      5. Chromosomal effects
         a. Clastogenicity: chromosome breakage
   b. <u>Cellular and Physiological</u>
      1. Binding to membranes, interrupting nerve signals, nutrient or ionic transport
      2. Disruption of membrane structure
      3. Increases in cell death; either necrosis (unprogrammed cell death) or apoptosis (programmed cell death)
      4. Increased levels of immunoglobulins (antibodies)
      5. Reduction of chlorophyll content (applicable to plants) leading to reduced productivity
      6. Altered respiratory metabolism energetics leading to stress

7. Reduced ability to tolerate cold temperatures
8. Reduced ability to tolerate salt water (anadromous species)

c. Morphological
1. Notable signs of irritation on the body surface or in the eyes
2. Excretory discharges
3. Developmental abnormalities (teratogenicity)
a. Skeletal abnormalities
b. Abnormalities in genitalia
c. Transgender characteristics

d. Functional and Behavioral
1. Inability to avoid predation
2. Inability to secure adequate food
3. Lack of appropriate sexual behavior leading to reduced mating success
4. Impairment of cognitive ability
5. Reduction in fertility

C. Testing Organisms
1. In mammalian toxicology studies, especially those used in regulatory toxicology, wherein data are being produced to pass review of a regulatory agency (such as approval of a drug by the FDA [Food & Drug Administration] or pesticide by the EPA), rodents (rats and mice) are the subjects of choice.
a. The EPA also accepts studies on dogs.
b. A key aspect of testing is to control for heterogeneity between individuals, so all breeding has to be carefully monitored and standardized.
2. For ecotoxicological testing, the common test species are representatives of aquatic and terrestrial organisms, encompassing invertebrates, vertebrates, and plants. (A brief description of the common ecological toxicity test organisms and their natural history is given in Landis and Yu, 1999. *Introduction to Environmental Toxicology*, Lewis Publishers, pp. 82-89.)
a. The most common aquatic invertebrate tested are microcrustaceans (Phylum Arthropoda).
1. The organism most frequently used is *Daphnia magna* or *Daphnia pulex*. *Daphnia* spp. are commonly called waterfleas.
a. *Daphnia* are used to test toxicity in the water column.
2. Periodically, in ecorisk assessments, EPA will rely on data from tests with crayfish, or aquatic insects (stoneflies, mayflies, midges).
3. Other common aquatic invertebrates include amphipods (a.k.a. scuds) (*Gammarus lacustris*, *Hyalella azteca*, and others).
a. These species are used to test toxicity in sediments.
4. One rationale for using the aquatic invertebrates commonly tested is their "role" as prey for vertebrates like fish.
a. Also, the life cycle of the aquatic invertebrates is short, making lab studies feasible.
b. The most common terrestrial invertebrates used in ecotox assessments would be insects, especially the honeybee (a beneficial pollinator).

1. Several years ago, the Monarch butterfly came into prominence as a key species with regard to the use of transgenic corn plants containing a gene encoding the synthesis of the Bt (*Bacillus thuringiensis*) toxin.
    a. For an interesting risk assessment study using Monarch butterfly, see Sears et al. 2001. Impact of Bt corn pollen on monarch butterfly populations: a risk assessment. Proc. National Academy of Sciences 98:11937-11942.

c. The most common vertebrates used in ecological toxicity testing for aquatic risk assessments are fish, and any one of several species is commonly used. These include rainbow trout (*Oncorhynchus mykiss*), fathead minnow (*Pimephales promelas*), and various species of sunfish [bluegill, *Lepomis macrochirus*; green sunfish, *Lepomis cyanellus*].
    1. Other species noted in ecorisk assessments for pesticides include bull trout (*Salvelinus confluentus*) and brook trout (*Salvelinus fontinalis*), or any of several species of salmon (Coho, *Oncorhynchus kisutch*, is most commonly used as well as Atlantic salmon, *Salmo salar*).

d. Birds are most commonly used for terrestrial toxicity testing. The rodent tests used in mammalian toxicology studies serve as a surrogate for mammalian wildlife in ecological risk assessments.
    1. The most common bird species include mallard duck (*Anas platyrhynchos*), northern bobwhite quail (*Colinus virginianus*), and ring-necked pheasant (*Phasianus colchicus*).

e. Both aquatic and terrestrial plants are used for ecotoxicity testing. Among aquatic plants, algae and submergent vascular plants are used. Among terrestrial plants, EPA requires root elongation and early growth studies with nontarget crop species. Tests with plants are especially important for herbicide registrations.

f. Pesticides may be tested for effects on soil microbial function (for example, denitrification).

