THE USE AND CONTROL OF SUBSTANCES IN IRISH INDUSTRY WHICH ARE CLASSIFIED AS 'SKIN SENSITISERS'

Dissertation presented in Part Fulfilment for the Degree of Master of Science in Environmental Protection to Sligo Institute of Technology

By

Sonya Morrissey BSc(hons)

Supervised by Mr. John Bartlett

Submitted to the National Council for Eduacation Awards, 1999



ACKNOWLEDGEMENTS

This dissertation would not have been possible without the help of so many people

I wish to thank, John Bartlett my supervisor, for his guidance, and encouragement.

Thanks to all the personnel of the various industries involved in the survey for returning the questionnaires.

Special thanks to all my family, my mother and father who encouraged me all the way, to my brothers and sisters, and especially to my nieces Jennifer and Nicole and my nephews Darragh and Cian who kept me entertained.

Thanks to my friends, Rhona, Anne, and Jo for everything.

To my colleagues at work, Deidre, Fiona, Niamh and Mike. Thank You.



ABSTRACT

Information relating to the extent of the use of substances in Irish Industry which are classified as 'skin sensitisers' is difficult to obtain, as details regarding the volume of these substances imported into Ireland on an annual basis is not available. Occupational skin diseases are the most frequently encountered occupational disease. Despite the fact that occupational skin disease often parallels the level of hygiene practiced by employers, occupational skin disease is largely preventable.

For technical reasons there are still no skin exposure limits to guide employers, nor techniques to measure skin exposure levels. Currently the use of substances which have the potential to cause skin sensitisation are widely used in Irish industry. There is a requirement to develop a structured approach to the management of operations where there is the likelihood of worker exposure to an agent, which has the potential to cause skin disease. Risk assessments are necessary procedures for the evaluation of working conditions and the potential for damage to the health of the employees from their specific tasks. When proper skin management procedures are in place the hazards of a substance can be identified and adequate control measures be implemented before a substance enters the work area.



TABLE OF CONTENTS

	PAGE
Title Page	
Acknowledgement	i
Abstract	ii
List of Contents	iii
List of Figures	v
List of Table	vi
LIST OF CONTENTS	
SECTION 1	
INTRODUCTION	
1.0 Background	1
1.1 Aims and Objectives	1
1.2 Methodology	2
SECTION 2	
LITERATURE REVIEW	
2.1 The Skin as a Barrier	
2.1(a) The structure and composition of the human skin	3
2.1(b) The functions of the skin	8
2.2 Mechanisms of Skin Absorption	
2.2(a) Dermal absorption	10
2.2(b) Dermal penetration	10
2.2(c) The distribution and elimination of percutaneously absorbed chemicals	10

A REFERENCE OF A REPORT OF A R

2.3 Dermatitis

iv	
2.9(b) Job-related factors	47
2.9(a) Predisposing factors for occupational skin disease	47
2.9 Predisposing Factors For Skin Diseases	
2.8 Variables in Occupational Skin Exposure	45
2.7(b) Criteria for the classification of skin sensitisers	37
2.7(a) European legislation in the classification of skin sensitisers	34
2.7 Classification of Skin Sensitisers	
2.6(d) Principle occupational sensitisers	29
2.6(c) Sensitisers	28
2.6(b) Direct causes of occupational dermatitis	27
2.6(a) Occupational dermatoses	26
2.6 Occupational Skin Disease	
2.5(d) Activation of supressor pathways	25
2.5(c) Elicitation phase	22
2.5(b) Sensitisation phase	21
2.5(a) Type IV reactions	21
2.5 Skin Sensitisation Reactions	
2.4(c) Types of allergic response	18
2.4(b) Allergic reactions	17
2.4(a) The human body and the immune system	17
2.4 Allergy and the Immune System	
2.3(c) Allergic Contact Dermatitis	15
2.3(b) Irritant Contact Dermatitis	14
2.3(a) Contact dermatitis	14

A PARTICIPACITY OF A PARTICIPACI

2.9(c) Host-related factors	49
2.9(d) The diagnosis of occupational skin disease-Patch testing	51
2.10 Prognosis of Occupational Dermatitis	
2.10(a) Prognosis of occupational dermatitis cases	54
2.10(b) Earlier reports on the prognosis of occupational dermatitis	54
2.10(c) Recent reports on the prognosis of occupational dermatitis	55
2.11 Contact Dermatitis and Workers Compensation	
2.11(a) Workers compensation laws	59
2.11(b) Work and skin conditions	61
2.11(c) Workers compensation in Ireland and the U K	61
2.12 Data Gathering on Occupational Skin Diseases	
2.12(a) Methods for data gathering	62
2.12(b) Incidence and prevalence	63
2.12(c) Passive data generation	64
2.12(d) Occupations most commonly associated with contact dermatitis	65
2.12(e) Data gathering for occupational skin disease in Ireland	66
2.13 Skin Management	
2.13(a) Occupational skin disease and its prevention	68
2.13(b) Elements of a skin management system	69
2.13(c) The use of protective gloves	75
2.13(d) Skin allergy to natural rubber latex	76

SECTION 3

RESULTS

3.1(a) Industries surveyed

77



3.1(b) Number of employees within the industries surveyed	78
3.1(c) Use of substances classified as skin sensitisers	78
3.1(d) The reported cases of 'contact dermatitis' among Irish Industry	79
3.1(e) Control measures for the of occupational skin disease	80
3.1(f) Specific characteristics of substances classified as 'skin sensitisers' which ma	ke
exposure control difficult	81

SECTION 4

DISCUSSION

SECTION 5

CONCLUSION

REFERENCES

88

91



LIST OF FIGURES

SECTION 2	
1.1 The Skin	1
2.2 Mechanism for the stimulation of an immune response.	20
2.5 Sensitisation and Elicitation reactions	24
2.13 Elements of a skin management system	69

SECTION 3

3.1(a)	Industries surveyed	77
3.1(d)	Cases of contact dermatitis among Irish Industries	79
3.1(e)) Control measures for the prevention of occupational skin diseases	80
3.1(f)	% industries which found exposure control difficult due to specific	
	characteristics of the substance	81



PAGE

LIST OF TABLES

1997

	PAGE
SECTION 2	
2.7 Classification scheme for skin sensitisers	42
SECTION 3	
3.1(b) The number of employees within the industries surveyed	78
APPENDIX A	
Allergic Contact Dermatitis and Patch Testing	
APPENDIX B	
Contact Dermatitis: Criteria for evaluating probable occupational causation	
APPENDIX C	
Reporting on Occupational Dermatological Diseases	
APPENDIX D	
Glove Selection Chart	
APPENDIX E	
Survey Questionnaire	
APPENDIX F	
Survey Matrix	
APPENDIX G	
National Authority for Occupational Health and Safety, Code of Practice.	
APPENDIX H	
Health and Safety Authority Annual Report 'Surveillance of work related duse	eases',



SECTION 1

INTRODUCTION



SECTION 1. INTRODUCTION

1.0 Background

This dissertation is concerned with the use and control of substances in Irish Industry, which are classified as 'Skin Sensitisers'. Skin diseases that are caused by a substance or condition in the workplace are the most frequently encountered occupational illness. While there are systems in place for the control of exposure of workers to respiratory hazards and exposure limits available to employers, such information is not available with regard to non-respiratory hazards such as those associated with skin disease. There is very little data available on the reported cases of occupational dermatitis and most of the data gathered is to assess the economic implications rather than the Health and Safety implications.

1.1 Aims and Objectives

- (i) To provide a review of the literature and research works of others so as to ascertain, the incidence and prevalence of skin diseases and to distinguish between skin diseases which are as a result of exposure to irritants and those which are sue to exposure to substances which cause allergic reactions in the skin.
- (ii) To review legislation and examine what information is available on skin sensitising substances.
- (iii) To survey Irish Industry to gather information on the extent of the use of substances, which have the potential to cause, skin sensitisation. To evaluate



the incidence of occupational skin disease within Irish Industry and assess how exposure control is managed.

(iv) To study the available statistical data available on the incidence of occupational skin disease and to establish whom is involved in the data gathering and the surveillance of occupational skin disease.

1.2 Methodology

So as to facilitate the literature review, library searches were carried out from the British Library, University College Galway and University College Dublin. Information was gathered from the World Health Organisation, Health and Safety Commission in Britain, Health and Safety Authority of Ireland, the Department of Health and the Department of Social Welfare. Various publications were purchased from the Government Publications Office and the National Institute of Occupational Safety and Health.

The legislation reviewed included current and proposed national and European legislation.

Information on the use and control of substances which are classified as 'Skin Sensitisers' was gathered from survey questionnaires sent to industries and from information collected from the Department of Health and the Health and Safety Authority of Ireland.



SECTION 2

LITERATURE REVIEW



SECTION 2. LITERATURE REVIEW

SUB-SECTION 2.1 THE SKIN AS A BARRIER

2.1(a) The structure and composition of the human skin.

In order to understand how skin disorders occur and to distinguish between those which are occupational and those which have little or nothing to do with the workplace it is necessary to have an understanding of the way in which the skin works. The correct functioning of the skin is vital if we are to survive. The skin barrier layer is very thin and easily damaged. To examine the way in which the skin works to protects us we need to know how it functions and how it is constructed.

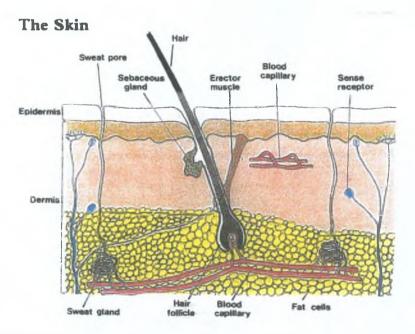


Fig.1 The structure of the human skin. (Adapted from 'Science Today' Kennedy, Porter, Scott, 1985)



The skin is the largest organ of the body forming an average around 10% of our total body weight and with a surface area of 2,880 square inches or 19 square feet. The skin is the body's outer layer and therefore as such it forms a two-way barrier. It not only works to prevent substances from the outside world from gaining access to internal organs but also retains body fluids. The skin is a tough flexible cover and because it is the first body barrier to come into contact with the elements, as well as industrial hazards of every type, the skin is subjected to attack from heat, cold, moisture, radiation, all kinds of dirt, fungus, bacteria, and penetrating objects (Anon, 1975a).

There are three distinct layers of tissue that make up the skin, the epidermis the dermis and the subcutaneous layer. The thickness of the skin varies from 0.5mm on the eyelid (the dermis is the thinnest here) to 3 or 4mm on the palms of the hand and soles of the feet (the epidermis is thickest here). The palms of the hands and the soles of the feet can have as many as sixty layers of cells whereas in some areas notably in the skin folds the axillae (armpits), the groin, under the breast and between the fingers and toes (Olishifski, 1988).

From the outside of the skin structure we find the lipid (oily) layer on the surface. This lipid layer has an acid pH, and it is composed of oil and sweat and can be easily washed off even with plain water. Beneath this lipid layer is epidermal cells variously called the horny layer, stratum corneum, or keratin layer. This layer stands up fairly well against chemical attack with the notable exception of alkalis. The layer is the chief barrier against water and aqueous solutions, but it offers no protection against lipid-soluble



materials such as solvents or gases. As the cells migrate and slowly transform from keratinocyte into corneocyte, small sacks appear within the cell filled with lameller bodies. The horny layer or stratum corneum, made up of several corneocytes, is constantly being replaced by cells pushed toward the surface as new cells are formed in the deeper germinative layer of the epidermis. This sloughing and regenerative characteristic serves to some extent to protect against chemicals and microorganisms.

There are four types of cells in the epidermis;

- Keratinocytes which make up the bulk of the epidermis form from below and move up to become dead horny cells.
- Melanocytes are cells which synthesise melanin (pigment) granules which are then transferred to keratinocytes. It is the amount of melanin in keratinocytes that determines the degree of pigmentation of skin and hair. Melanin proliferates under stimulus of certain wavelengths of sunlight and becomes visible as suntan or freckles. Albinism is an inherited abnormality in which melanin production is decreased. Vitiligo is a more common disorder where a loss in melanocytes results in areas of cutaneous pigment loss.

Occupational or environmental exposure to certain chemicals e.g. phenolic germicides can destroy pigment (Olishifski, 1988). Fukuyama *et al.* (1982) and Yonemoto *et al.* (1983), carried out studies on the pathomechanisms of chemically induced depigmentation by using tertiary butyl catechol (TBC) as the prototype depigmenting



compound. Anatomic alterations in melanin biosynthesis and in melanosomes have been clarified. In *vivo* and *in vitro* methods were employed on mouse and guinea pig skin and human melanoma cell lines. Assay of enzymes involved in melanin formation and light and electron microscopy have been employed. The major findings have included "conversion from eumelanin to pheomelanosome synthesis, enzymatic changes (in the melanocyte) such as increased activity of glutathione reductase and gamma-glutamyl transpeptidase, increased sulfus content of TBC-treated cells, and lightening of the melanocytes. Mice and guinea pig models were recommended for predictive screening for depigmentation produced by topically applied chemicals.

- Langerhan's cells, which are located in the mid-epidermis, play an important role in various immune process, especially allergic contact dermatitis and they account for four percent of all epidermal cells.
- Merkel cells function as slowly adapting receptors of the touch sensation.

The Lamellar bodies form the "seal" which keeps most of the moisture in the body permitting only enough to permeate through to keep the outer layers adequately moist. If this moisture were allowed to evaporate the stratum corneum would become more permeable. The layer is only a few microns thick and is essential for the correct functioning of our body. Any substance which has the capability of either emulsifying or dissolving fatty substances, is a potential hazard to the skin. Studies carried out by Overgaard *et al.* 1993, showed that if the barrier of the skin is measured by the amount



of moisture lost (trans-epidermal water loss - TEWL) then it can be shown that even plain water will affect the ability of the skin to provide protection as the TEWL will have increased.

The epidermis is not richly supplied with blood but it is bathed in lymph, a fluid derived from the blood. The epidermis is thin enough so that the nerve endings in the dermis are close and therefore supply the fine sense of touch. Beneath the epidermis is the dermis characterised by collagenous (connective) tissues which are a matrix of fibres called collagen and elastin in a base of jelly like substance. It is these fibres that give the dermis it tensile strength and toughness.

The dermis is the main natural protection against trauma and when injured, it can form new tissues in the form of a scar to repair itself. The dermis is laced with blood vessels, nerve fibres, receptor organs for sensations of touch, pain, heat and cold, contains muscular elements, hair follicles, and oil and sweat glands. A layer of tiny cone-shaped objects called papillae are present at the top of the dermis. Nerve fibres and special nerve endings are found in many of the papillae. The dermis is supplied with nerves to warn of changes in the environment, in addition to hair follicles, oil and sweat glands, and blood and lymph vessels. The sweat produced by the sweat glands may act as a protective mechanism to wash away an irritant but may also result in a chemical going into solution which may allow it to penetrate the skin more readily. Both the hair follicles and the sweat glands serve as routes of entry into the body through the skin. Physicians sometimes exploit the absorptive capability of the skin to administer certain drugs. Some chemicals that are placed on the skin can be detected in saliva a few minutes later. The absorptive characteristics of the skin can be an unfortunate one from the point of view of occupational health. Beneath the dermis is a layer of subcutaneous tissue with fatty and resilient elements which help cushion and insulate the skin above it. Present in the subcutaneous layer which distinguishes it from the other parts of the skin is fat. In the lower parts of this layer also lie eccrine and apocrine sweat glands and hairs as well as nerves, blood and lymphatic vessels, cells, and fibrous partitions composed of collagen, elastic tissue and reticulum. It links the dermis with tissue covering the muscles and bones (Anon, 1975a).

2.1(b) The Functions of the Human Skin

The skin performs a number of important functions and among these are the protection of the body against invasion of bacteria, against injury to vital internal organs, against the rays of the sun and against the loss of moisture.

The skin protects in the following ways:

1. The skin protects against physical damage and trauma, by the skin armed with sensory signals, by the strong resilient collagen tissue, and by its self repairing properties.

- 2. The skin has the defense of being naturally dry terrain (except in places like armpits, and in the groin, and during abnormal sweating), and has a normal contingent of bacteria that tends to destroy pathogenic bacteria. Free fatty acid oils in the surface oil may also have some antibacterial value.
- 3. Against sunlight, the skin has two defence mechanisms which include an increase in pigmentation and a responsive swelling to increase thickness.
- 4. The skin may protect against primary irritants by the natural defences such as the buffered acid mantle, the stratum corneum, thickening of the Keratin material and sweating.
- 5. Against the absorption of water and water soluble chemicals, there is considerable protection. Strong acids and caustics will produce chemical burns in short order.
- 6. The skin can offer like no protection in use, absorption of lipid-soluble chemical. Certain fat soluble chemicals such as benzene, carbon tetrachlorine, and carbon disulphide go through the skin easily and may cause serious system damage, or even fatal effects. The majority of solvents such as trichlorethylene naphtha, and toluene do not readily penetrate the lipid layer, but only prolonged contact with large skin areas will result in appreciable skin penetration (Anon, 1975a).

SUB-SECTION 2.2 MECHANISMS OF SKIN ABSORPTION

2.2(a) Dermal Absorption

The skin is made up of various types of cells which as mentioned in earlier sections form three distinct layers; the epidermis, the dermis and the hypodermis (consisting mainly of connective tissue and fat). Percutaneous absorption involves diffusion of the chemical through these layers until it reaches capillaries in the epidermis and hypodermis, and enters the systemic circulation. The epidermis also has a limited enzyme system and these can metabolise xenobiotics (Kao *et al.* 1985). Dermal absorption involves two major diffusion processes penetration through the stratum corneum and transfer into the capillary blood.