D. The U.S. EPA has published guidelines documents for conducting several different types of water toxicity tests that would satisfy the requirements for whole effluent toxicity testing (WET) required under the Clean Water Acts National Pollution Discharge Elimination System permitting process.
    1. U.S. EPA 2002. Methods for measuring the acute toxicity of effluents and receiving waters to freshwater and marine organisms. Fifth edition, EPA-821-R-02-012 URL http://www.epa.gov/waterscience/WET/disk2/
        a. "This manual describes test for effluents and receiving waters and includes guidelines on laboratory safety, quality assurance, facilities and equipment, dilution water, effluent sampling methods and holding times, test specied selection, data analysis, report preparation, organism culturing and handling, and mobile toxicity test laboratory design. The acute toxicity tests generally involve exposure of any of 20 test organisms to each of five effluent concentrations and a control water. The test duration ranges from 24-96 hours. This manual contains specified test conditions for 10 commonly used freshwater and marine organisms: *Ceriodaphnia dubia*, *Daphia magna*, *Daphnia pulex*, brine shrimp (*Artemia salina*), fathead minnows (*Pimephales promelas*), rainbow trout (*Oncorhynchus mykiss*), brook trout (*Salvelinas fontinalis*), mysids (*Mysidopsis bahia*, and

*Holmesimysis costata*), Bannerfish shiners (*Notropis leedsi*), sheepshead minnows (*Cyprinodon variegatus*), and sliversides (*Menida menidia, M. Beryllina, and M. Peninsulae*)."

2. EPA, U. S. 2002. Short-term methods for estimating the chronic toxicity of effluents and receiving waters to freshwater organisms. Fourth edition, EPA-821-R-02-013, URL http://www.epa.gov/waterscience/WET/disk3/
   a. "This manual describes four- to seven-day methods for estimating the chronic toxicity of effluents and receiving waters to three species: the fathead minnow, *Pimephales promelas*, the cladoceran, *Ceriodaphnia dubia*; and the alga, *Selenastrum capriconutum*. Guidelines are included on laboratory safety, quality assurance, facilities and equipment, dilution water, effluent sampling methods and holding times and temperatures, data analysis, report preparation, and organism culturing and handling."
3. EPA, U. S. 2000. Methods for measuring the toxicity and bioaccumulation of sediment-associated contaminants with freshwater invertebrates. Second edition 600/R-99/064, URL http://www.epa.gov/ost/cs/freshfact.html
   a. "Procedures are described for testing freshwater organisms in the laboratory to evaluate the potential toxicity or bioaccumulation of chemicals in whole sediments. Sediments may be collected from the field or spiked with compounds in the laboratory. Toxicity methods are outlined for two organisms, the amphipod *Hyalella azteca* and the midge *Chironomus tentans*. Toxicity tests with amphipods or midges are conducted for 10 d in 300-mL chambers containing 100 mL of sediment and 175 mL of overlying water. Overlying water is renewed daily and test organisms are fed during the toxicity tests. The endpoints in the 10-d toxicity test with *H. azteca* and *C. tentans* are survival and growth. The second edition includes new methods for evaluating sublethal effects of sediment-associated contaminants utilizing long-term sediment exposures with the amphipod *Hyalella azteca*, and the midge *Chironomus tentans*. The long-term sediment exposures with *H. azteca* are started with 7 to 8 day old organisms. Effect endpoints measured for *H. azteca* include survival (measured on days 28, 35, and 42), growth (measured on days 28 and 42), and reproduction (measured as number of young/female from day 28 to 42). The long-term sediment exposures with *C. tentans* start with newly hatched larvae (< 24 hours old) with effect endpoints including emergence, reproduction, and hatching of the next ($F_1$) generation (which requires about 60 days)."

## III. Quantitative Expression of Toxicity

A. Toxicity is measured by determining the relationship between dose or concentration of a substance and the response of the test organism under specified test conditions.
   1. The response can range from death to subtle changes in enzyme activity or everything in between.
   2. The most common parameter to express toxicity is the dose or concentration causing 50% of tested organisms to respond.

a. The median population response is expressed as the LD50 (if lethality is the endpoint) or the ED50 (if other types of responses are the endpoints), or the LC50 and/or EC50 if the concentration, but not the dose is known.

3. In addition to knowing the LD50 or ED50, and more importantly for purposes of determining "safety", we also want to know the dose or concentration causing no response, known as the NOEL or NOAEL (No Observable Adverse Effect Level).
   a. In aquatic toxicity testing, the concentration of test substance would be the independent variable, so the no-effect concentration would be the NOAEC.

B. How an LD50 or LC50 (or ED50/EC50) are determined
   1. Hypothetical response of a population to a stimulus (adverse or favorable) can be described as a normal distribution ("bell-shaped" curve) if we graph the numbers responding at each dose or concentration tested (Figure 1).
      a. To generate the required data, organisms are exposed via the diet, skin, or environment (i.e., air, water [aquatic organisms], soil [worms, bacteria] to a series of increasingly higher doses, starting with zero concentration as a control. The organisms are randomly assigned to experimental groups, and each group receives one dose.

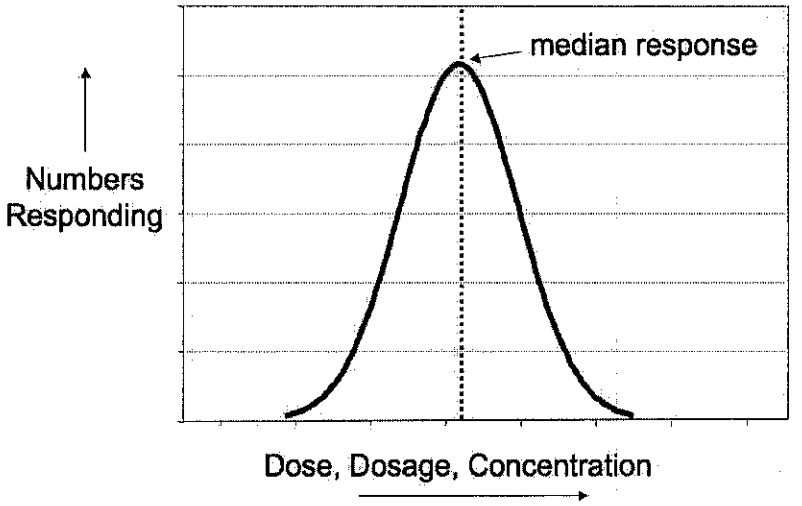



Figure 1. Normal distribution of responses to increasing dose of toxicant.

      b. The response, i.e., the endpoint, must be strictly specified; the magnitude of this endpoint is then recorded at each dose. Many times we are interested in outright death, but other effects, such as decreased weight or enzyme activity, are equally valid just as long as they are specified and measurements can be validated.
         1. Concerning endpoints, we distinguish between acute toxicity, which is usually an immediate response to the short term or single dosing of an organism, and chronic toxicity, which is a systemic effect developing over a period of time beyond the actual dosing.

a. In mammalian acute toxicity studies, a rodent is exposed usually by intubation (direct application to the stomach through a tube) to high doses; mortality is measured after 24 hours and further physiological effects monitored for the next 14 days. After 14 days the animal is sacrificed for histological observations. (Ecobichon, D. J., 2001, p. 287 in Handbook of Pesticide Toxicology, R. Krieger (ed.), Academic Press).
b. In ecological toxicity studies (i.e., testing for environmental effects), acute toxicity observations depend on the organism
   1. For fish, exposure occurs via water for 96 hours
   2. For invertebrates, exposure occurs via water for 48 hours;
   3. For birds, acute exposure can occur similarly to rats, via force-feeding directly into the stomach, and subsequent monitoring of effects 24 hours later and beyond.
c. We normally think of chronic toxicity as resulting from repeated non-acutely lethal (i.e., from sublethal) dosing.
   1. For example, in mammalian toxicity studies, chronic toxicity would be measured as developmental/reproductive effects or as carcinogenicity.
   2. For ecological toxicity studies, chronic toxicity would be measured as an adverse effect during the reproductive cycle of an invertebrate or vertebrate.
      a. The exposure would last throughout the reproductive phase.

2. The cumulative proportion responding (which can be expressed as a percent) to increasing doses can be depicted as a sigmoidal function; note that the tangent to the function would be the slope (Figure 3).
   a. The slope of the response would indicate the variability in response within the test population.
   b. The LD50, ED50, LC50, EC50 represent the dose or concentration corresponding to the median (or 50%) population response.

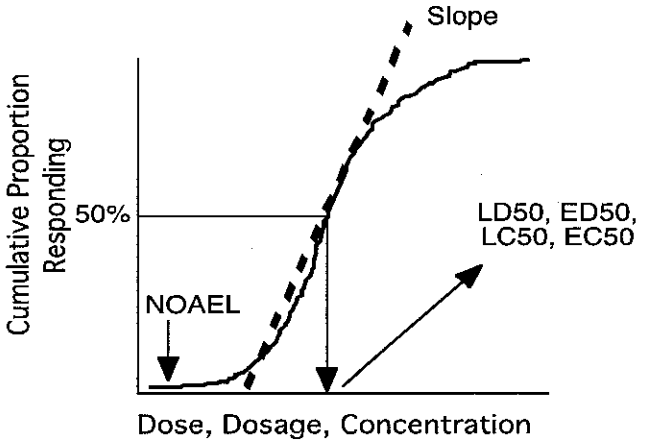
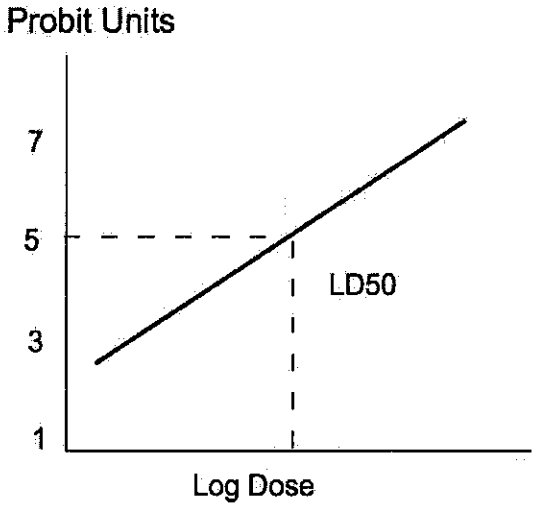


Figure 2. Sigmoidal distribution of proportion of population responding relative to dose.