2.2(b) Dermal Penetration

Penetration through the stratum corneum, by diffusion via polar and lipophilic pathways, is restricted to small molecules (molecular weigh < 500). High solubility in water and in fat facilitates rapid penetration. The measure of penetration is either the permeability coefficient (which is the velocity constant in cm h -1) or flux (which is the penetration rate in mgcm-2 h-1). Flux (F1)and permeability coefficient (K) are related by the equation.

Equation 1 $F1 = k \Delta C.$

where ΔC is the concentration gradient across the stratum corneum.



Both K an d F1 vary over the body, depending on the composition and thickness of the stratum corneum, the presence of skin appendages (hair glands) and the amount of perspiration. The differences among animal species are even larger (Wester *et al.* 1977). Environmental temperature and humidity and, most importantly, the dispersant (vechicle) in which the chemical is administered also affect penetration (Dutkiewicz *et al.* 1961). Moreover, the nature of the skin and thus flux can be gradually altered by the applied chemical or by the dispersant. K and F1 can be measured both in vivo and in vitro.

The determination of the concentration on the receptor side of the skin is the most controversial step in the measurement. The methods were recently reviewed by the Environmental Protection Agency (1992). Because of differences in skin composition and in methodology the results reported vary widely. For example, the flux of Xylene, measured in a diffusion chamber using excised rat skin, was 0.006 mgcm-2h-1 (Tsuruta, 1982), whereas excretion of metabolites in humans indicates a flux of 0.13 mgcm-2h-1 (Engstrom *et al.* 1977).

Several theoretical approaches for the prediction of dermal penetration rate based on the physiological function of the skin and on the chemical structure and physical properties of the chemical were developed. Models based on similarity of chemical structure are reviewed in the EPA Interim report on dermal exposure assessment (EPA, 1992). Other models are based on the diffusion process and on the physiochemical properties of the



chemical and skin composition. In these models penetration is defined either by permeability coefficients (EPA, 1992) or by flux (models reviewed by Osbourne, 1986).

Although the permeability constant is time - independent, the penetration rate changes during the exposure as does the concentration gradient across the skin (Equation 1). In vivo, the change is apparent at the beginning of the exposure but becomes negligible when the apparent steady state is approached.

2.2(c) The Distribution and Elimination of Percutaneously Absorbed Chemicals

The transfer of the chemical from the dermis, into the capillary blood depends on the perfusion rate of the dermis, (and thus on physical activity of the person and environmental temperature), and on the dermis blood distribution coefficient of the chemical, (and thus on body fat and on the hydration of the skin), (Fiserova- Bergerova, 1990).

At the beginning of exposure, when the absorption rate is a function of time, the concentration builds up in the epidermis and dermis. During this period, the concentration gradient diminishes. The absorption rate becomes constant after the lag time period, when steady state is approached (that is, when the penetration and uptake rates are equal and the concentration gradients are constant). The rate limiting step is usually the penetration rate through the stratum corneum, but for lipophilic chemicals in poorly perfused areas it may be the removal of the chemical from the skin by capillary blood. Pharmaco-kinetic models were developed to describe the distribution and



elimination of percutaneously absorbed chemicals (Fiserova-Bergerova *et al.* 1990, Guy *et al.* 1985). The passing of the chemical into the capillary blood at steady state can be described by a balance equation.

Equation 2

Inflow = Outflow.

$$FC_{art} + F1 = FC_{ven}$$

where F is the perfusion rate of skin under the exposed area and C_{art} and C_{ven} are concentrations of the chemical in arterial blood and venous blood under the exposed area, respectively. If the diffusion is rapid, as in the case of volatile solvents, the concentrations in blood, alveolar air and dermis are instantly equilibrated and equation 2 can be rewritten in order to compare penetration rate with uptake rate;

Equation 3
$$F1 = F(C_{derm} \lambda_{b1/derm} - C_{alv} \lambda_{b1/air})$$

where C_{derm} and C_{alv} are concentrations of the chemical in skin under the exposed area and in alveolar air, respectively, and λ 's are the appropriate partition coefficient (Fiserova-Bergerova, 1990).



SUB-SECTION 2.3 CONTACT DERMATITIS

2.3(a) Contact Dermatitis

A number of different morphologic types of cutaneous reactions may occur when skin is topically exposed to chemical agents. The initial interation can lead to a variety of cell and agent-dependent biologic events resulting in an array of cutaneous and even systemic responses. These may include localised or generalised urticaria with and without anaphylaxis, which is mediated by most cell activation; acneiform eruptions in melanocyte biology resulting in hypopigmentation or hyperpigmentation; interaction of the chemical agent with non-ionising radiation and effects on dermal vessels that result in atropy or purpura. The most common pathologic response pattern resulting from skin contact with a chemical agent is contact dermatitis. Even the most experienced dermatologist can have difficulty distinguishing the two, as the signs, symptoms, and even histopathology overlap (Marks *et al.* 1997a).

2.3(b) Irritant Contact Dermatitis

An irritant is any substance that damages and causes an inflamatory reaction in the skin by direct action through a nonimmune mechanism. Several factors help determine the security of the skin reaction. They include properties of the irritant, such as pH, solubility, physical state (gas, liquid, or solid), and host factors. Host factors include the area of affected skin, oil gland and sweat gland activity, and the presence of or tendancy toward other skin diseases. Environmental factors such as temperature and humidity also play a role. Irritant dermatitis can occur in anyone if the concentration of an irritant



is high enough and the exposure is long enough. The hands and forearms are affected most often. Clinical findings in irritant dermatitis vary from mild erythema, itching and chapping to severe blistering and ulceration. The worst cases can be categorised as chemical burns, but most cases are mild. Mild cases may be insidious in onset. The dermatologist should be alert to the fact that chronic irritant dermatitis may be indistinguishable from allergid contact dermatitis. Even when a patient clearly has an irritant dernatitis, definitive diagnosis frequently can pick up an additional allergic etiology (Skellchock., 1995).

2.3(c) Allergic Contact Dermatitis

Allergic contact dermatitis can be defined as an acquired delayed, cell mediated reaction. The body's immune system recognises a foreign substance and responds in defence through a very complex interaction between many different cells, molecules and enzymes. Sensitising agents differ from primary irritants in their mechanism of action and their effect on the skin. Unless they are concomitant irritants, most sensitisers do not produce a skin reaction on first contact.

An essential difference between primary irritation and allergic contact dermatitis is that an irritant usually affects a number of people whereas a sensitiser generally only affects a few. Exceptions exist as in the case of potent sensitisers such as poison oak or epoxy resin and components. Differentiation of marginal irritants and cutaneous sensitisers may be extremely difficult. The former may require repeated or prolonged exposure before a dermatitis appears. Development of allergic contact dermatitis may not occur



for months or years after exposure to an agent, and sensitisation may be produced or maintained by allergens in minute amounts and in concentrations insufficient to irritate the nonallergic skin e.g. Nickel, chromates, formaldehyde and turpentime. Crosssensitivity is an important phenomenon in which a person sensitised to one chemical will also react to one or more closely related chemicals. Patch testing is an important diagnostic tool in differentiating allergic contact dermatitis from irritant dermatitis (Taylor, 1982).



SUB-SECTION 2.4 ALLERGY AND THE IMMUNE SYSTEM

2.4(a) The human body and the immune system

The human body possesses what we call the immune system, which is a highly complex network of molecules and cells. This system is designed to protect us from bacteria, viruses, chemicals and parasites. It does this by distinguishing between 'self' and 'non-self'. The system is continuously checking to identify 'foreign bodies'. If a body is recognised as 'foreign', then one of a series of defence mechanisms will come into play to deal with the invader either by destroying it or by rendering it harmless. These defence mechanisms are working for us virtually all the time, mostly without our being aware of this. Occasionally, they malfunction. They may react excessively either to something against which they should react or, commonly, to something which, whilst it may penetrate into our body, would normally be considered harmless. It is this overreaction which we generally call an Allergy or more accurately, hypersensitivity.

2.4(b) Allergic Reactions

Allergic reactions are those which occur when the immune system of the body is stimulated to react in a particular way. This may be the result of a toxic molecule being sufficiently large to be regarded as foreign by the immune system so as to act as an antigen. Chemical allergy is an adverse reaction that results from previous sensitisation to a particular chemical or to one that is structurally similar. Such reactions are mediated by the immune systems. The term 'hypersensitivity' is often used to describe the allergic state. For a low-molecule weight chemical to cause an allergic reaction its



or its metabolic product usually acts as a Hapten combined with an endogenous protein to form an antigenic complex. Such antigens induce the synthesis of antibodies, usually after a latent period of at least one or two weeks. Subsequent exposure of the organism to the chemical results in an antigen-antibody interaction that provokes the typical manifestation of allergy. Dose-response relationships are usually not apparent for the provocation of allergic reactions.

2.4(c) Types of Allergic Responses

The allergic responses have been divided into four general categories, based on the mechanism of immunological involvement.

- ⇒ Type 1 or anaphalactic reactions in man are mediated by IgE antibodies. The Fc portion of IgE can bind to receptors on Host cells and basaphils. If the antibody molecule then binds with antigen, various mediators (histamine, leuotrotrienes, prostaglandins) are released and they cause vasodilation edema and an inflamatory response. The main targets of this type of reaction are GIT (food allergies), the skin (utricary and atropic dermitis), the respiratory system (rhinitis and asthma) and the vasculature (anaphylactic shock). These responses tend to occur quickly after challenge with an antigen to which the individual has been sensitized and are termed Immediate hypensitivity reactions.
- ⇒ Type 2 or cytalytic reactions mediated by both IgG and IgM antibodies and are usually attributed to their ability to activate complement. The major target tissues are



the cells in the circulatory system and they can be destroyed. Examples of this phenomenon include penicillin - induced haemalytic anemia quinidine - induced granulocytopenia and hydralazine or procainamide - induced systemic lupus erythematosus. Fortunately, these autoimmune reactions to drugs usually subside within several months after removal of the offending agent.

- ⇒ Type 3 or Arthus reactions are predominantly mediated by IgG; the mechanism involves the generation of antigen - antibody complexes that subsequently fix complement. The complexes become deposited in the vascular endothelium where a destructive inflammatory response called serum sickness occurs. This is in contrast to the Type 2 reaction in which the inflamatory response is induced by antibodies directed against tissue antigens. The clinical symptoms of serum sickness include urticarial skin erruptions, arthralagia, or arthritis lymphadenopathy, and fever. These reactions usually last for six to twelve days and then subside after the offending agent is eliminated. Several drugs, such as sulfonamides penicillins, certain anticonvulsants, and iodides, can enduce serum sickness. Stevens Johnson syndrome such as that caused by sulfonamides, is a more severe form of immune vasculitis. Symptoms of this reaction include erythema multiforme arthritis nephritis, CNS abnormalities and mycocarditis.
- ⇒ Type 4 or delayed-hypersensitivity reactions are mediated by sensitized lymphocytes and macrophages. When sensitized cells come in contact with antigen, an

inflammatory reaction is generated by the production of lymphotines and the subsequent influx of neutrophil and macrophages (Packham, 1998c).

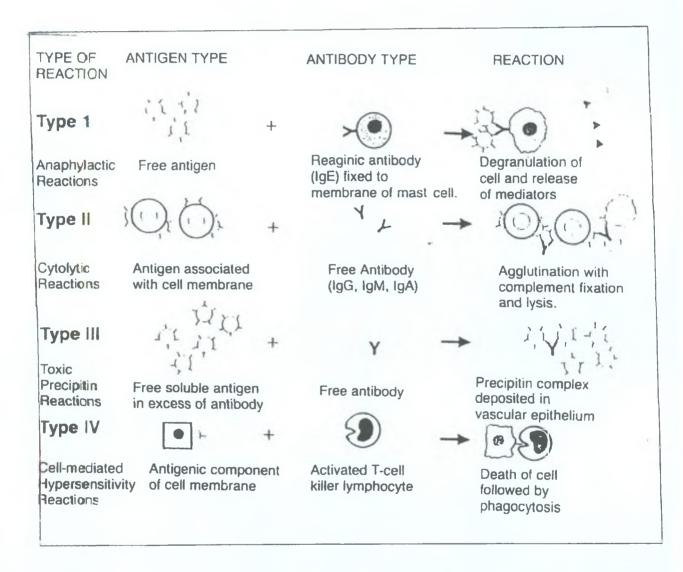


Fig.2. Mechanisms for the stimulation of an immune response (Adapted from Bowman, W.C. and Rand

M.J., Textbook of Pharmacology, 2nd Ed., Blackwell Scientific Publishers, Oxford,



SUB-SECTION 2.5 SKIN SENSITISATION REACTIONS

2.5(a) Type IV Reactions

Allergic contact dermatitis is a classic delayed hypersensitivity, or a Type 4 immunologic reaction. By definition it is mediated by immune cells rather than by antibodies. The reaction can be thought of as occurring in two phases, initially a sensitisation and then an elicitation response. It is the first or sensitisation phase that is the basis for its classification as an immune-mediated reaction

2.5(b) Sensitisation Phase

The allergen is a chemical that is usually, but not always, of low molecular weight, lipid soluble, and highly reactive. An unprocessed allergen is more correctly referred to as a hapten. The hapten is applied to the stratum cormeum, penetrates to the lower layers of the epidermis, and is taken up by the Langerhans' cell by pinocytosis. Within the cell lysosomal or cytosolic enzymes chemically alter the hapten, and it is conjugated to a newly synthesized HLA-DR molecule to form the complete antigen. This complex is expressed on the surface of the Langerhans' cell.

The next step is presentation of the HLA-DR-antigen complex to specific helper T cells that express both a CD4 molecule that recognizes the HLA-DR of the Langerhans' cells and more specifically a T-cell receptor-CD3 complex that recognizes the processed antigen. The presence or absence of specific T cells is most likely genetically determined. As stated earlier, this specificity that allows interaction with thousands of



antigens is developed by T-cell receptor rearrangements during early thymus development. It is unlikely that this initial HLA-DR-antigen and T-cell receptor-CD3 interaction occurs in the skin. It is believed that the Langerhans' cell migrates via the lymphatics to regional nodes where it presents the HLA-DR- antigen complex to specific T cells. Once antigen recognition occurs, both cells are activated. A series of cytokines is synthesized by both the Langerhans' cell and the T cell. Within the T cell this message is transmitted via the CD3 molecule.

The Langerhans' cell secretes IL-1, which stimulates the T cell to secrete IL-2 and to express IL-2 receptors. This cytokine leads to stimulation of T-cell proliferation, thereby expanding the clone of specific T cells capable of responding to the inciting antigen. This occurs during the classic lag phase of sensitisation. The primed or memory T cells that are generated are now much expanded as compared with the original population of cells with the specific T-cell receptor, and they leave the node and circulate throughout the body. The individual is now sensitised, or primed, to respond when these circulating T cells are re-exposed to antigen.

2.5(c) Elicitation Phase

The second phase, or elicitation of the delayed type of hypersensitivity, occurs on reexposure. Once again, hapten diffuses to the Langerhans' cell, it is taken in and chemically altered, it is bound to the HLA-DR, and the complex is expressed on the surface of the Langerhans' cell. The complex interacts with primed T cells in either the

skin or the node (or both), and the activation process takes place. In the skin the interaction is even more complex because other cells are present.

Langerhans' cells secrete IL-1, which stimulates the T cell to produce IL-2 and express IL-2R. Once again, this leads to proliferation and expansion of the T-cell population, this time within the skin. In addition, the activated T cells secrete IFN- γ , which activates the keratinocyte and causes it to express both ICAM-1 and HLA-DR. The ICAM-1 molecule allows the keratinocyte to interact with T cells and other leukocytes that express the LFA-1 molecule. Expression of HLA-DR allows for the keratinocyte to interact directly with CD4-bearing T cells and may allow for antigen presentation to these cells as well.

In addition, HLA-DR expression may make the keratinocyte the target for cytotoxic T cells. Activated keratinocytes also produce a number of cytokines, including IL-1, II-6, and GMCSF, all of which can further expand the involvement and activation of T cells. In addition, IL-1 can stimulate keratinocytes to produce eicosanoids. This combination of cytokines and eicosanoids leads to activation of mast cells and macrophages. Histamine from mast cells and eicosanoids from mast cells, keratinocytes, and infiltrating leukocytes lead to vascular dilation and increased permeability to circulating pro-inflammatory soluble factors and cells. This cascade leads to the clinical ACD response of inflammation, cellular destruction, and reparative processes (Rietschel *et al.* 1995a).



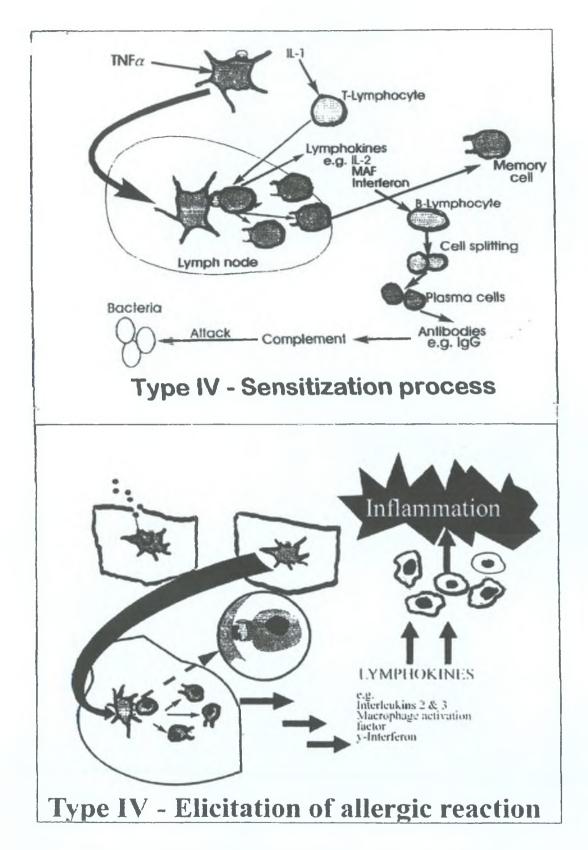


Fig.3 Sensitisation and Elicitation Reactions. Taken from Packham, 1998



2.5(d) Activation of Supressor Pathways

In addition to sensitisation followed by the elicitation scenario outlined earlier, exposure to antigen may also result in activation of suppressor pathways. The net balance of sensitisation and suppression resulting in disease or no disease on exposure to antigen depends on many factors. Presentation of a high concentration of antigen during the first exposure may result in the generation of specific suppressor T cells. Exposure to antigen through a site other than skin (eg, orally or intravenously) may also result in specific suppressor-cell generation. Such responses may be due to exposure of T cells to antigen that has not been processed by Langerhans' cells.