3. Bliss (1935) linearized the sigmoidal function using probability units (i.e., probits, which turn out to be unit standard deviations above and below 50% mortality, or probit 5.0) plotted against the logarithmic dose [Bliss, C. I. 1935. The calculation of the dosage-mortality curve. Annals of Applied Biology 22:134-167]. (Fig. 3).
   a. The LD50/LC50 is the region having the narrowest confidence intervals, and thus the most reliable indication of response at a particular dose.
   b. See addendum starting on page 16 for an example of the computer program input and output used to estimate the dose-response function and the LC50 (or any other level of mortality).




| % Mortality | Probits |
|---|---|
| 10% | 3.72 |
| 20 | 4.16 |
| 30 | 4.48 |
| 40 | 4.75 |
| **50** | **5.00** |
| 60 | 5.25 |
| 70 | 5.52 |
| 80 | 5.84 |
| 90 | 6.28 |

Figure 3. Transformation of the sigmoidal function to a probit function

C. Determination of the NOAEL/NOAEC
   1. Examination of Figure 2 showing the sigmoidal dose-response curve shows a concentration at which the effect being measured is essentially zero; in other words, the endpoint chosen was not found to occur among the test population.
   2. This coordinate corresponding to the dose or concentration with no measurable effect is called the no observable adverse effect level (NOAEL) if the dose is known, or the no observable adverse effect concentration (NOAEC) if the concentration but not the dose is known.
      a. Although one can model this coordinate from the empirical portion of the dose-response function, more often than not (at least in the data that US EPA uses to conduct pesticide risk assessments), the NOAEL or NOAEC is an empirical observation derived from the actual toxicity test.
   3. Note that the NOAEL or NOAEC is not usually used as an estimation of toxicity magnitude when dealing with acute exposure and lethality.
      a. For mammalian toxicology studies, the NOAEL is usually derived from either chronic toxicity testing or shorter term, multiple exposure testing known as subchronic tests.

1. For rodent toxicity tests, subchronic tests last from about one month to three months (90 days).
2. For ecological toxicity tests, the NOAEL and NOAEC is reserved as a parameter associated with life cycle (chronic) studies, which usually focus on reproductive effects.

4. The NOAEL/NOAEC are usually thought of as a threshold for toxicity, but bear in mind the threshold is only applicable to the specific endpoint being measured.
   a. It is common in mammalian toxicity testing to seek the most sensitive toxicological endpoint's NOAEL.
      1. In other words, the most sensitive endpoint would be the toxicological effect occurring at the lowest dose.
         a. The NOAEL would be determined in the experiment by comparing the response of the dosed (treated) animals with the non-dosed (control or untreated) animals, and then applying a statistical test to compare the groups.
      2. Presumably, when the threshold for the most sensitive endpoint is used, then there is protection against all effects occurring at all equal or lower doses.
   b. In ecological risk assessment, the NOAEC refers to a concentration below which no adverse effect is expected in the test organisms.
      1. Because there is a tendency to find and use the most sensitive test organisms, then there is a presumption that the NOAEC can be predictive of effects on many organisms.
         a. Unfortunately, it is impossible to know if one actually has in hand the most sensitive organism.

**D. Hormesis**

1. Recently, a lot of attention has been given to hormesis, a phenomenon described in the modern literature nearly 50 years ago.
   a. Hormesis is a positive or favorable physiological response to low doses of a toxicant.
   b. At low doses, the toxicant produces a stimulatory response (for example, greater growth rates) but an inhibitory response at higher doses.
2. Recent statistical examination of dose-response curves from many toxicity tests shows that a beneficial (favorable physiological effect) is common for many compounds. Following are some of the articles recently published by E. Calabrese et al.
   a. Calabrese, E. J. and L. A. Baldwin. 2002. Hormesis and high-risk groups. Regulatory Toxicology and Pharmacology 35:414-428.
   b. Calabrese, E. J. and L. A. Baldwin. 2003. The hormetic dose-response model is more common than the threshold model in toxicology. Toxicological Sciences 71:246-250.
   c. Calabrese, E. J. and L. A. Baldwin. 2003. Toxicology rethinks its central belief. Nature 421(13 February):691-692.

d. Calabrese, E. J. 2005. Pardigm lost, paradigm found: The re-emergence of hormesis as a fundamental dose response model in the toxicological sciences. Environmental Pollution 138 379-412.

3. An example of the hormetic response can be seen in the following figure (Figure 4) taken from Calabrese and Baldwin (2002) and modified. The data was from an experiment by Ukeles 1962 (Ukeles, R. 1962. Growth of pure cultures of marine phytoplankton in the presence of toxicants. *Appl. Microbiol.* **10,** 532–537).