Many other poorly understood processes surely "downregulate" the immune response; for example, atopic individuals have decreased capacity to be sensitised to common allergens. This effect probably resides within the T cell. The balance between sensitisation and suppression on exposure to antigen undoubtedly results most frequently in the latter effect; otherwise, allergic contact dermatitis would be a much more common problem. Such downregulation is certainly necessary for the survival of humans exposed frequently to a myraid of possible environmental allergens (Rietschel *et al.* 1995c).



SUB-SECTION 2.6 OCCUPATIONAL SKIN DISEASES

2.6(a) Occupational Dermatoses

Occupational dermatoses are any abnormal conditions of the skin caused or aggravated by substances or processes associated with the work environment. Occupational skin disease is still tha most frequent of all occupational illness. As in other disciplines associated with occupational medicine, it is essential to think of occupational dermatology not just in terms of diagnosis and treatment but also from the standpoint of preventative medicine. The latter requires multidisciplinary approach solving industrial medical problems with knowledge of chemistry, physics, industrial hygiene and safety, industrial relations and governmental laws and regulations. Packham (1998d) offers the following definition:

"A clinically recognisable impairment of the skin's normal state due entirely or substantially to conditions in the workplace".

In many cases, the origin of the skin disease is multi-factorial and has both workplace and non-workplace causes. The definition given allows for this, the definition however does not incorporate skin penetration causing damage to internal organs (ie Systemic toxicity). This is a significant problem, but since there may be no apparent damage to the skin it cannot be considered under the heading of skin disease itself.



2.6(b) Direct Causes of Occupational Dermatitis

Agents directly responsible for occupational skin disorders may be divided into five groups;

1) Chemical

2) Mechanical

3) Physical

4) **Biological**

5) Botanical

Organic and inorganic chemicals account for most occupational skin disorders. All occupational health personnel are confronted each year by increasing number of chemical substances introduced into the workplace. Approximately 1.95 million chemicals are tested by the Chemical Abstract Registry Number system with 250,000 chemicals added to this list annually. Very few of the estimated three hundred to five hundred new chemicals with commercial application each year have been subjected to any significant amount of toxicologic investigation. The dermatologic effects of such agents may go unrecognised for long periods following introduction into industrial use (Lucas, 1974).

Mechanical causes include, friction and trauma, pressure and fibrous glass.

Physical agents include, heat, cold, vibration, sunlight and ionising radiation.

Biological agents include, bacterial viruses, fungi parasites and anthropods which may attack the skin and sometimes produce systemic disease of occupational origin.

2.6(c) Sensitisers

Contact dermatitis is the most frequent cause of occupational skin diseases. Two types are recognised; irritant and allergic. Environmental agents that are potential antigens and may cause disabling ezematous allergic dermatitis pose a great occupational health problem. Some chemical and many plant substances as well as biological agents are classified as skin sensitisers. Initial skin contact with them may produce no irritation, but after repeated or extended exposure, some individuals will develop an allergic type of reaction termed sensitisation (Taylor, 1982).

Sensitisation dermatitis varies greatly from individual to individual, and the onceimmune person may, at any time, suddenly develop an allergic reaction to a particular substance. This allergic reaction or sensitisation often looks like a contact dermatitis ie small pimples or watery blisters. Peculiar to this form of dermatitis, the skin reaction does not necessarily appear at the site where actual skin control occurred. The reaction is due to the physiochemistry of the individual, thus explaining an outbreak within the person, although he has worked with a product for a number of years. Once a person has become sensitised to any material, about the only way to prevent future occurrences, besides medication or desensitising by a physician, is to remove him from all future contact with that particular product (Anon, 1975b).



2.6(d) Principal Occupational Sensitisers

Packham (1998c) gives a list of those substances which have been shown to be sensitisers and to which workers may be exposed. This listing does not show which sensitisers are the most common, nor does it claim to be a comprehensive listing of all sensitisers.

The following list attempts to identify those substances which have been shown to be sensitisers and to which workers may be exposed. It does not attempt to show which sensitisers are the most common, nor does it claim to be a comprehensive listing of all sensitisers.

1. Antimicrobial agents

Parabens, p-chloro-m-cresol, formaldehyde and formaldehyde releasers (eg Grotan BK, Bronopol, Dowicil 200), quaternary ammonium salts, organic mercury compounds, hydroxyquinolines, hexachlorophane, phenoxyethanol, chloramine, thiurams, resorcinol, dichlorophene, Chloracetamides, DNCB, para-tertiary-Butylphenol. Ethylenediamine, Irgasan, Isothazolinimes.

2. Antioxidants

Derivatives of anilines, eg. Para-phenylene-diamine (PPD), phenols, Carbamates.



3. Perfumes

It is recommended that any perfume contained in a product should be one approved by IFRA (International Fragrance Association). Many perfumes can be sensitisers, even in minute quantities.

4. Colophony

Different types of colophony exhibit different sensitising potential, depending upon their source and the degree of refining that has taken place.

5. Metals

Nickel and chrome are the two most common sensitisers, but others may occasionally sensitize. Nickel is probably the most common sensitiser of all, largely due to the level of exposure that occurs, particularly among women.

6. Medicaments

Many medicaments contain chemicals that can sensitise however, these are unlikely to be occupational unless the worker is employed in a factory producing such products.

7. Organic dyes

Most of the organic dyes known to be sensitisers are aniline derivatives. They occur in textiles, shoes, rubber, plastics etc.



8. Pesticides (see also Fungicides)

A wide range of substances contained in pesticides are known to sensitise. In view of their potential toxicity, contact between the skin and all pesticides should be avoided.

9. Photographic chemicals

Many of the chemicals contained in photographic chemicals are sensitisers. As a general rule, contact should be avoided through the use of gloves and appropriate applicators.

10. Plants and Woods

Many plants are sensitisers. The most common are probably those of the primula family. Many woods, particularly tropical hardwoods, contain substances that may sensitise.

11. Plastics

Many plastics contain substances such as methacrylates. Many of these are known to be potent sensitisers. Epoxy and phenolic and polyester resins are notorious for sensitising effect of these substances remains for several days, even after the resin has set.

12. Rubber compounds

Natural rubber contains proteins which may cause contact urticaria. It also contains a range of additives (such as thiurams, mercaptobenzothiazoles, carbamates) known to be sensitisers. Items which may sensitise are gloves, shoes, rubber handles on tools, rubber tyres and fan belts etc. With gloves, good manufacturers take considerable care to

reduce the amount of the sensitising substances to an absolute minimum. "Cheap" gloves, by comparison, may contain very high levels and lead to sensitisation.

13. Tars

Coal tar, creosote, asphalt etc..can all sensitise. Creosote is particularly hazardous on the skin if exposed to UV radiation (sunlight).

14. Turpentine

Oil of turpentine can sensitise. The probability of a reaction will depend upon the origin of the turpintine.

15. Wood preservatives

The main problem is creosote, particularly when applied in sunny conditions, but many other preservatives for wood contain substances capable of sensitising.

16. Alcohols

Some types of alcohol may sensitise.

17. Benzoyl peroxide

Used as a flour improver.



18. Foods

These are more likely to result in an urticarial reaction, but sensitisation does occur from time to time.

19. Organic silicones

These are sometimes used in so-called "barrier creams". They are also found in specialised lubricants used in industry.

20. Glues, adhesives and sealants

These are frequently based on methacrylates and often cause sensitisation.

21. Lanolin

Whilst many people can use lanolin successfully as an excellent product for skin conditioning, there is a number of people who will become sensitised and who should therefore select skin creams etc. not containing this substance.

22. Metalworking fluids

The most common problem with these is irritant contact dermatitis from the degreasing effect of the fluid. The second most common problem is allergic contact dermatitis to the biocides in the fluid. These are usually formaldehyde releasers. Other ingredients, eg. The corrosion inhibitors and extreme pressure additives can occasionally sensitise.



23. Biocides

The same comments apply as for fungicides and pesticides. Almost all the known biocides will damage the skin, many are potent sensitisers.

SUB-SECTION 2.7 CLASSIFICATION OF SKIN SENSITISERS

2.7(a) European legislation in the classification of skin sensitisers

In the 1960's the national provisions of the six member states on chemicals differed widely and thus hindered Community trade. It was recognised that there was a need to ensure the protection of public health, in particular the health of workers handling dangerous substances. This resulted in the adoption of Directive 67/548/EEC in 1967 to approximate the national provisions relating to dangerous substances.

EC Directive 67/548/EEC (European Council, 1967)

The Directive introduced common provisions for :

- The classification of dangerous substances, since placing a substance into one or several defined classes of danger characterises the type and severity of the adverse effects that the substance can cause.
- The packaging of dangerous substances, since adequate packaging protects from the unknown danger(s) of a substance

• The labelling of dangerous substances, since the label on the packaging informs about the nature of the danger(s) of the substance inside and about the safety measures to apply during handling and use.

The 6th amendment to the Directive 67/548/EEC adopted in 1979 introduced the notification systems for 'new' substances and consequently required the establishment of the list of 'existing' substances. EINECS, the European Inventory of Existing Commercial Chemical Substances (European Commission Communication, 1990) lists all substances that were reported to be on the market on or before 18th September 1981. The substances placed on the market for the first time after this target date are 'new'.

The presence of a substance in the European List of Notified Chemical Substances (ELINCS) does not authorise any new importer and/or manufacturer placing it on the community market from notifying it in accordance with Directive 79/83/EEC. However if the substance has already been notified, the competent authority may accept that the new notifier refer, as far as the technical dossier is concerned, to the results of studies carried out by a previous notifier or notification, with his or their written agreement. This is in particular to avoid as far as possible the repetition of tests using vertebrate animals. In accordance with Decision 85/71/EEC the classification of these substances is included in ELINCS only if it has been officially adopted at community level and therefore appears in Annex 1 to the Directive.



In Annex 1 to the Directive 79/831/EEC the section relating to classification is only present if the substance has been officially classified at community level. Where the substance has not been officially classified at community level but has been provisionally classified by the notifier, an asterisk is placed in the classification section. Where the substance has not been provisionally classified by the notifier and no decision has yet been taken to classify it or not classify it at community level, the classification section is not present for the substance in question (European Commission, 1994).

The 7th amendment of the 67/548/EEC Directive ,(EC Directive 93/67/EEC) of 1992 essentially required that the principles of risk assessment for 'new' substances be laid down. It further introduced the 'sole representative' in the notification system, and added the Safety Data Sheet as a hazard communication facility for the professional user.

Currently there are fifteen classes of danger in Directive 67/548/EEC, such as 'explosive', 'very toxic', 'carcinogenic', or 'dangerous for the environment'. The EU Directive (67/548/EEC) offers the following definitions of substances and preparations;

Substances 'chemical elements and their compounds in the natural state or obtained by any production process, including any additive necessary to preserve the stability of the products and any impurity deriving from the process used, but seperated without affecting the stability of the substance or changing its composition'. Preparations 'mixtures or solutions composed of two or more substances'

(European Council, 1996).

The Directive 67/548/EEC also includes a list of substances classified as dangerous in Annex 1, danger symbols (such as skull with crossed bones underneath) in Annex 11, standard phrases on the nature of special risks from substances (R-phrases) in Annex 111 and the wording of safety precautions phrases (S-phrases) relating to the handling and use of dangerous substances in Annex 1V. Annex V contains testing methods to determine the dangerous properties of substances, Annex V1 provides detailed criteria on the proper choice of the class of danger and how to assign the danger symbols, Rand S- phrases to a tested substance. Annexes V11 and V111 relate to the notification of 'new' substances. Annex 1X includes provision on child proof fastenings and tactile warning devices as special packaging and labelling elements.

The Directive is permanently updated to take into account of the scientific and technical progress in the field of dangerous substances. Until today it has been amended 8 times and adapted to technical progress 24 times.

2.7(b) Criteria for Classification of substances

Classification of chemicals in order to identify their adverse properties has been taking place in a number of governmental and scientific bodies throughout the world. Efforts to harmonise the criteria for classification of substances has been initiated. At the UN conference in RIO in 1992 (United Nations, 1992) there was a commitment of participants to work toward a global harmonisation of classification systems. The World Health Organisation (WHO) regional office for Europe in comprehension of this agreement is working to develop a system for classification of allergens that is compatible with existing regulation trends. The Organisation for Econmic Co-operation and Development (OECD) has been designated as the co-ordinating body to achieve the harmonisation of classification systems relating to toxicological properties of sensitising substances.

One of the widely used rules for classification in the OECD is the European Union (EU) legislation. The various effects of chemicals can be divided into various categories of danger. Among these, sensitising substances and preparations can be defined as;

'Substances and preparations which, if they are inhaled or if they penetrate the skin are capable of eliciting a reaction of hypersensitisation such that a further exposure to the substance or preparation, characteristic adverse effects are produced' (European Council, 1996).

When classifying sensitisers, the criteria of the directive are applied. The following is a draft of the EU criteria for the classification of Skin Sensitisers, 1997.

There is sensitisation by skin contact :

- (i) If practical experience shows the substance or preparation to be capable of inducing sensitisation by skin contact in a substantial number of persons.
- (ii) Where there are positive results from an appropriate animal test.
- (iii) Substances producing signs of immunological contact urticaria.

Human evidence would include;

- a) Positive data from appropriate patch testing normally in more than one dermatological clinic.
- b) Epideminological studies showing allergic contact dermatitis caused by the substance (situations in which a high proportion of those exposed exhibit characteristic symptoms are looked at with special concern, even if the number of cases is small.
- c) Positive data from experimental studies in man. If evidence is available to demonstrate in practice that the toxic effect of substances and preparations, on man is or is likely to be, different from that suggested by the experimental results obtained in animal tests or by the application of concentration limits for classification of preparations, then such substances and preparations should be classified according to their toxicity in man. However, tests on man should be discouraged and should not normally be used to negate positive animal data.

A substance may be classified as a skin sensitiser when there is supportive evidence such as:

- a) Isolated episodes of allergic contact dermatitis or
- b) Epidemiological studies where chance, bias or confounders have not been ruled out fully with reasonable confidence.

This supportive evidence may include, data from animal tests performed according to existing guidelines, with a result that does not meet the criteria given in the section on

animal studies but is sufficiently close to the limit to be considered significant, or data from non-standard methods or appropriate structure – activity relationships.

Animal studies used to classify substances as Skin Sensitisers would include positive results obtained from appropriate animal tests. In the case of the adjuvant type test method for skin sensitisation, detailed in the test methods described in Directive (67/548/EEC, Annex V) a response of at least 30% of the animals in the Guinea Pig Maximisation test is considered as positive, and for any other test method a response of at least 15 % of the animals is considered positive (Tobiassen, 1997).

The Swedish Chemicals Inspectorate, in its capacity of rapporteur to the Harmonisation Advisory Body under OECD, produced a draft summary review of the criteria used in the OECD member states for the classification of sensitisers. They concluded that the various member states only differed on minor points from the EU criteria described above. Some of the countries outside the EU apply similar criteria including Australia, Norway and Switzerland.

According to the Swedish report, classification of sensitisers in Canada are also close to the EU criteria, although the focus is more specifically on evidence from the workplace. In the United States, several agencies are involved in the regulation of chemicals. The criteria addressing sensitisation have many points in common with the EU criteria and use clinical pictures of sensitisation when describing the effects (Organisation for Economic Co-operation and Development, 1996).



In January 1996, in Copenhagen, Denmark, the WHO regional office for Europe and the National Institute of Occupational Health, Copenhagen, Denmark, organised a working group on criteria for classifying skin substances in the work and general environments with invited international experts. The meeting was co-sponsored by the Nordic Council of Ministers and the Swedish Building Research Council. The participants reached a consensus on criteria to identify and classify significant skin sensitisers. Evidence relevant for classifying substances was grouped as human, animal and other. Significant skin sensitisers were classified into four classes. The classification principle corresponds to the criteria for carcinogens of the International Agency for Research on Cancer.

For skin sensitisers, a classification scheme was developed for categorising substances as significant contact allergens (Class I) and probably significant allergens (Class II). Class III was designated as substances non classifiable. Class IV, not a significant contact allergen was used if many people have been extensively exposed to the substance for a long time, but contact allergy is extremely rare. It was recommended that the proposed criteria be adopted by member governments (Anon, 1997a).



Class	Human Evidence	Animal Evidence	Other Evidence
I. Significant	Sufficient	Evidence may be	Evidence may be
Contact allergan	evidence present	present <u>or</u> absent.	present or absent.
	Limited evidence present	Sufficient	Evidence may be
		evidence present.	present or absent
II. Probably	Inadequate	Sufficient	Evidence may be
a significant contact allergan	evidence present	evidence present.	present or absent
		Limited evidence present	Evidence present
	Limited evidence present		
III.			L
Not Classifiable	All other	All other possible combinations, but see Class IV	
		below.	
IV,		I	
Not a significant	Many people have been extensively exposed to the substance for a long time, bu		
contact allergan	contact allergy is extremely rare.		