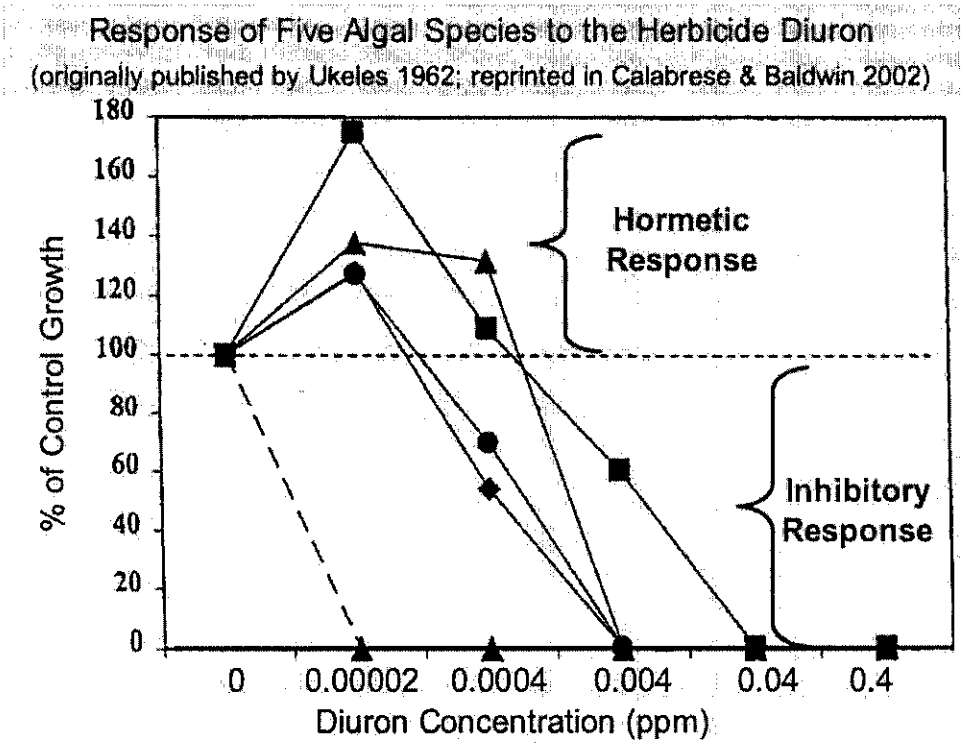



Figure 4. Response of five algal species to the herbicide diuron. Diuron, a phenylurea herbicide, is heavily used in roadside spraying to control weeds that encroach near the paved roadway. Calabrese et al. have pulled together an extensive database showing that all kinds of organisms seem to exhibit a hormetic response. However, note in the graph above, that there was one species of algal that did not exhibit this effect (represented by the dashed line). Indeed, by comparing the position of the dose-response curve to the other four algal species, you can see that its susceptibility to diuron is much greater.

## IV. Factors Influencing Toxic Response

A. Two of the most important factors influencing the toxic response are dose or dosage (concentration when dealing with aquatic organisms) and time of exposure. When time is controlled or held constant in a test, then dose is the prime factor determining the appearance of injury. However, there are other

factors that can influence the expression of toxicity in addition to the dose itself. Casarett and Doull (1975, "Toxicology: the Basic Science of Poisons", Macmillan Publishing Co.; p. 134) have summarized the "toxicity-influencing factors" from the perspective of mammalian toxicology.

1. Factors related to the toxic agent
   a. Chemical composition (pH, choice of cations or anions if a salt, etc.)
   b. Physical characteristics (particle size, method of formulation, etc.)
   c. Presence of impurities or contaminants
   d. Stability and storage characteristics of the toxic agent
   e. Solubility of the toxic agent in biologic fluids
   f. Choice of the vehicle for delivering (dosing) the test organism
   g. Presence of excipients (materials used to dissolve, stabilize, and or deliver the test agent (including adjuvants, emulsifiers, surfactants, binding agents, coating agents, coloring agents, flavoring agents, preservatives, antioxidants)
2. Factors related to the exposure situation
   a. Dose, concentration, and volume of administration of the toxic agent
   b. Route, rate, and site of administration
   c. Duration and frequency of exposure
   d. Time of administration (time of day, season of the year, etc.)
3. Inherent factors related to the exposed organisms (or test subjects)
   a. Species and strain differences (i.e., taxonomic classification)
   b. Genetic status (littermate, siblings, multigenerational effects, etc.)
   c. Immunologic status
   d. Nutritional status (dietary factors, state of hydration, etc.)
   e. Hormonal status (pregnancy, etc.)
   f. Age, sex, body weight, and maturity
   g. Central nervous system status (activity, crowding, handling, presence of other species, etc.)
   h. Presence of disease or specific organ pathology
4. Environmental factors related to the subject
   a. Temperature and humidity
   b. Barometric pressure (hyper- and hypobaric effects)
   c. Ambient atmospheric composition
   d. Light and other forms of radiation
   e. Housing and caging effects
   f. Noise and other geographic influences
   g. Social factors
   h. Chemical factors
5. Note that many of the factors listed in (3) and (4) could be characterized generally as stress-producing factors.

B. With a few exceptions, most of the factors listed above have not been thoroughly studied from a quantitative perspective. For purposes of ecological toxicity testing, some of the factors have been given more attention, especially those related to age and environment. Here are some examples of quantitative data

generated regarding some of these endogenous and exogenous influences on toxicity.