Table No.1Classification Scheme for Skin Sensitisers (WHO, 1996)

With regard to the classification scheme above the following terms are used:

Significant contact allergan refers to:

Substances which are (presumed) capable of causing more than isolated cases of allergic contact reactions.

Sufficient evidence refers to:

- (i) Epidemiological studies and/or studies in consecutive skin tested patients conducted in accordance with well established principles which demonstrate an association between exposure and the clinical evaluation of dermatitis/contact urticaria, including positive skin tests.
- (ii) The substance is found to cause contact sensitisation in at least two separate animal studies (at least one of which must be in the guineapig) – the contact sensitising capacity should be statistically significant in comparison with nonsensitised control animals.

Limited evidence refers to:

- (i) Isolated cases of allergic contact reactions demonstrated by properly conducted skin tests in the presence of relevant exposure and in more than one independent centre.
- (ii) Where the substance is found to have contact sensitising ability in one OECD test method. The contact sensitising capacity should be statistically significant in comparison with non-sensitised control animals.

Inadequate evidence refers to:

Where individual cases of allergic contact reactions demonstrated by skin tests in which the requirement for limited evidence is not satisfied (WHO, 1996).

In Ireland, the control on importation of substances is largely on the onus of the Revenue Commissioners who operate a tariff code system that groups chemicals into broad categories. It is not possible to identify individual chemicals, hence information relating to the volume of substances classified as skin sensitisers, which are imported into Ireland on an annual basis is currently unavailable.

The Health and Safety Authority is the sole representative in the notification system for substances and preparations in Ireland in accordance with Council Directive 92/93/EEC. As the NONs authority in Ireland, the H.S.A. has a record of the new chemicals classified as skin sensitisers that have been brought into the country since the notification scheme started in 1982. The precise figures on the quantities is not available, also there are no records of imports of 'existing' substances classified as skin sensitisers. There are three thousand, seven hundred chemicals that can cause allergic contact dermatitis (De Groot, 1994) and data on new ones are published every year.



SUB-SECTION 2.8 VARIABLES IN OCCUPATIONAL SKIN EXPOSURE

2.8 Evaluation of dermal absorption

The variables which are critical in the evaluation of dermal absorption in the workplace are:

- Form of the chemical.
- Duration of dermal exposure.
- Exposed area (size as well as location on the body).
- Presence of other chemicals (mixtures constituent dispersant).
- Workload and environmental factors (humidity and temperature).

(i) Form of Chemical

In an industrial setting dermal absorption can result from exposure to vapours or from skin contact with liquid chemicals or their solutions. Dermal absorption of gases and vapours of volatile chemicals is usually negligible compared to pulmonary absorption (Riihimaki *et al.* 1978). However, the vapours of chemicals with low vapour pressure, such as furfural (Flek *et al.* 1978) or chemicals with high aqueous solubility, such as methanol (Sedivec *et al.* 1981) can condense on the body surface, and consequently their availability for dermal penetration can be increased. Dermal penetration of solids (dust, aerosols, etc) can be facilitated by their dissolution in pespiration.



(ii) Exposed area of the Skin

While the surface area of skin exposed to vapours and gases is the same as the whole body surface area of skin exposed to aerosols, dust and liquids is difficult to estimate. Protective apparel can reduce the exposed area, but contaminated, dirty apparel can enhance the chemical availability for dermal absorption (Trojanowska, 1959). Spills can result in an unpredictable dermal exposure of a large body surface. Moreover, the thickness of the layers of skin cells on different parts of the body varies so that the penetration rate varies (Scheuplein and Bronaugh, 1983).

(iii) Presence of other chemicals

Studies with drugs have proved that absorption and therapeutic effects depend on the vehicle by which the drug is administered (Cooper, 1985). The same applies to industrial chemicals. Dermal contact with a mixture of chemicals can alter the penetration rate by two mechanisms;

- 1) The penetration may be slowed down if the chemical is readily soluble in the dispersant.
- 2) Biochemical and skin permeability changes may occur in the skin as a result of prolonged contact with liquids. If the dispersant damages the skin, then the penetration rate of mixture components is expected to increase.



(iv) Workload and Environmental Factors

Since the dermal absorption depends on blood perfusion of the dermis and hypodermis under the exposed area the absorption rate increases with movement and ambient temperature; this means that increased workload and heat enhance dermal absorption (Dutkiewicz *et al.* 1961; Fiserova-Bergerova, 1990).

SUB-SECTION 2.9 PREDISPOSING FACTORS FOR SKIN DISEASES

2.9(a) Predisposing Factors in Occupational Skin Disease

Predisposing factors for occupational skin disease as a subject appears to take a back seat to history and therapy when occupational physicians are confronted with a patient with a work related disease. Encompassing all occupational skin disease cases, the affected workers who emerge as cases from a population of many workers do not do so randomly. Certain factors related to the workplace and certain factors intrinsic to the employees have to be present that, when combined, result in a work related disease. It os important to isolate and examine these special host and environmental factors as a specific area.

2.9(b) Job-related factors

There are ways in which job-related factors predispose to the development of occupational skin diseases. The one most direct involves the actual work environment. Job-related pre-disposing factors for occupational dermatoses that are unique to the



workplace or task include the following; wet work, irritating chemicals and allergens, heat, cold, humidity, vibration and radiation.

- a. Wet work looms as one of the most ubiquitous workplace factors, but oddly enough receives little attention (Orris *et al.* 1982). While singling out more obvious irritants and allergens, water as an irritant is often overlooked. In clinical practice, one is struck by the preponderance of skin problems among workers whose jobs involve intermittent water exposure (Lammintausta, 1981).
- b. Irritating chemicals in the workplace are well known and thus usually considered in the evaluation of work-related dermatitis. The irritants themselves may often be a mixture, including solvents, soaps, detergents, plant juices, antioxidants, acids, alkalis, reducing agents, cutting oils and many more
- c. Allergens are chemicals that provoke specific delayed hypersensitivity. Although each industry has its own set of common potential allergens, certain chemicals are ubiquitous enough or are potent enough sensitisers to be frequent offenders.
- d. Heat, cold, humidity, radiation and vibration are important physical and mechanical environmental factors that may contribute to the effect of chemical agents.
- e. Workers in certain outdoor industries namely forestry, agriculture, fishing and food processing have significant problems associated with mites, plants, bites, zoonotic mycors and viruses.
- f. As a predisposing cause of occupational skin disease, trauma on a micro scale may figure importantly. For example, machinists have ample opportunity to develop

minute cuts in addition to chemical exposures. Similar micro trauma plays a role in the inoculation of pathogens in industries with biohazards (Marks *et al.* 1997b).

2.9(c) Host Related Predisposing factors

Host related factors are of two types, those that represent variation within normal but that still predispose to work-related disease, and those that are abnormal enough to be considered clinical skin disease and that can flare under certain conditions or predispose to other work-related disease.

Host variations are usually not enough to cause work-related disease, however. Where job-related factors and host variation heighten the vulnerabilities of the employee's skin, then the possibility that a work-related skin disease will result is increased.

- Individuals with dry skin fare poorly in work environments involving solvents, soap, detergents and intermittant water exposures. These workers can easily develop hand eczema if their hands are exposed long term to chemicals in a work environment.
- The resistance to skin disease decreases with age. Clinical dermatitis may seem less reactive at first, but both allergic and irritant eruptions tend to persist and to be more resistant to therapy in aging skin (Fischer, 1986).
- Greater degrees of hairiness have been cited as a predisposing factor in the development of falliculitis, and it would appear that follicles bearing large terminal hairs in areas of friction, sweating, and oil exposures would be predisposing to follicular irritation (Cohen, 1982).

- Sweating serves a crucial role in many aspects of occupational skin diseases, and individuals who are hyperhidrotic may be relatively predisposed. Sweating may place in solution potential allergens and transport them to the skin, where if drying does not occurt, percutaneous penetration is enhanced by the occlusive environmental set up by this process.
- The rate of existing skin disease in the development of other work-related skin disease is very important and may result in reasonable intervention at the job applicant stage. Some intercurrent skin diseases are, atopic eczema, acne, psoriasis and cutaneous allergies. Atopic skin is uniquely vulnerable to dermatitis because of its diminished threshold for irritation (Rajka, 1975).
- Individuals who are acne prone, are predisposed to aggravation of acne under workrelated conditions (Ancoma, 1986).
- Psoriasis being a common dermatalogic disorder in general, may be of particular significance in terms of occupational aggravation.
- Cutaneous allergies can be an important predisposing host factor, particularly if the allergen is a ubiquitous one and if the degree of sensitivity to the substance is great. Individuals allergic to chromate are a good example, and it is hard to find work environments free of chromates that would not aggravate an individual who is very sensitive. A person who is allergic to one substance is not necessarily more likely to become allergic to other dissimilar substance than one who is not allergic at all, therefore monoallergy does no predispose to polyallergy (Fischer, 1986b).

Awaremess of certain predisposing factors, by employers and employees should have a significant impact on work-related skin diseases and the role of the dermatologist in providing this information is essential (Fischer, 1986a).

2.9(d) The Diagnosis of Contact Dermatitis - Patch Testing

Contact sensitisation is never hereditary but a consequence of earlier exposure to a chemical. In humans contact sensitisation is diagnosed by a positive patch test performed with correct technology. The inflammatory skin disease, allergic contact dermatitis may occur when contact sensitised individuals are exposed to the specific chemical.

Patch testing is an essential tool that used to established the diagnosis of allergic contact dermatitis. The patch test was introduced in 1896 by the Swiss dermatologist Jadahsson. In 1931 Sulzberger and Wise formally introduced patch testing to the American dermatologic community. It is a biological test where contact sensitisation is proved by re-exposing the individual on a 0.5-1cm² large skin area. This procedure involves placing a small amount of each of the suspect substances in a suitable 'vechicle', usually petrolatum, in a small aluminium or plastic cup attached to sticking plaster. The series of cups are then placed on the skin, usually on the back and left there for 48 hours.



When the plaster is removed the dermatologist will examine the skin to ascertain which substances have provoked a reaction. Patch testing is a highly skilled art. The dilution of the substance for testing must be enough to trigger an allergic reaction should the patient be sensitised, but not elicit an irritant one due to the length of contact and the occlusion. Intrepreting the various red inflammed blotches on the skin requires knowledge and experience. Patch testing will only indicate the presence of an existing sensitisation it is absolutely no use in determining who will or might become sensitised (Rietschel, 1995b).

This test system is a valuable practical tool for the dermatologist. Standardisation has taken place since its introduction, first by the Scandinavian group for standardisation of Patch Testing and later by the International Contact Dermatitis Research Group (ICDRG). The patch test is particularly valuable in ascertaining the cause of outbreaks of isolated cases of allergic contact dermatitis in an industry where workers are directly or indirectly exposed to many sensitising chemicals. Although a careful history and personal investigation of the patient's exposure to contactants often reduce the necessity for routine patch tests such procedures are often necessary to confirm a diagnosis of allergic contact dermatitis.

Patch testing may help in differentiating occupational and non-occupational dermatitis, particularly when a person with contact dermatitis is exposed to sensitisers not only at work but also at play or in persuit of hobbies. Properly performed patch testing may pinpoint the offending contactant quickly and efficiently whereas reliance on history and trial may prolong the dermatitis while the offending allergen is been persued, (Marks, 1997c).

Contact allergy can be quantified by the degree of positive patch tests, by patch testing with graded concentrations and by experimental use testing. The individual patch test is graded according to internationally agreed scoring system. A useful scoring system is that used by the North American Contact Dermatitis Group and is referred to as the patch test reading morphology codes. This system allows for a four point positive reading scale of +/-, +, ++, and +++. A +/- is a questionable reaction; with a definite positive reaction (erythema with edema or papules) being marked +; ++ indicates a strong edematous or vesicular reaction and +++ extreme spreading bullous or ulcerated responses (Plates 1-8). A negative reaction is coded with -. In addition, the system allows for an irritant morphology reading (IRR). This would be seen as a glazed or 'burned' appearance or pulsutor or purpuric reactions (*Appendix A*), (Marks, 1997c).



SUB-SECTION 2.10 PROGNOSIS OF OCCUPATIONAL DERMATITIS

2.10(a) Prognosis of Occupational Dermatitis Cases

Persistance of dermatitis after avoidance of primary contactants is well known. Prognosis in contact dermatitis refers to the outcome of dermatitis over time, with and without intervention. Understanding the prognosis of contact dermatitis enables the dermatologists to forecast probable outcome of the dermatitis to patients. Long term outcome of contact dermatitis especially occupational contact dermatitis, has important medicolegal implications. Prognosis of dermatitis helps dermatologists and employers to implement risk management of patients who are exposed to potential irritants and allergens, and hence plan preventative measures against contact dermatitis.

The prognosis of patients with contact dermatitis following secondary preventative measures refers to those patients who were confirmed to have contact dermatitis based on clinical findings and those who had received councelling on avoidance of contactants and on preventative measures. There is an association of prognosis outcome with risk factors including atopy, age, job change, contactants (irritants and allergens), and occupation.

2.10(b) Earlier reports on the prognosis of contact dermatitis

Earlier reports on the prognosis of contact dermatitis (especially occupational dermatitis) documented poor prognosis for total clearance of dermatitis. Burrows in

1972 reported that 79% of patients who were followed up over 10 - 13 years, still required treatment for their contact dermatitis.

A. Prognosis - Age

The age of onset of contact dermatitis does not appear to influence its prognosis as Burrows reported no significant difference in the prognosis between patients of < 40years old and those > 40 years old (dermatitis clearance rate was 15% versus 16% respectively) followed up over 10 - 13 years. Chromate allergy from cement is associated with poor prognosis and Burrows reported that only 8% of patients with cement dermatitis had clearance of dermatitis after 10 - 13 years follow-up.

B. Prognosis – Job Change

With regard to prognosis and job change earlier reports indicated that job change was not associated with significant improvement in the prognosis of occupational contact dermatitis. Burrows reported that only 20% of workers with dermatitis had stopped working when followed up over 10 - 13 years. Among these workers, only 18% had clearance of their dermatitis.

2.10(c) Recent reports on the prognosis of contact dermatitis

Recent reports have indicated that present prognosis is much better (Chia *et al.* 1991, Rosen *et al.* 1993, Nethercott *et al.* 1994). Reports between 1961 and 1972 (Burrows 1972, Skog *et al.* 1961) documented the prognosis for total clearance ranging from 8% to 33%. Reports after 1990 documented a clearance rate of about 70%. In Singapore, total clearance of dermatitis after one year of follow-up of patients with occupational dermatitis was 72% with slightly better prognosis for patients with allergic contact dermatitis (77%) than irritant contact dermitis (70%) (Chia *et al.* 1991).

In Sydney, Rosen *et al.* 1993, reported total clearance of dermatitis in 38% of patients with allergic contact dermatitis and 30% for patients with irritant contact dermatitis over a two to three year follow-up period. However, if patients who reported improvement were included as a favourable prognosis, then the rates were 74% and 68% respectively. In the United States, Nethercott *et al.* 1994, reported clearance in 63% of patients with mild eczema were included, the improvement rate was 81%. There were more workers with allergic contact dermatitis who reported that they were free of dermatitis than irritant dermatitis in the study.

A. Prognosis - Age and Sex

Chia *et al.* 1991, reported a slight improvement in prognosis in the older patients (>39 years old) with clearance rate of 85% compared with those of younger patients with a clearance rate of 65%. Nethercott *et al.* 1994, did not find any difference in prognosis among patients with contact dermatitis among age groups. Most reports showed that there was no significant difference in the prognosis of patients with contact dermatitis between males and females. In Singapore, the prognosis of male patients with allergic contact dermatitis (clearance rate of 90%) was significantly better than females (clearance rate of 50%). There was no significant difference in the clearance rate of 60%.



irritant contact dermatitis between male and female patients (Chia *et al.* 1991). Similarly, Nethercott *et al.* 1994, reported no significant difference in the prognosis of males and females with occupational contact dermatitis followed up over two years (with clearance rates of 60% for males and 75% for females). Chia *et al.* 1991, reported that the overall prognosis from occupational allergic contact dermatitis was good with 77% of their patients reporting total clearance of dermatitis. However, patients with metal allergy eg, nickel and cobalt, had a poor prognosis. 75% of patients with metal allergy had persistent dermatitis despite 'avoidance' of metals. The chronicity in contact allergy to these allergens is associated with their ubiquity and the fact that daily avoidance of these allergens is almost impossible.

In contrast to earlier reports (Burrows, 1972), the prognosis of patients with chromate dermatitis in Singapore was reported to be good. All five patients with chromate allergy had clearance of dermatitis upon avoidance of chromate (Chia *et al.* 1991).

B. Prognosis - Job Change

With regard to prognosis and job change, the prognosis for patients with contact dermatitis (irritant and allergic) who stopped being exposed to the contact irritants and allergens were slightly better than in those who continued. The overall clearance rates for patients who stopped were 73% compared to 69% for those who continued. The corresponding rates for allergic contact dermatitis were 71% for workers who stopped and 74% for those who continued, and for those with irritant contact dermatitis the rates were 74% and 68% respectively (Chia *et al.* 1991). In Sydney the prognosis was



significantly poorer in patients who continued to work (clearance rate 28%) compared to those who changed jobs (clearance rate 43%) (Rosen *et al.* 1993).

C. Prognosis - Atopy

A personal history of atopy also appeared to significantly affect the prognosis of patients with occupational contact dermatitis. Rosen *et al.* 1993, reported that the clearance rates in patients with atopy (30%) were significantly poorer than those in workers without atopy (41%). In contrast, Nethercott and Holness, 1994, did not report any significant difference in prognosis between atopics and nonatopic workers with occupational dermatitis. The clearance rates were 59% and 65% respectively.