1. Exposure situation: it is well known that route of administration or exposure of terrestrial organisms can influence the degree of toxicity, assuming an equal dose rate.
   a. Dermal exposures of pesticide, for example, are estimated to be four times less hazardous than oral exposures (range of ratio of toxicity of oral to dermal toxicity ranged from 0.2 to 21 with an average of 4.2; there were several compounds in which dermal exposure was more hazardous. Information was cited on p. 54 in Rozman et al. 2001). Much of what we know about differences in toxicity due to routes of exposure comes from rodent studies.
   b. Figure 5 shows a comparison of insecticide toxicity when the pesticide is administered to rodents via an acute oral dose or a patch on shaved skin. The exposure duration is 24 hours.

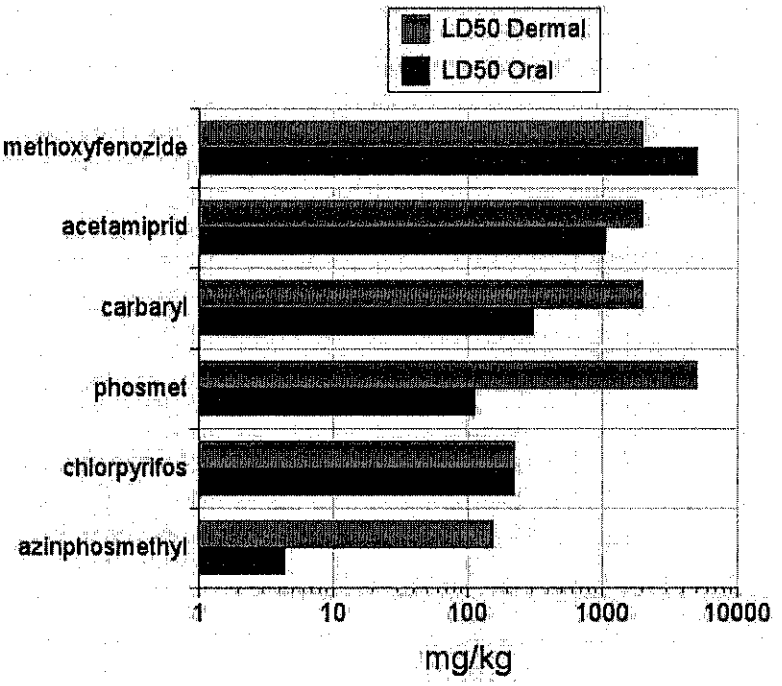



Figure 5. Comparison of toxicity of 6 insecticides by oral or dermal exposure of rodents.

   c. Dermal exposure also seems to be the most toxic route of exposure for birds. For example, Driver et al. 1991 exposed northern bobwhite quail in a wind tunnel to methyl parathion (an organophosphorus insecticide) and concluded that the routes of uptake in order of their contributionto toxicologica response from 8-48 hours after spraying were dermal > preening ≥ oral > inhalation (Driver, C. J., M. W. Ligotke, P. Van Voris, B. D. McVeety, B. J. Greenspan and D. B. Drown. 1991. Routes of uptake and their relative contribution to the toxicologic responses of northern bobwhite (colinus virginianus) to an organophosphate pesticide. Environ. Toxicol. Chem. 10 21-33.)

1. Note that the acute oral LD50 for methyl parathion to northern bobwhite quail was estimated to be 7.56 mg/kg, and the acute dermal toxicity was 9.172 mg/kg (data from the Methyl Parathion Re-registration Eligibility Decision Document released by EPA, URL http://www.epa.gov/oppsrrd1/op/methyl_parathion.htm).
    a. The bobwhites were able to tolerate 6.3 ppm methyl parathion in the diet over a longer period of time. This NOAEC was based on number of eggs laid and bodyweight.

2. Factors inherent to the organism
    a. **Species differences in response:** note in the following table that the insecticides tested putatively have the same mechanism of causing toxicity, i.e., excessive inhibition of acetylcholinesterase, the neuromodulatory enzyme present at the nerve terminal synapses in the central nervous system and at the neuromuscular junctions. Yet, azinphos-methyl is much more toxic to fish than is diazinon, but diazinon is much more toxic to birds (a notorious "bird killer"). Thus, fish and birds react differently to two different compounds with the same mode of action. (Data in the table are taken from EPA Registration Eligibility Documents; index to published documents at URL http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg#M.)

| | Avian LD50 (mg/kg) | Fish LC50 (µg/L) | Ratio Avian/Fish |
|---|---|---|---|
| **azinphos-methyl** | 75 | 20 | 3.75 |
| **diazinon** | 4.3 | 16000 | 0.0003 |

    b. **Age:** a study with starlings and redwing blackbirds exposed at different developmental stages to the organophosphorus insecticides terbufos and diazinon showed marked age susceptibility differences. Data in the table below were taken from Wolfe, M. F. and R. J. Kendall. 1998. Age-dependent toxicity of diazinon and terbufos in European starlings (*Sturnus vulgaris* ) and red-winged blackbird (*Agelaius phoeniceus*). Environmental Toxicology and Chemistry 17(7):1300-1312.

| Age | Terbufos Starling LD50 (mg/kg) | Diazinon Starling LD50 (mg/kg) |
|---|---|---|
| 2 | 2.3 | 12.7 |
| 5 | 5 | 35.6 |
| 9 | 20.3 | 93.2 |
| 15 | 29.9 | 102 |
| 19 | 60.8 | 145 |
| Adult | 204 | 602 |

**c. Environmental Factors**

1. Some of the common environmental factors affecting toxicity that have been quantitatively studied in environmental toxicology include temperature and pH of water. Some studies have also focused on

stress related factors that could be considered related to environmental conditions. For example, starvation would be a nutritional factor imposed by environmental conditions during certain times of the year. Another possible stress factor is infection by parasites.

a. The table below shows the relationship between temperature, infection status, and toxicant exposure in clams. (Data are from Heinonen et al. 2001. Temperature- and parasite-induced changes in toxicity and lethal body burdens of pentachlorophenol in the freshwater clam *Pisidium amnicum*. Environ. Toxicol. Chem. 20(12):2778-2784.)

| Temperature | Exposure (PCP, µg/L) | Infection Status | Mean Survival Time (h) |
|---|---|---|---|
| 5 | 100 | Infected | 611 |
| 5 | | Uninfected | 574 |
| 5 | 300 | Infected | 525 |
| 5 | | Uninfected | 506 |
| 19 | 100 | Infected | 136 |
| 19 | | Uninfected | 60 |
| 19 | 300 | Infected | 63 |
| 19 | | Uninfected | 33 |

d. When the empirical database on factors affecting toxicity is adequate, than deterministically predictive model can be built using regression analysis.
   1. For example, the database for pesticide toxicity to aquatic organisms (several invertebrate species and fish species) has been analyzed for temperature effects on LC50 (Mayer and Ellersieck 1986; summarized in Suter et al.1993).

      Mayer, F. L., Jr. and M. R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 410 chemicals and 66 species of freshwater animals. U. S. Department of the Interior, Fish and Wildlife Service Resource Pulibcation 160, Washington, DC

      Suter, G. W., L. W. Barnthouse, S. M. Bartell, T. Mill, D. Mackay and S. Paterson. 1993. Ecological risk assessment. Lewis Publishers, Ann Arbor, MI

      a. The relationship for the temperature effect of most pesticides was modeled using linear regression; the output was the following linear function:
         1. Log LC50t±10 = log LC50t ± 0.4956, where t = temperature, ± 10°C
            (a) However, for the specific class of organophosphorus insecticides, the following regression function was more predictive:
            (b) LC50t±10 = log LC50t ± 0.7113

   b. One important objective of environmental toxicology is prediction, and the types of empirical exercises described above can help us understand specific toxicological responses under certain circumstances using "back-of-the-envelope" calculations and armchair reasoning.

Addendum: Analysis of Toxicity Data for Determination of Dose-Response Function and Estimation of LC50 using SAS (Statistical Analysis System). Five neonate codling moth larvae were placed on replicates of insecticide-treated leaf disks. After three hours, number of dead larvae were recorded. Four insecticide concentrations and an untreated control were tested.

```
Data Guthion1;   (Data input step)
      Input Dose N Dead;
      Observed=dead/N;
datalines;
0.0000   43    02
0.0099   42    13
0.0198   50    35
0.0296   36    28
0.0395   48    43
;

Proc Probit LOG10 OPTC INVERSECL;  (Programming procedure step)
   Model Dead/N=Dose;
run;
```

(Data Output Step)

Probit Procedure

```
Data Set          =WORK.GUTHION1
Dependent Variable=DEAD
Dependent Variable=N
Number of Observations=  5
Number of Events     =    121   Number of Trials =     219
Number of Events In Control Group =      2
Number of Trials In Control Group =     43

Log Likelihood for NORMAL -100.1627644
```

Probit Procedure

| Variable | DF | Estimate | Std Err | ChiSquare | Pr>Chi | Label/Value |
|---|---|---|---|---|---|---|
| INTERCPT | 1 | 5.33581127 | 0.870144 | 37.60266 | 0.0001 | Intercept |
| Log10(DOS) | 1 | 2.92578526 | 0.5153 | 32.23771 | 0.0001 | |
| _C_ | 1 | 0.04566056 | 0.031473 | | | Lower threshold |