Overall, there are numerous factors that influence the outcome of contact dermatitis. Patients with allergic contact dermatitis, (with the exception of chromate allergy), appear to have slighter better prognosis than patients with irritant contact dermatitis. In allergic contact dermatitis, a specific contact allergen can often be identified and patients are advised to avoid it. Risk factors include the present and past history of eczema and atopy. Job change tends to improve outcome but many continued to have dermatitis after changing (Chia *et al.* 1991, Burrows, 1972, Rosen *et al.* 1993 and Nethercott *et al.* 1994).



SUB-SECTION 2.11 CONTACT DERMATITIS AND WORKERS COMPENSATION

2.11 (a) Workers Compensation Laws

Worker's compensation laws were and important development of the industrial revolution. They provided a satisfactory means of handling occupational disabilities as the economy evolved from being predominantly agricultural to industrial. These laws were first enacted in Germany in 1884, followed by Great Britain in 1897, the United States in 1911 and Canada in 1915.

Before workers' compensation laws, the employee or the survivor according to common law principle sued the employer for damages that were due to employer negligence. This was a slow, costly, uncertain legal process that put the employee at a great disadvantage. Thus the essence of the workers' compensation laws that were enacted entitled the employee to medical treatment and compensation without regard to any fault and held that the employer should assume the cost of occupational disabilities. The workers' compensation statutes vary from country to country and various individuals become involved with these laws; the employer, the employee, insurance agents, attorneys, physicians and administrators of the law.



Workers' compensation laws should meet the following objectives;

- 1) Regardless of fault, provide occupationally induced illness or accident victims with a sure, prompt, reasonable income and medical benefits.
- 2) Reduce lengthy and costly court action.
- Relieve public and private financial drains, since workers compensation is paid for by the employer.
- 4) Encourage employer interest in safety and rehabilitation of the worker.
- 5) Promote investigation of the causes of accidents and disease, which will, it is hoped, reduce human suffering.

A principal element of workers' compensation is to show that the injury or illness has an occupational causation and, in addition, to determine to what extent and for how long the worker is disabled.

The physician plays an important role in workers' compensation and this includes:

- 1) Providing care for the injured or diseased workers
- 2) Evaluating the relationship to work
- 3) Determining the degree and the period of disability and
- Providing advice to the worker and industry about rehabilitation and peventative measures (Ross, 1994).



2.11(b) Work and Skin Conditions

Establishing a casual relationship between work and skin conditions is one of the areas that seem to cause most difficulty for the physician. Mathias (1989) very clearly outlined the criteria for establishing occupational causation and aggravation. He suggested seven criteria that should be present before the clinician conclude that the dermatitis was occupationally induced. Any criterion that was answered negatively suggested that the dermatitis may not be work related (Appendix B).

Together these criteria form a logical uniform basis for assessing the probability of causation from workplace exposures. Because workers' compensation law requires that there be only reasonable probability (more than 50% likelihood) of causation, the answer to at least four of the criteria should be "yes" before the clinician concludes that dermatitis probably was caused by a workplace exposure. If four or more of the criteria cannot be answered affirmatively, a conclusion of probable occupational causation may be difficult to justify without further investigation (Mathias, 1989).

2.11(c) Workers Compensation in Ireland and the United Kingdom

In Ireland and the United Kingdom, employers pay into the Industrial Injuries Scheme (Pay Related Social Insurance), and contribute until their retirement. The work injury benefits are part of the national security system of the country. Industrial diseases are tabulated in schedules due to cause. Doctors determine if the applicant is suffering from a defined illness and they determine the extent of the disability and the estimate how long it will last. A percentage rating is then made. The award that is decided on the



basis of medical recommendation is purely an impairment award and is independent of other losses of earnings which are covered by other parts of the Irish (British) Insurance program (Health and Safety Commission, 1997/98, Health and Safety Authority, 1997).

SUB-SECTION 2.12 DATA GATHERING ON OCCUPATIONAL SKIN DISEASE.

2.12(a) Methods used for data gathering

Contact dermatitis caused by allergens is the most important allergic skin disease related to occupation. A number of methods are utilised to correlate data from the incidence of skin diseases.

*Surveillance is the 'ongoing scrutiny, generally using methods distinguished by their practicability, uniformity, and frequently their rapidity rather than by complete accuracy. Its main purpose is to detect changes in trend or distribution in order to initiate investigative or control measures.

*A register is a 'file of data, collected for a specific public health purpose containing all (identifiable) cases of a particular disease or other health relevant condition, in a defined population such that the cases can be related to a population base'. With this information, incidence rates can be calculated.



*Epidemiology may be defined as 'the study of the distribution and determinants of health related states or events in specified populations, and the application of this study to control of health problems (Last, 1988).

2.12(b) Incidence and prevelance

Incidence and prevalence are measures of disease frequency. The frequency is expressed as a rate i.e. Number of cases per number of persons in the group within which the disease occurred. Cumulative incidence refers to a static group (e.g. People exposed during the whole period of observation), whereas incidence density refers to a dynamic group - new (exposed) participants may be added during the study period, while others may be deleted (i.e. no longer exposed). The prevalence rate is a measurement of the numbers of existing disease in a population at one point in time (Kramer, 1988).

There are various reasons to favour prevalence rates rather than incidence rates in studies of skin allergy. Allergic contact dermatitis is a non-fatal, chronic disease. Even with low incidence rates the prevalence may be high the new cases being added to an already high basic rate. Incidence rates can be calculated only for the disease, not for sensitisations, since the exposure which led to sensitisation may be different from the exposure, which led to elicitation. Prevalance rates can be applied both to sensitisation rate to a certain allergen and to the manifest disease, allergic contact dermatitis.



2.12(c) Passive data generation

Passive data generation occurs when people consult a physician because they suffer from contact dermatitis. Clinical examination and patch testing would provide the diagnosis (allergic contact dermatitis) and the cause of the illness (sensitisation to an allergen). Thus, passive data generation is the basis of morbidity statistics. Registers of occupational skin diseases are kept in several European countries and in the United Kingdom (Mathias, 1988, O' Malley, 1988, Roche, 1993). Most of the registers contain data on all type of skin disease and no distinction is made with regard to the type of disease (irritant or contact dermatitis).

The most important data source on occupational allergic skin diseases is the statistics generated by dermatologists. Dermatologists record the patient's history, (e.g. occupation), examines the case clinically (including constitutional risk factors) and uses patch testing to make the allergic diagnosis. Until recently, this data was used only sporadically for evaluation purposes. In the United Kingdom, dermatologists from sixteen centres started a pilot study in order to assess the viability of maintaining a surveillance register of occupational dermatitis through a simple card reporting system. In the first twelve months, this group has identified one thousand, four hundred and sixty six new cases of occupational dermatitis.

Since February, 1993, consultant dermatologists in the United Kingdom have been reporting to Epi-Derm the surveillance scheme for work related skin disorders. Occupational physicians reported to the same scheme from May 1994 to December



1995. Since January 1996, occupational physicians have reported skin disease along with other types of occupational disease to their own scheme OPRA (Occupational Physicians Reporting Activity).

In recent years, there have been several thousand cases per year leading to some level of specialist intervention. An estimated three thousand, seven hundred cases were seen by dermatologists and occupational physicians in the EPIDERM and OPRA surveillance schemes, approximately 80% of which were contact dermatitis (Beck, 1992).

The Health and Safety Commission, 1997/1998, reported that there is some indication of a downward trend in the number of cases of occupational dermatitis over the last five years from the data on disablement benefit cases, although the EPIDERM and OPRA data indicate a slightly increasing trend over the last four years.

2.12(d) Occupations most commonly associated with Contact Dermatitis

The occupations estimated to be the most commonly associated with contact dermatitis in EPIDERM / OPRA seen by dermatologists are other occupations in sales and services (9.0%), hairdressers and beauticians (8.4%) and health associates (7.8%). The most common occupations seen by occupational physicians are chemical operatives (10.4%) and health associates (9.3%). Those with the highest estimated rates of contact dermatitis among occupations in mining and manufacturing (8.9%) and hairdressers and beauticians (8.3%) (Health and Safety Commission, 1997/98).



2.12(e) Data gathering for occupational skin diseases in Ireland

In Ireland up to 1995, the only source of data for occupational diseases was the Department of Social Welfare. During 1995, the basis of data collection was broadened to include hospital pathologists, dermatologists, chest physicians, pesticide adverse reaction reports, National Poisons Centre, National Cancer Registry and the Department of Health. Under reporting of occupational diseases is still a problem.

The Health and Safety Authority (H.S.A) compile the statistics for occupational diseases. The H.S.A annual report 1997 presented data relating to occupational dermatitis cases reported from 1994-1996 as 44, 63 and 38 respectively. The 1997 annual report is the most current report available however, it does not distinguish between allergic/irritant dermatitis . With regard to the collection of data for occupational skin diseases, the H.S.A are currently gathering information from the Department of Social Welfare Occupational Injury Benefit section and direct from the Register of Occupational Dermatological Disease (Appendix C). A number of dermatologists in Ireland take part in this surveillance similar to the EPIDERM survey conducted in the United Kingdom (Health and Safety Authority, 1997).

2.12 (f) Statistical Information on Social Welfare services

In Ireland there are approximately fifteen thousand claims per year. In the most recent annual report issued by the Department of Social Welfare there were 14,774 injury benefit claims of which 11,169 were allowed (of those two hundred were prescribed diseases). Total Social Welfare expenditure in 1997 was £4,524 million, which represented 32.3% of net current government expenditure. The main areas of expenditure by programme group were old age (22.88%), widows, widowers and one parent families (17.4%), child related payments (9.63%), illness, disability and caring (3.0%), unemployment and employment supports (23.15%). In 1997 there was an expenditure of £624,185 on social welfare for illness, disability and caring. Of this £179,919 was spent on disability benefit £6,740 on injury benefit and £38,287 on disablement benefit.

The Social Welfare services deal with approximately 15,000 claims per year (10,000 awarded) for injury benefit, 3,000 per year (2,000 awarded) for disablement benefit and 12,000 per year (9,000 awarded) for disability. In 1997, there were 14,774 claims received and 11,169 were awarded.

The conditions for benefit to qualify include the following:-

- You must be suffering from one of the prescribed diseases. A prescribed occupational disease is one of the diseases listed that has developed due to the nature of your employment.
- You must have been employed after May 1st 1967 in one of the occupations prescribed in relation to that disease.
- The disease must be due to the nature of the occupation.

Injury benefit is a weekly payment made during periods of incapacity for work as a result of an injury received or a disease contracted at work. Benefit is payable for a maximum of twenty-six weeks. The Disablement benefit is payable as a weekly or four-weekly pension; this is normally payable after injury benefit has ceased to be payable.

SUB-SECTION 2.13 SKIN MANAGEMENT

2.13(a) Occupational skin disease and its prevention

The optimal strategy in dealing with occupational skin disease is its prevention. This is a multidisciplinary endeavor that requires planning by the employer, employee, government officials and healthcare personnel to develop preventative measures. The responsibility for prevention of occupational skin diseases rests on a number of individuals including toxicologists, chemical and safety engineers, manufacturing management, industrial hygienists, workers, government regulators and scientists, and healthcare providers. It is the integration and co-operation among these individuals that prevent occupational skin disease. The ultimate aim of any skin management system should be to prevent all contact between any substance capable of causing damage.

According to Packham (1998), an "effective skin management is a system to ensure that so far as practicable, the workplace is intrinsically safe as regards damage to health through skin exposure. Where this is not achievable, the system will incorporate appropriate provisions for personal protective equipment to achieve adequate control of exposure. The system is not static, but must reflect actual workplace conditions and adapt to changes and developments in our knowledge of how the skin reacts to substances".

2.13(b) Elements of a skin management system

Packham (1998) offers a structured approach to take into account the different elements of a skin management system.

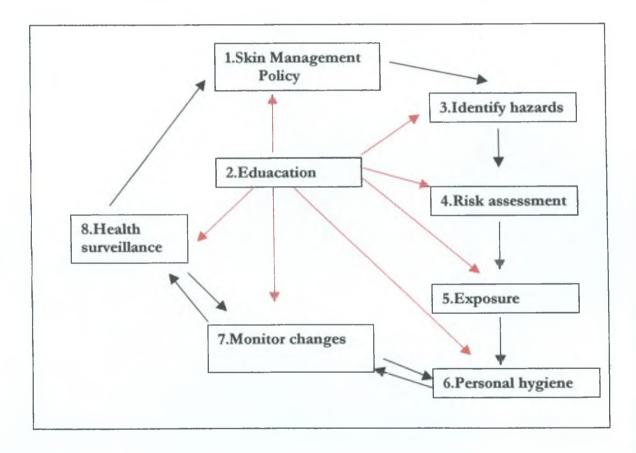


Fig.4. Elements of a Skin Management System (Adapted from Packham, C.L, Essentials of Occupational Skin Management, 1998).



This system is designed to ensure that the risk of damage to health through skin exposure is kept to an absolute minimum and that any health problems that may arise are identified at the earliest possible stage so that remedial action can be taken.

1) Management Policy

According to Packham (1998), the policy should provide a clear and comprehensive statement to include information relating to what the employer is trying to achieve and how this is to be done. The policy sets down the objectives that are set by management and the responsibilities and duties of the employer, employee and any one else involved. The skin management policy may include the following information;

- The purpose of the policy.
- The responsibilities of the company.
- The responsibilities of the employees.
- The methods to be used for the prevention of occupational skin problems.
- Provisions relating to training and education.
- Information and labelling for products.
- Working practice
- Health surveillance.
- The procedures to be used for reporting.

2) Education

Worker education is an integral part of occupational skin disease prevention. The worker should be provided with information on the toxic nature of chemicals in the



workplace, instructions concerning the use of protective measures, and procedures to follow in the case of accidental exposure. According to Packham (1998), a skin management system should involve '*comprehensive education and training which is relevant, accurate and applicable*', This means that the material contained in the educational programme must address the needs of that particular operation. Both management and workers must have the appropriate knowledge both about the processes and substances present in the workplace and also an understanding of the skin, its functions and the problems which can arise from the above.

3) Hazardous Material Identification.

The recognition of potentially hazardous chemicals should be accomplished by toxicologic testing before introduction into the workplace. For allergens and irritant, risk assessment testing can define the inherent irritant and allergenic properties of the chemical. This information should be found on the material safety data sheets and should be reviewed before new materials or processes are used.

4) Risk Assessment

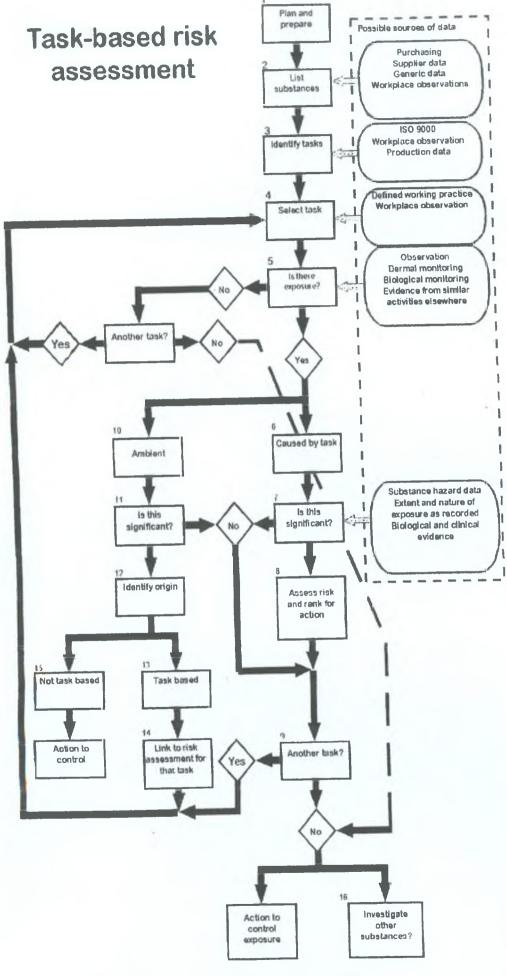
Techniques for the assessment of risk to health through skin contact are poor and are mostly highly subjective. According to Packham (1998a), 'Assessment of the risk to health through skin exposure is complex. There are few technical aids to help us and no standards such as exposure limits which can act as a guide moreover, the data with which we have to work may be inaccurate and/or incomplete. A structural approach will enable us to rank the many risks in the average workplace in some order of priority



so that we can deal with the most serious potential risk first. Risk assessment is an ongoing process".

Packham (1998a), approaches the measurement of risk assessment of skin disease by dividing the way in which exposure occurs into two groups, "ambient" and "task based". A task can be defined as a discrete action or set of actions with a clear start and finish which can be described in such a way that it can be repeated with a considerable degree of accuracy. Task-based exposure, therefore is exposure that is caused directly during the execution of the task. Ambient exposure is all other forms of exposure (ie the presence in the workplace in such a way that exposure of the worker is occurring not specifically associated with his actual task).

Since virtually all ambient exposure will be as a result of some activity being carried out within the workplace, therefore it is essentially task based. Packham (1998a), provides a strategy for a task approach to risk assessment (see pg73). The flow chart illustrates one possible structured approach to risk assessment based on the individual task, but which also takes into account of ambient exposure, where this can be identified.





5) Exposure Control

The employer has several potential means of preventing occupational dermatoses, including environmental control, good housekeeping, warnings on hazardous material, and education of the workers. Ideally, exposure to hazardous chemicals can be eliminated by the engineering of closed systems that allow the manufacturing process to proceed without exposing the worker to harmful chemicals. Engineering systems such as automated samplers, computerised manufacturing, and robotic packaging may be implemented. Although this protects the line worker, consideration must also be given to maintenance personnel who may have exposure to hazardous chemicals. The goal of these engineering controls is to minimise cutaneous contamination.