Probit Model in Terms of Tolerance Distribution

| MU | SIGMA |
|---|---|
| -1.82372 | 0.341789 |

Probit Procedure
Probit Analysis on Log10(DOSE)

| Probability | Log10(DOSE) | 95 Percent Fiducial Limits | |
|---|---|---|---|
| | | Lower | Upper |
| 0.01 | -2.61884 | -3.12633 | -2.36712 |
| 0.02 | -2.52567 | -2.98501 | -2.29688 |
| 0.03 | -2.46655 | -2.89546 | -2.25221 |
| 0.04 | -2.42208 | -2.82817 | -2.21853 |
| 0.05 | -2.38591 | -2.77349 | -2.19108 |
| 0.06 | -2.35512 | -2.72699 | -2.16768 |
| 0.07 | -2.32813 | -2.68625 | -2.14712 |
| 0.08 | -2.30396 | -2.64981 | -2.12867 |
| 0.09 | -2.28197 | -2.61670 | -2.11187 |
| 0.10 | -2.26174 | -2.58625 | -2.09638 |
| 0.15 | -2.17796 | -2.46053 | -2.03188 |
| 0.20 | -2.11138 | -2.36113 | -1.98008 |
| 0.25 | -2.05425 | -2.27640 | -1.93512 |
| 0.30 | -2.00295 | -2.20089 | -1.89415 |
| 0.35 | -1.95542 | -2.13161 | -1.85549 |
| 0.40 | -1.91031 | -2.06671 | -1.81798 |
| 0.45 | -1.86667 | -2.00498 | -1.78061 |
| 0.50 | -1.82372 | -1.94563 | -1.74245 |
| 0.55 | -1.78077 | -1.88814 | -1.70242 |
| 0.60 | -1.73713 | -1.83221 | -1.65926 |
| 0.65 | -1.69202 | -1.77764 | -1.61140 |
| 0.70 | -1.64449 | -1.72414 | -1.55697 |
| 0.75 | -1.59319 | -1.67091 | -1.49372 |
| 0.80 | -1.53606 | -1.61626 | -1.41867 |
| 0.85 | -1.46948 | -1.55699 | -1.32675 |
| 0.90 | -1.38570 | -1.48669 | -1.20682 |
| 0.91 | -1.36546 | -1.47019 | -1.17738 |
| 0.92 | -1.34348 | -1.45242 | -1.14524 |
| 0.93 | -1.31931 | -1.43303 | -1.10974 |
| 0.94 | -1.29232 | -1.41156 | -1.06992 |
| 0.95 | -1.26153 | -1.38725 | -1.02432 |
| 0.96 | -1.22535 | -1.35891 | -0.97054 |
| 0.97 | -1.18089 | -1.32432 | -0.90415 |
| 0.98 | -1.12177 | -1.27868 | -0.81557 |
| 0.99 | -1.02860 | -1.20732 | -0.67538 |

Probit Procedure
Probit Analysis on DOSE

| Probability | DOSE | 95 Percent Fiducial Limits | |
|---|---|---|---|
| | | Lower | Upper |
| 0.01 | 0.00241 | 0.00075 | 0.00429 |
| 0.02 | 0.00298 | 0.00104 | 0.00505 |
| 0.03 | 0.00342 | 0.00127 | 0.00559 |
| 0.04 | 0.00378 | 0.00149 | 0.00605 |
| 0.05 | 0.00411 | 0.00168 | 0.00644 |
| 0.06 | 0.00441 | 0.00188 | 0.00680 |
| 0.07 | 0.00470 | 0.00206 | 0.00713 |
| 0.08 | 0.00497 | 0.00224 | 0.00744 |
| 0.09 | 0.00522 | 0.00242 | 0.00773 |
| 0.10 | 0.00547 | 0.00259 | 0.00801 |
| 0.15 | 0.00664 | 0.00346 | 0.00929 |
| 0.20 | 0.00774 | 0.00435 | 0.01047 |
| 0.25 | 0.00883 | 0.00529 | 0.01161 |
| 0.30 | 0.00993 | 0.00630 | 0.01276 |
| 0.35 | 0.01108 | 0.00739 | 0.01395 |
| 0.40 | 0.01229 | 0.00858 | 0.01521 |
| 0.45 | 0.01359 | 0.00989 | 0.01657 |
| **0.50** | **0.01501** | **0.01133** | **0.01809** |
| 0.55 | 0.01657 | 0.01294 | 0.01984 |
| 0.60 | 0.01832 | 0.01472 | 0.02192 |
| 0.65 | 0.02032 | 0.01669 | 0.02447 |
| 0.70 | 0.02267 | 0.01887 | 0.02774 |
| 0.75 | 0.02552 | 0.02133 | 0.03208 |
| 0.80 | 0.02910 | 0.02420 | 0.03814 |
| 0.85 | 0.03393 | 0.02773 | 0.04712 |
| 0.90 | 0.04114 | 0.03261 | 0.06211 |
| 0.91 | 0.04311 | 0.03387 | 0.06647 |
| 0.92 | 0.04534 | 0.03528 | 0.07157 |
| 0.93 | 0.04794 | 0.03689 | 0.07767 |
| 0.94 | 0.05101 | 0.03877 | 0.08513 |
| 0.95 | 0.05476 | 0.04100 | 0.09455 |
| 0.96 | 0.05952 | 0.04376 | 0.10702 |
| 0.97 | 0.06593 | 0.04739 | 0.12469 |
| 0.98 | 0.07555 | 0.05264 | 0.15291 |
| 0.99 | 0.09363 | 0.06204 | 0.21117 |