Substances may contaminate surfaces directly or indirectly. Direct contamination occurs where the substance is placed on the surface as part of the task. The ambient contact follows when another worker uses the same surface for some other task. Indirect contamination may occur, for example, from deposition of airborne aerosol or dust or from condensation of vapour onto a cold surface. Detection of such contamination may be simply a matter of observation however, where such contamination is suspected a simple wipe test may be required.

The worker is critical in hazard control, since total avoidance of cutaneous contact with hazardous materials in many occupations cannot be accomplished by engineering controls alone. This requires the worker to use personal protection. The protective equipment/clothing must fulfill requirements dictated by the type of physical and chemical exposure and type of work been carried out.

2.13(c) The use of protective gloves

Because hand dermatitis is the most common site of occupational contact dermatitis, gloves are the most useful protective gear. Because a large number of gloves are available, knowledge of the physical and biological hazards, and the job that is to be performed is required. Degradation and permeation are two types of chemical resistance properties that should be considered in the selection of gloves. The deterioration of the glove's physical properties can cause the glove to crack, tear easily or dissolve so that large amounts of hazardous material come in contact with the skin.

Once it is determined that the glove is not degraded by a hazardous chemical, the second consideration is how much of the chemical diffuses through the glove. This is measured in the testing laboratory by breakthrough time and the steady-state permeation rate. No single glove is protective from all possible chemicals. Packham (1998b), introduces a simplistic glove selection chart (Appendix D).

It should be remembered that all glove materials are to some extent permeable to chemicals and that there is no universal protective material suitable for all possible chemicals (Packham 1998b).

2.13(d) Skin Allergy to natural rubber latex

Occasionally, an allergy to a component of the glove or irritation from the glove itself, can be the cause of contact dermatitis, not the hazardous chemical for which glove protection was intended.

Of increasing significance is the problem of contact dermatitis caused by the proteins contained in natural rubber. This is of particular concern in those occupations where gloves are worn for extended periods of time, such as health care workers, hospitals, pharmaceutical manufacture, electronics. Natural rubber latex is a complex blend of different chemicals and some of the chemicals present in latex gloves are responsible for the Type IV skin reaction, allergic contact dermatitis (Hunt *et al.* 1995).

The National Institute for Occupational Safety and Health (NIOSH) recently published an article on latex gloves to promote the prevention of allergic reactions to natural rubber. Recent reports in the scientific literature indicate that from 1% to 6% of the general population and 8% to 12% of regularly exposed healthcare workers are sensitised to latex (Anon, 1997b).



SECTION 3. RESULTS

SUB-SECTION 3.1 RESULTS OF INDUSTRIAL SURVEY

3.1(a) Industries surveyed

A survey was carried out in order to determine the use and control of substances in Irish industry which would be classified as 'Skin Sensitisers'. The companies were selected from the Industrial Development listing of industries in Ireland. One hundred and twenty industries were surveyed. Of the industries surveyed 63 replies were returned. The respondence came mainly from the pharmaceutical (41%) and chemical (35%) industries. Other industries (24%) involved in the survey included, electroplating, electronic, healthcare and medical device manufacturers. The contents of this survey are included in a matrix (see Appendix F).

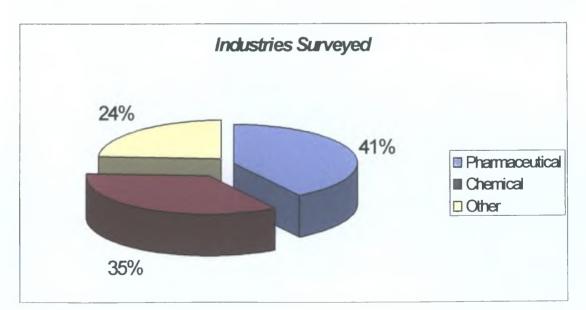


Fig. 5 Industries involved in the survey

3.1(b) The number of employees within the industries surveyed

Table No.2

% of industries surveyed
23%
41%
14%
21%

As can be seen from the above table the largest portion of the industries surveyed employ >50 people. 97% of the industries involved in the survey had Material Safety Data Sheets available to all employees.

3.1(c) Use of substances classified as skin sensitisers

Results from the survey reveal that 49% of industries use substances which are classified as 'Skin Sensitisers'.

Some key	y findings from the surveys conducted
*49% of i	industries surveyed use substances classified as 'Skin Sensitisers'.
*Only 2%	6 of the industries surveyed have documented 'Risk Assessments' relating to
the use of	f these substances in the workplace.
	f these substances in the workplace. f the industries surveyed have 'Skin Management Systems' or 'Skin

3.1(d) The reported cases of 'Contact Dermatitis among Irish Industry

The results from the surveys conducted revealed that 49% of the industries surveyed reported cases of contact dermatitis within their workplace. 51% reported no cases of contact dermatitis among their workforce. Of the 49% that reported cases of dermatitis within their company 2% revealed that the contact dermatitis was as a result of allergy to natural rubber latex.

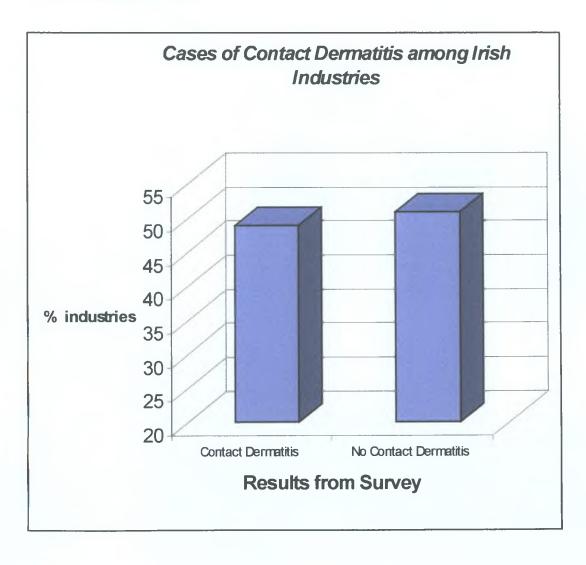


Fig.6 Cases of Contact Dermatitis among Irish Industries.



3.1(e) Control measures for the prevention of 'Occupational Skin Diseases'

There are a number of provisions relating to the protection of workers from exposure to hazardous substances in the workplace. Among the industries surveyed there were a number of procedures for dealing with exposure to potential skin allergens and irritants.

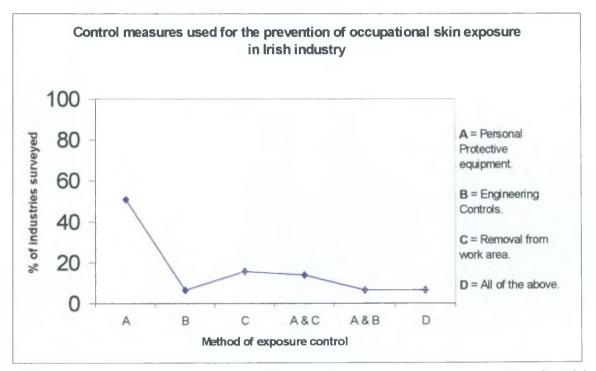


Fig.7 Control measures for the prevention of occupational skin exposure in Irish Industry.

From the industries surveyed it is clear that there is a heavy reliance on the use of personal protective equipment for the prevention and/or the control of occupational skin diseases. There is less emphasis on engineering controls which rank much higher in the overall hierarchy of control from an occupational health perspective. Administrative



controls by way of reducing the time spent in an area and/or the removal of a sensitised person from the area where there is the potential of exposure to the allergen is also rated above engineering controls for the prevention of occupational skin diseases within the context of the results of this survey.

3.1(f) Specific characteristics of substances classified as 'Skin Sensitisers' which make exposure control difficult.

Results from the survey revealed that although 80% of those industries surveyed found no specific characteristics of the substances that made exposure to them difficult to control, 20% reported some difficulties. The reported characteristics included substances present as powders, aerosols and fumes.

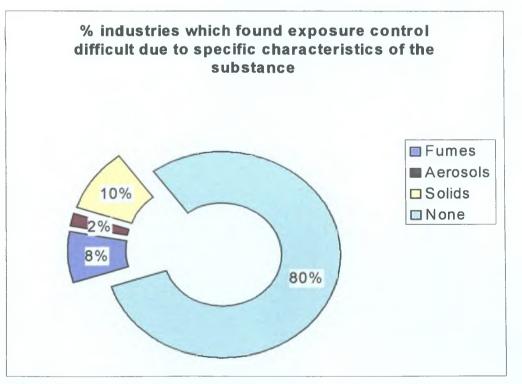


Fig.8 % industries which found exposure control difficult due to specific characteristics of the substance.



SECTION 4

DISCUSSION



SECTION 4. DISCUSSION

In Ireland, at present, the control on the importation of substances is largely on the onus of the Revenue Commissioners who operate a tariff code system that groups chemicals into broad categories. It is not possible at present to identify individual chemicals imported and hence the information relating to the volume of substances classified as "Skin Sensitisers" imported into Ireland on an annual basis is currently unavailable.

In addition to this, a current list of substances classified by the European Commission is not accessible. Annex 1 of the EU Directive 67/548/EEC hasn't been updated since 1994 and is due for release within the next 2 years.

In an effort to determine the extent of the use and control of substances in Irish Industry which would be classified as 'Skin Sensitisers' a detailed survey was developed and sent to 120 industries within the Republic of Ireland (Section 3).

The results of this survey revealed the widespread use of substances which are potential skin sensitisers. All of the industries surveyed are involved in manufacturing, with more than half employing >50 people. The industries surveyed include, pharmaceutical, chemical, electroplating, electronic, healthcare and medical device manufacturers. The bulk of the companies involved in the survey were from the pharmaceutical and chemical sector.



Almost half of the companies surveyed experienced cases of contact dermatitis among their workforce. Skin disease caused by a substance in the workplace is the most frequently encountered occupational illness and the results of the survey reitterate this fact. The statistical data reported in the Health and Safety Authority Annaul Report 1997 noted that the highest number of cases of occupational disease were due to occupational dermatitis.

Further results of the survey revealed that there is a heavy reliance on personal protective equipment (PPE) to control exposure to potential skin allergens/irritants. Greater than half of the respondents reported reliance on the use of PPE to reduce or eliminate exposure to potential allergens. The hierarchy of control for occupational hygiene does not begin with personal protective equipment. Where a substance represents a potential to cause injury or damage to health, then the operation involving the substance should be investigated to determine the possibility of eliminating or substituting the substance. If this option is not feasible then adequate engineering or administrative controls should be put in place to reduce or eliminate exposure to the substance.

The use of personal equipment should only be addressed when the above approaches have been investigated. It is necessary to understand that while personal protective equipment offers protection to the individual carrying out a specific task, it offers no protection to any other person working in the area who also may be exposed to the substance although they may no be in direct contact with it. In order to comprehend



the hazards of various chemicals such as those classified as 'skin sensitisers' one requires knowledge of the nature of the substance, its mode of action (i.e. sensitisation and elicitation of allergic reactions) and if and how it penetrates the skin. For this purpose education and training is essential.

Occupational skin diseases can occur in workers of all ages, in any work setting and cause a great deal of illness, personal misery and reduced productivity. Although the frequency of occupational skin disease often parallels the level of hygiene practiced by employers/employees, occupational skin diseases are largely preventable.

A variety of industrial chemicals are potential allergens. The incidence of allergic contact dermatitis varies depending on the nature of the materials handled and predisposing factors. Cross-sensitivity is an important phenomenon in which workers sensitised to one chemical will also react to one or more closely related chemicals. The cutaneous absorption rate of some organic compounds rises when temperature or perspiration increases. The absorption of liquid organic compounds may follow surface contamination of the skin or clothes while for other compounds it may occur directly from the vapour phase, in which case the rate of absorption is proportional to the air concentration of the skin surface followed by absorption through the skin.

Of the industries surveyed 20% reported difficulty in exposure control as a result of specific characteristics of the substances. The majority reported difficulties with



powders while fumes and aerosols were also reported as problematic. A number of industries are developing potent substances that require processing in a powder or other potentially respirable form. These substances may be active if inhaled or if they come in contact with the skin or eyes and can therefore pose a significant hazard to workers.

The need for determining worker exposure to hazardous substances in the workplace environment demands the availability of appropriate tested sampling methods. A critical part of the protocol for testing industrial methods is the preparation of controlled test atmospheres of the specific materials over the concerned range of interest. While the technology probably exists for developing validated sampling methods in each company, the cost for development of low concentration generation techniques are high and most companies may not have the in-house staff, facilities and equipment for such activities.

For technical reasons there are still no skin exposure limits to guide the employer nor techniques to measure skin exposure levels. As a result assessing whether a workplace is safe for skin exposure is still highly subjective and dependent upon the expertise of the observer. Substances, which cause sensitisation by skin contact, are not specifically identified in the Health and Safety at Work Act, 1997 Code of Practice (Appendix G). Further work is required to evaluate worker exposure to substances (allergens) which cause skin diseases. In order to do this the employer and the industrial hygienist need to have a standardised approach to assessing the exposure risks. They also require further information regarding the implementation of Skin Management Policies within companies, and the current listing of substances classified as skin sensitisers should be



available. It is on the onus of the Health and Safety Authority of Ireland to address the problems of occupational skin diseases and to develop a more structured approach to its prevention. Employers require guidelines from the relevant authority in order to implement policies within their industry.

The loss of productivity resulting from skin problems, even though this may not be apparent in terms of sickness absence or compensation claims, is a real cost to the business in question and the government. The examination of government expenditure for disability and disablement benefit and the data available from Social Welfare payments for occupational dermatitis, reinforces the requirement for further improvements to the protection of workers exposed to substances that have the potential to cause skin disease.

Evaluation of the casual relationship, degree of disability and advice to the worker and industry is often overlooked or poorly done because of ignorance or lack of physician time. Adequate evaluation of the worker requires an extended office visit to obtain a detailed occupational history. If this is done dealing with workers compensation becomes relatively straightforward for the physician. The role of the physician is to determine the amount of alteration of health status i.e. impairment. Impairment is often blurred with determining the alteration in the patient's capacity to meet personal, social, or occupational demands, i.e. disability. Disability is determined by professional disability rating personnel.



Detailed statistics on the actual incidence of occupational skin disease is hard to find in Ireland. For some years (until 1995) occupational dermatitis was not reported. Today with the involvement of dermatologists and physicians in the surveillance reports, further data should become more readily available.

There is no way of ensuring absolutely the prevention of occupational skin disease. What we are concerned with is the probability that the interaction between the skin and the working environment will result in damage to health. There is virtually no occupation where there is not some risk of skin disease occurring due to conditions in the workplace. However, certain occupations place workers at particularly high risk. An awareness of these risks can be of considerable benefit in both assessing the advisability of an individual taking up a particular operation and in creating an effective skin management system.

A skin management system would provide a structured approach, which would take into account different elements such as education and training, risk assessment, exposure assessment and monitoring, by integrating them into a single effective system. The system should not only ensure that the risk of damage to health through skin exposure is kept to an absolute minimum but also that any health problem which does arise is identified at the earliest possible stage so that remedial action can be taken. Skin management is about ensuring that the interaction between the skin and the working environment does not cause damage to the skin nor permit toxic chemicals to penetrate the skin and damage internal organs.



SECTION 5

CONCLUSION



SECTION 5. CONCLUSION

There is a widespread use of substances in Irish industry which are classified as 'Skin Sensitisers'. There are very limited procedures in place for the prevention of occupational skin disease.

Further attention to detail is required to put in place a structured approach to the management of the exposure of workers to substances which have the potential to cause skin disease. The fact that the largest number of reported cases of occupational disease is occupational dermatitis it is clear that immediate action is needed to reduce the number of cases. In order to do this, there needs to be some guidelines for employers to follow by way of a standard protocol, detailing the criteria to be followed for assessing the use of substances which have the potential to cause skin disease.

Due to the lack of information available to employers, such as criteria for carrying out a detailed risk assessment for skin exposure along with an up to date list of substances classified as R43 (skin sensitisers), exposure limits and techniques to measure skin exposure, exposure control is difficult.

Until such time as this type of information and documentation become available to employers it is necessary for industries to develop in-house policies on skin management. A systematic approach involving risk assessment surrounding individual operations involving substances which have the potential to cause skin disease, should be investigated, implemented and documented. The onus is on the Health and Safety Authority of Ireland to address the issues relating to occupational skin diseases and to develop a structured approach to its prevention, control and management. It is to the interest of employees, employers and the governing body of Ireland to reduce the incidents of skin diseases in the workplace.



REFERENCES



REFERENCES

Ancoma. AA; Occupational acne. Occup. Med. State Art Rev 1:229-243, 1986.

Anon. (1975a). Industrial Dermatitis. Part 1: The National Safety News, 1975, 112, pp. 59-64.

Anon. (1997a). Consensus reached on criteria for clarifying skin and airway sensitising substances in the work and general environments. –Scandinavian Journal of Work and Environmental Health, 1997, Vol.23, No. 3, pp.157.

Anon. (1975b). Sensitisation. Part III: The National Safety News, 1975, 112 pp.65-68.

Anon. (1997b) Preventing Allergic Reactions to Natural rubber latex in the workplace.DHHS (NIOSH) Publication No. 97-135, 1997.

Beck, M.H. Occupational dermatoses surveillance in the United Kingdom. British Journal of Dermatology, 127(540): 16-17 (1992).

Bowman, W.C. and Rand, M.J., Textbook of pharmacology, 2nd edition, Blackwells Scientific Publishers, Oxford.

Burrows, D: Prognosis in industrial dermatitis.Br J Dermatol 1972; 87:145-148.



Chia. S.E., GOH C.L.: Prognosis of occupational dermatitis in Singapore worker. Am J Contact Dermatitis 1991; 2: 105-109.

Cohen. S.R.: Risk factors in occupational skin disease. In Maibach H.I., Gellin G.G. (eds) Occuational and Industrial Dermatology. Chicago, Year Book Medical Publishers, 1982.

Cooper, E.R. (1985) Vehicle effects on skin penetration in percutaneous absorption; mechanisms –methodology – drug delivery (edited by Bronaugh, R.L. and Maibach, H.I.), pp.525-530. Marcel Dekker, New York.

De Groot, A.C. Patch Testing, Test concentrations and Vehicles for 3700 chemicals. 2nd ed. Amsterdam, London, New York, Tokyo, Elsevier, 1994.

Department of Social, Community and family affairs. Statistical information on Social Welfare Services. 1997.

Dutkiewicz, T. and Piotrowski, J. (1961) *Experimental investigations the quantitative* estimation of aniline absorption in man. Pure and Appl. Chem. 3, 319-323.

EPA (1992) Dermal exposure assessment: principles and applications. Interim report EPA/600/8-91/001B. Office of Research and Development, Environmental Protection Agency, Washington, DC, pp.5.21-5.32 and pp.5.53-5.64.

Engstrom, K., Husman, K. and Riihimaki, V. (1977) Percutaneous absorption of mxylene in man. Int. Archs occup. Environ. Hith. 39, 181-189.

EUROPEAN COUNCIL. Council Directive 67/548/EEC of June 1967 on the approximation of laws, Regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances. Official Journal of the European Communities 196, 16.8. 1967, p.1.

EUROPEAN COUNCIL. Council directive 92/32/EEC amending for the seventh time Directive 67/548/EEC on the approximation of the laws, regulations, and administration provisions relating to the classification, packaging and labelling of dangerous substances. Official Journal of the European Communities L248 of the 30th September 1996.

EUROPEAN COMMISSION. Communication Pursuant to Article 2 of Commission Decision 85/71/EEC of 21st December 1984 concerning the list of chemical substances notified pursuant to Council Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances, Official Journal of the Eurpoean Communities C361 17.12.1994.

EUROPEAN COMMISSION. Commission Communication pursuant to Article 13 of the laws, regulations and administrative provisions relating to the classification



packaging and labelling of dangerous substances, as amended by Directive 79/831/EEC Official Journal of the European Communities C146A, 15.6 1990, p.1.

EUROPEAN COMMISSION. Commission Directive 93/67/EEC of 20th July 1993 laying down the principles for assessment of risks to man and the environment of substances notified in accordance with Council Directive 67/548/EEC Official Journal of the European Communities L227, 8.9.1993, p.9.

Fischer AA : Chromate dermatitis and cement burns. Contact Dermatitis. edition 3. Philadelphia, Lea and Febieger, 1986b.

Fischer AA: The role of age, sex and color of skin in contact dermatitis. edition 3. Philadelphia, Lea and Fiebiger, 1986a.

Fischer T. : Prevention of irritant dermatitis. Occup. Med. State Art Rev 1:335-342,1986.

Fiserova-Bergerova, V. (1990a). Application of toxicokinetic models to establish biological exposure indicators. Ann. Occup, Hyg. 34, 639-651.

Fiserova-Bergerova, V. Pierce, J.T and Droz, P.O.(1990b). Dermal absorption potential of industrial chemicals, Criteria for skin notation. Am. J. Ind Med. 17,617-635.

Fleck, J. and Sedivec, V. (1978). The absorption, metabolism and excreation of furfural in man. Int. Archs. Occup. Environ. Hlth. 41, 159-168.

Fukuyama K., Gellin G.A., Nishimura M., et al., Occupational Leukoderma: Morphological and biological studies of 4- tertiarybutyl catechol depigmentation, Kligmon A.M., Leyden J.J. New York, 1982, Grune and Stratton Inc., pp.135-155.

Guy, R.H., Hadgraft, J. and Maibach, H.I. (1985) Percutaneous absorption in man; a kinetic approach. Toxicol. Appl. Pharmac.78, 123-129.

Health and Safety Commission. Health and Safety Statistics 1997/98.

Health and Safety Authority. Health and Safety Statistics, Annual Report 1997.

Hunt, L.W, Fransway, A.F., Reed, C.E., Miller, L.K., Jones, R.T., Swanson, B.S., Yunginger, J.W. An epidemic of Occupational Allergy to Latex involving health care workers. JOEM Vol. 37, No.10, pp1204-1209, 1995.

Kao, J., Patterson, F.K. and Hall, J. (1985) Skin penetration and metabolism of topically applied chemicals in six mammalian species including man: an in vitro study with benzo/a/pyrene and testerone. Toxicol. appl. Pharma. 81, 502-516.

Kelly, K.J, Sussman, G, Fink, J. N. (1996). Stop the sensitisation. J Allergy Clin. Immunol. 98(5), 857-858.

Kramer, M.S. Clinical Epidemiology and Biostatistic. Berlin-Heidelberg, New York, Springer, 1988.

Lammintausta K., Kalino K.; Atopy and Land dermatitis in hospital wet work. Contact Dermatitis 7: 301-308,1981.

Last, J.M., ed. A Dictionary of Epidemiology, 2nd ed. New York–Oxford– Toronto, Oxford University Press, 1988.

Lucas, J.B. Common industrial dermatoses. Cutis, 13: 535-543 (1974).

Marks J.G., Jnr, Deleo V.A. Contact and Occupational Dermatology 2nd ed. p.1 (1997a.)

Marks J.G., Jnr, Deleo V.A. Contact and Occupational Dermatology 2nd ed. pp277-279 (1997b).

Marks, J.G., Jnr, Deleo, V.A. Contact and Occupational Dermalalogy 2nd edition, 1997 pp 32-54. (1997c).

Mathias, C.G.T and Morrison, J.H.: Occupational Skin diseases, United States: Results from the Bureau of labour Statistics Annual Survey of Occupational Injuries and Illnesses, 1973 through 1984, Archives of Dermatology, 124:1519-1524 (1988).

Mathias C.G.T.: Contact Dermatitis and workers' compensation: criteria for establishing occupational causation and aggravation. J.Am. Acad. Dermatol 20:842-848, (1989).

National Authority for Occupational Safety and Health, 1997 Code of Practice for the Safety, Health and Welfare at work (Chemical Agents) Regulations, (1994).

Nethercatt J., Holness L. : Disease outcome in workers with occupational skin disease. J. Am. Acad. Dermatol. (1994); 30:569-574.

Olishifski, J.S. The Skin. Fundamentals of Industrial Hygiene, pp. 47-57, (1988).

Orris, L., Tesser M.; Dermatoses due to water, soaps, detergents and solvents. In Maibach H., Gellin G. (eds): Occupational and Industrial Dermatology, Chicago, Year Book Publishers, 1982.

Osbourne, D.W. (1986) Computational methods for predicting skin permeability. In Pharmaceutical Manufacturing Technical Update, pp.41-47. Upjohn Co., Kalamazoo, Michigan.



Overgaard Olsen L., Jemec G.B.E.; The influence of water, glycerin, paraffin oil and ethanol on skin mechanics; Acta Dermato Venerologica, 1993, 74, 404-406.

O'Malley, M. et al. Surveillance of occupational skin disease using the supplementary data system. American Journal of Industrial Medicine, 13:291-299 (1988).

Organisation for Econmic Co-operation and Development. Classification systems on sensitising substances in OECD countries- a review- Draft 1996 01 26-OECD Room Document 4 for the second meeting of the advisory group on harmonisation of classification and labelling systems, Paris 29-30 January (1996).

Packham, C.L. Essentials of Occupational Skin Management. A practical guide to the creation and maintenance of an effective skin management system. pp.72-81 (1998c).

Packham, C.L. Essentials of Occupational Skin Management. A practical guide to the creation and maintenance of an effective skin management system. pp. 325-348(1998a).

Packham, C.L. Essentials of Occupational Skin Management. A practical guide to the creation and maintenance of an effective skin management system. pp.217-261.(1998b).



Packham, C.L. Essentials of Occupational Skin Management. A practical guide to the creation and maintenance of an effective skin management system. pp.53 (1998d).

Rajka G.: Atopic Dermatitis, London, WB Saunders, 1975.

Rietschel, R.L., Fowler, J.F., Jnr.; Fischer's Contact Dermatitis, 4th edition, pp.1-9,1995a.

Rietschel, R.L., Fowler, J.F., Jnr, Fischer's Contact Dermatitis, 4th edition, pp.11-13,1995b.

Rietschel, R.L., Fowler, J.F., Jnr; Fischer's Contact Dermatitis, 4th edition, pp. 551-560, 1995.

Riihimaki, V., and Pfaffli, P. (1978), Percutaneous absorption of solvent vapours in man. Scand. J. Wk. Environ. Hlth. 4, pp.73-85.

Roche, L.M. Use of employer illness reports for occupational disease surveillance among public employees in New Jersey. Journal of Occupational and Environmental Medicine 35(b):581-586, (1993).

Rosen R.H., Freeman S.: Prognosis of occupational contact dermatitis in New South Wales, Australia. Contact Dermatitis 1993; 29: 88-93.

Ross J.B.; Workers compensation for Skin Disease. Occup. Med. 9:25-36,1994.

Scheuplein, R.J. and Bronaugh, R.L., (1983) *Percutaneous absorption*. In Biochemistry and Physiology of the Skin (edited by Goldsmith L.A.), Vol II pp.1255-1295. Oxford University Press, Oxford.

Sedivec, V., Mraz, M. and Flek, J., (1981) Biological monitoring of persons exposed to methanol vapors. Int. Archs. Occup. Environ. Hlth. 48, 257-271.

Shmunes, E.; Predisposing factors in occupational skin diseases, Dermatol. Clin., 1988, 6:7-9.

Skellchock L.E., Contact Dermatitis is a Costly Skin Disease. Skin Care Today for the Health Professional, Vol 1. Number 1., 1995.

Skog E., Tottie M. : Occupational eczema causing disablement. Acta Derm Venerol., 1961; 41:205-212.

Sulzberger M.B., Wise F.: The contact or patch test in dermatology. Arch Dermatol. Syph. 23:519-531, 1931.



Taylor, J.S. Occupational Dermatoses. Clinical Medicine for the Occupational Physician ed. by Alderman, M.H. and Hanley, M.J., 1982, pp.309-311.

Taylor, J.S. Occupational Dermatoses. Clinical Medicine for the Occupational Physician ed. by Alderman, M.H. and Hanley, M.J., 1982, pp.299-344.

Tobiassen, L.S. Classification of sensitisers in the European Union in relation to global harmonisation, criteria for classification of skin and airway sensitising substances in the work and general environments, pp. 110, 1997.

Trojanowska, B. (1959). The pollution with nitro- and amino compounds of the work clothes and of the skin of the dye industry workers. Med. Pracy. 6, 387-392.

Tsuruta, H. (1982). Percutaneous absorption of organic solvents. III. On the penetration rates of hydrophobic solvents through the excised rat skin. Ind. Hlth. 20, 335-345.

UNITED NATIONS. Environmentally sound management of toxic chemicals, including prevention of illegal international traffic in toxic and dangerous products. In; Agenda 21: Programme of Action for Sustainable Development. RIO Declaration on Environment and Development. United Nations, 1992: 186-196.



Wester, R.C., and Maibach, H.I., (1997). Percutaneous absorption in man and animal; a perspective. In Cutameous Tocicity (edited by Drill, V.A. and Lazar, P.), pp111-126. Academic Press, New York.

World Health Organisation. Criteria for classification of skin-and airway sensitising substances in the work and general environments. Report on a W.H.O working group, Copenhagen, Denmark, 17-20 January, 1996.

Yonemoto, K., Gellin, G.A., Epstein, W.L. et al.; Reduction in eumelanin by the activation of gluthathione reductase and gamma glutamyl transpeptidase after exposure to a depigmenting chemical. Biochem, Pharm. 32: 1379-1382, 1983.



APPENDIX A

Allergic Contact Dermatitis and Patch Testing





Plate 33 Photoallergic contact dermatitis from 6-methylcoumarin in a sun lotion. Note the sparing beneath the wristwatch.

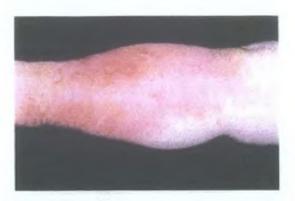


Plate 34 This stastis dermatitis was made much worse after application of a topical antibiotic. Patch testing was positive to neomycin found in the topical antibiotic.



Plate 35 Persistence of this generalized eczematous dermatitis requires patch testing to rule out an occult allergen.



Plate 36 Allergic contact cheilitis due to cinnamic aldehyde found in the tartar control toothpaste that this patient was using.



Plate 37 Allergic contact dermatitis from oak moss, a fragrance ingredient in this patient's husband's cologne a "consort" dermatitis.



Plate 38 Chronic allergic contact dermatitis due to nickel in earrings and jeans buttons. Note involvement of the earlobe and neck as well as the abdomen.





Plate 23 This individual was allergic to benzocaine found in a medication used to treat poison ivy.



Plate 24 This nurse was allergic to the rubber gloves she wore when taking care of patients. Patch tests revealed positive reactions to thiuram mix and a portion of her gloves.



Plate 25 This patient was allergic to quaternium-15 present in topical steroid used to treat a chronic irritant contact dermatitis. Her dermatitis flared and failed to clear when the topical steroid was used.



Plate 26 This child's foot dermatitis was caused by an allergy to mercaptobenzothiazole found in her sneakers.



Plate 27 This individual was allergic to mercaptobenzothiazole found in his flip-flops.



Plate 28 This foot dermatitis was due to an undefined shoe component. Patch test results to parts of shoes were positive, but test responses to rubber and leather antigens in the standard and miscellaneous trays were all negative.



APPENDIX B

Contact Dermatitis: Criteria for evaluating probable occuaptional causation



Contact dermatitis: Criteria for evaluating probable occupational causation

Criterion	Yes	No	Don't know
. Is the clinical appearance consistent with contact dermatitis?	Eczematous morphologic or histologic findings or Adequate clinical description in history or medical records	morphologic or histologic findings	No dermatitis on clinical examination; inadequate clinical description in history or medical records <i>or</i> Noneczematous reaction sometimes mimicked by contact dermatitis (e.g., lichenoid eruptions)
2. Are there workplace exposures to potential cutaneous irritants or allergens?	Supported by toxicologic data or clinical experience	Not supported by toxicologic data or clinical experience	Toxicologic properties of the exposure not known
anergens: 3. Is the anatomic distribution of dermatitis consistent with cutaneous exposure in relation to the job task?	Dermatitis is most severe on skin surfaces with maximal exposure (depends on physical form of irritant or allergen)	Dermatitis does not affect skin surfaces with greatest exposure	Dermatitis affects skin surfaces with maximal exposure but is more severe on other body areas (excluding eyelid, facial, genital skin) <i>or</i> Dermatitis spares skin surfaces with maximal exposure but affects eyelid, facial, or genital skin
4. Is the temporal relationship between exposure and onset consistent with contact dermatitis?	First or increased exposure preceded onset or aggravation and Onset or aggravation within 6 months of first or increased exposure	Onset or aggravation preceded the first exposure or Onset or aggravation occurred more than 3-4 days after last exposure (exception: initial allergic reaction)	Onset or aggravation occurred more than 6 months after first or increased exposure
Criterion	Yes	No	Don't know
5. Are nonoccupationa exposures excluded a probable causes?		Likely on the basis of a thorough history or patch tests	Inadequate history or Exposure to irritants o allergens both withir and outside the workplace
6. Does dermatitis improve away from work exposure to th suspected irritant or allergen?		No improvement after more than 1 week away from work exposure and No concomitant exposure to other irritants or allergens	Improvement coincides with medical treatment or Failure to improve ma be attributed to othe irritants or allergens or No improvement but
7. Do patch or provocation tests identify a probable causal agent?	Positive reaction, with tests performed according to established guidelines <i>and</i> Exposure has occurred in the workplace	Negative reaction, with tests performed according to established guidelines and All potential workplace allergens tested	according to established guideline or All potential workplac

A Intrinsic Research State

APPENDIX C

Reporting of Occupational Dermatological Diseases



REPORTING OF OCCUPATIONAL DERMATOLOGICAL DISEASES

<u>CENTRE NAME</u> FROM (date) <u>TO</u> Below are a list of the diagnostic groups to be used in the First column of the Reporting Table eg Group **G.4** would be used in the column marked Group for a farmer with malignant melanoma.

A	CONTACT DERMATITIS	D	MECHANICAL - Traumatic
	1 Allergic 2 Irritant 3 Allergic and Irritant	F	NAIL - 1 Dystrophy 2 Paronychia
	4 Unclear	G	Neoplasia
В	CONTACT URTICARIA		1 Keratosis 2 Basal cell 3 Squaemous cell 4 Melanoma
С	FOLLICULITIS/ACNE		
D	INFECTIVE - 1 Tinea 2 Warts 3 Others		H OTHER DERMATOSES - (specify) 1 2

3

I have nothing to report

DETAILS OF ALL CASES. If one line is insufficient use the line below

Group	patient	I.D Nos	SEX	DOB	County	TYPE OF WORK	SUSPECT AGENT
					1		1
			1	1			

eporter's Name _

Initials _____

eturn to Occupational Medical Service, 10 Hogan Place, Dublin 2



APPENDIX D

Glove Selection Chart



Glove selection chart

Gloves should only be used as indicated in this chart and only in accordance with the approved working practice.

CARBO CARBO	Natural rubber, flock lined Suitable for: Detergents, mild acids, water Not suitable for: Solvents, strong acids, metalworking fluids. Use: Canteen, general cleaning (floors, toilets, washrooms etc.) but NOT machine cleaning.
	Nitrile rubber, flock lined Suitable for: Detergents, mild acids, water, some emulsions. some solvents. Not suitable for: Toluene, Xylene, MEK, Trichloroethylene, strong acids Use: Protection against metalworking fluids, general machine cleaning, but NOT where solvents are being used.
k	Cotton lined PVC gauntlet Suitable: As general protection against sharp edges, wet components, splinters in pallets etc. Not suitable: For protection against any chemicals Use: Handling oil drums, pallets, castings etc.
4	Viton rubber, unlined Suitable for: Solvents Use: In degreasing plant, when handling toluene, xylenc, trichloroethylene but NOT when handling hydrofluoric acid. These gloves are for splash protection only - use only as directed in work procedure.
4	Butyl rubber, unlined Suitable for: Hydrofluoric acid Not suitable: Any solvent or other substance in the plant. Use: In degreasing plant when working with hydrofluoric acid. These gloves are for splash protection only - use only as directed in work procedure.



APPENDIX E

Survey Questionnaire



QUESTIONNAIRE SURVEY MSc.1999

<u>NOTE</u> : All the information on this questionnaire is considered confidential and will remain so . The information gathered is for informational purposes only.
Company Name;
Please place a tick $$ in the box for the correct answer
Q1. Nature of business;
Pharmaceutical Chemical Other
specify other.
Q2. Number of employees
10-50 50-100 100-250 >250
Q3. Type of company;
Manufacturing Other
Q4. What are the main products manufactured;

Q5. Would any of the substances/chemicals that your company would use/manufacture be classified as Skin Sensitisers? (i.e Have R43 risk phrase displayed on a Material Safety data sheet).

Yes

QUESTIONNAIRE SURVEY MSc.1999

if yes;

Please list the substances;

Q6. Do any of the above listed substances (carrying R43 risk phrase) possess any specific characteristics that make the control of exposure to them difficult?

•
(

if yes

What ? (e.g solid/liquid, MP, BP,)

Q7. Have there been cases of contact dermatitis/skin irritation at your workplace? (i.e do any workers have skin allergies?)

Yes

No 🔽

Q8. What is the general procedure for dealing with a worker that develops/has and allergy to a specific substance;

Prevent exposure by:

(i) Removing the person from the area where the substance is been used

(ii) Provide the person with personal protective equipment to reduce/eliminate exposure.

(iii) Engineering controls

Q9. Was there a Risk Assessment carried out for the area/process where the substance classified as a skin sensitiser is used?

	Yes	No
<u>If yes</u>		
Is this	Risk Assessment do	ocumented?
	Yes	Νο
Q10.	Is there a Skin Mana	agement Plan/Policy in place in your company?
	Yes	No 🗌
<u>lf yes</u>		
Is this	Skin Management p	plan/policy documented?
	Yes	Νο
		nal protective equipment are available to someone classified as a skin sensitiser?

I sincerely thank you for participating in this questionnaire.

A 120

Sonya Morrissey

APPENDIX F

Survey Matrix



Questions No.1Nature of business	1	1						1	9	10	11	12	13	14	15	16	17	18	19	20	21
pharm	X	+	ť	+-	F	+-"	x	F	F	X				X	X			X	++	+	X
chem	Ê	×		x	x	x	Ĥ	\vdash	x	Ĥ	Ĥ	Ê	Ê	Ê	f		\vdash	Ê	\square	<u>^</u>	Ê
other	+	-	×	-	Ê	1		x	Ĥ	-	+'	\vdash	\vdash		+'	x	×	-	X		
5000	+-	+-	-	+	\vdash	\vdash		Ĥ	\vdash	\vdash	\vdash		\square	<u>+</u> '		<u> </u>	Ĥ	\square	Ê	\square	-
No.2 No.of employees	+-	+-	\vdash	+	\vdash	\vdash	\vdash	\vdash	+'		\vdash		\vdash	\vdash	+'	-	+-'	1-			\vdash
10 to 50	x	+	x	+		x		\vdash	+-'		\vdash	\vdash	\vdash	\vdash	\vdash	\vdash	\vdash	\vdash	1-		\vdash
50 to 100	+	+	f	+	x	Ĥ	\vdash	x	\vdash	\vdash	x	\vdash	\vdash	\vdash			x	1-	x		\vdash
100 to 250	+	+	+	1x	Ĥ	+	\vdash	Ĥ	\vdash	-	-	x	-	x	\vdash	x	Ĥ	x	Ĥ	\square	-
>250	+	x	+	<u> </u>	-	+-'	x	\vdash	x	x		++	x	Ĥ	x	Ĥ	\vdash	Ĥ	\vdash	x	x
>250	+-	+	+	+	\vdash	+	Ĥ-	'	Ĥ'	<u> </u>	+-'	<u>+'</u>	Ĥ	\vdash	<u> </u>	\vdash	\vdash	\vdash	\vdash	Ĥ	Ê
No.3 Type of company	+	t		+					E			1	1	1							
Manufacturing	x	x	x	x	×	x	x	x	x	x	x	x	x	х	x	x	x	х	x	х	х
Other	1			\square		1													\Box	\Box	
	1			\square																	
No.4 (not applicable)	t	t																	\Box		
	T		F	L		T				F			Ľ		L	L	L		\Box'	Ľ	F
No.5 R43 substances					1				1					1				1		1	4_
Yes	X	X	X	X	X	X	X	Х	X	X	X	Х	X	X	X	X	X	Х	Х	X	X
No			L																		
No.6 Specific characteristics																					
Yes (Y)		L	Х		X							Х	Х	Х				X	X		X
No (N)	X	X	Ι	Х	L	Х	Х	Х	Х	Х	X	Ĺ			X	X	X			X	
	L	L	L	L	L					L	L	L								Ĺ	
No.7 Cases of Contact Dermatitis	Ĺ	L	L	L	L	L				L			L					L			L
Yes(Y)	L	Х	X	L	X	Х		X	X	Х		Х	Х		L	Х	Х			Х	X
No(N)	Х	L	L	X	L	L	X		L	L	Х	L	L	Х	X			Х	Х		L
	T	T	L	T	L						I	L	L					Ĺ			
No.8 Control Measures	L	L	L	L	L	L			L	L	L	L	L	L				L	L		
Personal Protective equipment	T	T	T	X	Х	Х	X	X	L	Х	X	Х		L	X	Х	Х	_	Х		
Remove from area	Х	Х	T	t	t	X	T	T	х	T	Х	Х	T	T	X	T	Х	Х	X	X	X
Engineering controls	T	t	X	T	t	X	T	T	t	t	t	X	Х	Х	X	T	t	t	Х	t	T
	T	T	T	T	t	T	T	T	T	t	T	T	T	T	T	t	t	t	t	t	T
No.9 Risk Assessment	T	t	T	t	t	t	t	t	T	t	T	T	t	T	T	t	t	T	L	L	L
Yes	t	T	T	t	t	T	Х	t	t	t	T	t	t	t	T	T	T	T	t	T	T
No	X	X	X	X	X	X	T	X	X	X	X	X	X	X	X	X	X	X	X	X	X
	1	1	1	1	1	1	T	Ļ	T	1	1	1	L	1	1	1	1	1	1	1	1
No.10 Skin Management Policy		\perp	\perp	\perp	\perp	1	1-	1	1	+		-		1	\perp	_		1		+-	+
Yes			\perp				\perp		1	\perp	1	1	1	L				\perp	L		\perp
No	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X



Questions	Co	om	pa	ny	Su	Irv	eye	d													
No.1Nature of business	22					27		29	30	31	32	33	34	35	36	37	38	39	40	41	42
pharm		Х		X	Х	Х			Х	Х										Х	
chem			x				х	Х				X		Х		Х	Х	X			X
other	x										Х		Х		Х				X		
No.2 No.of employees																					
10 to 50	X	X												х	Х	Х	х				X
50 to 100			X	Х			X	Х	Х			Х	Х						Х		
100 to 250																		Х			
>250	1				Х	Х				Х	Х									Х	
No.3 Type of company																					
Manufacturing	x	х	х	х	Х	X	Х	Х	х	х	x	X	Х	Х	х	Х	Х	Х	Х	х	Х
Other																					
No.4 (not applicable)																					
No.5 R43 substances																					
Yes	X	X	Х	Х	X	X	Х	Х	Х	X											
No											X	Х	Х	X	Х	Х	X	X	X	Х	X
No.6 Specific characteristics																					
Yes	X		X																		
No		Х		X	Х	X	Х	Х	Х	Х	Х	X	Х	Х	Х	X	Х	Х	Х	X	X
No.7 Cases of Contact Dermatitis																					
Yes		X	X			X		Х	Х	Х	Х						X	Х		X	
No	X			Х	Х		x					X	X	X	Х	X			Х		X
No.8 Control Measures																					
Personal Protective equipment	X	X		X	X	X	X	X	X	X			X	Х	Х	X	X	X	X	X	X
Remove from area		X	X			X	X				X	X		X		X					
Engineering controls		X																			X
No.9 Risk Assessment																					
Yes																					
No	X	Х	Х	X	Х	Х	Х	X	X	Х	Х	Х	X	Х	X	X	X	X	X	X	X
No.10 Skin Management Policy	-	-	-	-	-		-	-	+	+	-	+		+	-	-		+-	-	+	+-
Yes	+-	+	-	+	+		+	+	+	+	+		-	+	+	+	-	+-	+	+	+
No	x	x	x	x	×	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x



Questions	C	om	ра	ny	Su	rve	eye	ed													
No.1Nature of business		_			47			50	51	52	53	54	55	56	57	58	59	60	61	62	63
pharm		X		X	Х		Х	Х		Х						X			Х		Х
chem	x		х			Х			х				х		х			Х			
other											Х	х		х			x			х	
				-																	
No.2 No.of employees																					
10 to 50	X					х					х						Х				
50 to 100			Х	X	Х				Х	Х		Х	Х		Х	Х		Х	Х	Х	Х
100 to 250		х					Х	х						Х							
>250																					
No.3 Type of company	-	-	-	-				-													-
Manufacturing	x	x	Х	x	x	x	х	x	x	х	х	х	х	х	x	x	x	х	x	x	x
Other																					
							-						\square								
No.4 (not applicable)																					
No.5 R43 substances	-			-				-			<u> </u>			-	-	\vdash	-		-	-	\vdash
Yes																					
No	x	x	х	x	x	x	х	x	x	х	х	х	x	х	x	x	x	x	x	x	x
No.6 Specific characteristics																				T	Γ
Yes			-																		Γ
No	x	x	x	x	x	x	x	x	X	x	X	x	x	X	X	X	x	x	X	X	X
				1																	Г
No.7 Cases of Contact Dermatitis								1													Γ
Yes		x					x					Х	х		X	X		X	X		Γ
No	x		х	x	х	х		X	X	х	х			X			х			X	X
No.8 Control Measures	\vdash	\vdash	-	-	-			\vdash	\vdash	-	-		-	-	-		-	\vdash	-	-	+
Personal Protective equipment	x		x		x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	X	İX
Remove from area	1	1	t -			<u> </u>	†	-	1						1			1			t
Engineering controls	x	x		x	\square		-					1	x		1	x			x	\top	TX.
				1				1					1	1					1	1	T
No.9 Risk Assessment					E									-							Ţ
Yes														1							+
No	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	¥
No.10 Skin Management Policy	\vdash	\vdash	\vdash	\vdash		\vdash	\vdash	\vdash	$\left \right $	\vdash	-	-	\mathbf{T}	+	+-	+			\vdash		t
Yes															T						T
No	x	x	x	x	X	x	X	X	X	x	X	X	X	X	X	X	X	X	X	x	6



APPENDIX G

National Authority for Occupational Safety and Health Code of Practice for the Safety, Health and Welfare at Work (Chemical Agents) Regulations, 1994

NATIONAL AUTHORITY FOR OCCUPATIONAL SAFETY AND HEALTH

1997

CODE OF PRACTICE

for the

Safety, Health and Welfare at Work (Chemical Agents) Regulations, 1994



Notice of issue of a Code of Practice

The Code of Practice provides practical guidance as to the observance of Regulations 3 and 4 of the said Regulations as regards occupational exposure limits for the chemical agents listed in Schedule 1 to this Code.

This Code of Practice comes into effect on, 1997, and replaces the Code of Practice issued by he National Authority for Occupational Safety and Health on 23rd December, 1994. Schedule II to this Code lists the chemical agents for which the occupational exposure limit has charged in this Code of Practice compared to the Code issued in 1994.

Signed

C D Body Secretary to the Board, 1996



INTERPRETATION

For the purposes of this Code of Practice:

1. "Occupational Exposure Limit" means the maximum permissible concentration, of a chemical agent in the air at the workplace to which workers may be exposed, in relation to a 8 hour or a 15 minute reference period, as set out in Schedule 1 to this Code. The concentration of the chemical agent in air is expressed as parts per million (ppm), milligrams per cubic metre (mg/m³), or fibres per millilitre as appropriate.

"8 hour reference period" relates to the procedure whereby the occupational exposures in any 24 hour period are treated as equivalent to a single uniform exposure for 8 hours (the 8 hour time weighted average (TWA) exposure). The TWA may be expressed mathematically by:

 $(C_1 T_1 + C_2 T_2 + \dots + C_n T_n) / 8$, where $C_1 \dots C_n$ are the occupational exposures and $T_1 \dots T_n$ are the associated exposure times in hours in any 24 hour period.

"15 minute reference period" means the short term exposure reference period and is the sampling period used for assessing compliance with the associated exposure limit.

- 2. For exposure periods of less than the short term reference period, appropriate action shall be taken to ensure that exposure does not exceed three times the short term exposure limit unless a suitable and sufficient assessment has indicated that such exposures do not present a risk to health.
- 3. For those substances which have not been assigned a short term exposure limit and where exposure periods are less than the 8 hour reference period, appropriate action shall be taken to ensure that exposure does not exceed three times the 8 hour exposure limit unless a suitable and sufficient assessment has indicated that such exposures do not present a risk to health.
- 4. Schedule 1 to this Code of Practice stipulates the occupational exposure limits for substances listed in that Schedule.
- 5. Within Schedule 1 five groups of substances are additionally identified as having the potential to cause particular and significant reactions in the employees following exposure. These groups may be identified by the



following notations which are included in the notes column of the Schedule:

- C1 Substances known to be carcinogenic for man (Category 1 carcinogens) to which the Safety, Health and Welfare at Work (Carcinogens) Regulations, 1993 (S.I.No. 80 of 1993) apply;
- C2 Substances which should be regarded as if they are carcinogenic for man (Category 2 carcinogens) to which the Safety, Health and Welfare at Work (Carcinogens) Regulations, 1993 (S.I.No. 80 of 1993) apply;
- Sk Substances which have the capacity to penetrate intact skin when they come in contact with it, and be absorbed into the body;
- Asphx Gaseous chemical substances which may not produce significant physiological effects in the exposed employee, but when present in high concentrations will act as simple asphyxiants;
- Sens Chemical agents which following exposure may cause sensitization of the respiratory tract and lead to asthma, rhinitis or extrinsic allergic alveolitis. Substances which cause skin sensitisation (allergic contact dermatitis) are not specifically identified by this notation in the Schedule.

PERIODIC REVISION OF THE CODE OF PRACTICE

A revision of the occupational exposure limits listed in Schedule 1, to reflect current knowledge concerning the health hazards of the listed chemical agents, will be undertaken by the National Authority for Occupational Safety and Health on a biennial basis, in consultation with its Dangerous Substances Advisory Committee. Schedule III to this Code provides a list of chemical agents for which it is the intention to introduce an occupational exposure limit or to change the existing occupational exposure limit in 1999. Comments may be made in writing to the Authority at its headquarters, 10 Hogan Place, Dublin 2, concerning any of the limits proposed.

cb1111sl.96

APPENDIX H

Health and Safety Authority of Ireland, Annual Report 'Surveillance of Work Related Diseases', 1997



absent from work for more than three days in 1993 to 5400 in 1994 and 4000 in 1993. Frowever, even at these levels the extent of occupational ill-health remains considerable. The LFS returns indicate that the 4600 persons absent from work for more than three days due to occupational ill-health in 1995 accounted for 178800 work days lost and, in all, 185000 work days were lost due to occupational ill-health during the year.

Back to Index

Department of Social Welfare

The following is the data on occupational disease returns for 1994 and 1995 obtained mainly from the Department of Social Welfare, Ireland:

Disease	Cases Reported 1994	Cases Reported 1995	Cases Reported 1996
Occupaional Dermatitis	44	63	38
Musculoskeletal	18	29	15
Occupational Asthma	9	9	10
Asbestos-related	6	7	4
Occupational Deafness	6	1	2
Pesticide	4	2	6
Coal or Silica-related	4	3	3
Tuberculosis	3	1	1
Brucellosis	2	3	2
Other Lung Disease	2	0	0
Other	7	5	2
Leptospirosis	0	0	3
TOTAL	105	123	86

Back to Index

DISEASES REPORTED SWORDS

Disease	Cases Reported 1995	Cases Reported 1996
Asthma	63	70
Inhalation Accidents	8	11
Allergic Alveolitis	6	5
Bronchitis / Emphysema	0	2
Infectious Diseases	1	1
Non-Malignant Pleural Disease	14	7
Mesothelioma	4	2
Pneumoconiosis	1	0
TOTAL	99	99

This table gives the totals recieved at the HSA from the "Surveillance of Work Related

