Respiratory exposure and potential dermal exposure to volatile organic compounds in nail salons: a pilot study

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Preface

This mini-dissertation was written in article format. The practical work consists of a pilot study that quantified the 8 hour exposure of nail technicians to Hazardous Chemical Substance (HCS). The use of charcoal pads as a method to quantify potential dermal exposure was also investigated to assess its accuracy and to promote insight into dermal sampling. The improvement of dermal exposure assessment is an important goal for occupational hygiene research and is likely to lead to better health for worker populations (Fenske, 1993:687).

Chapter 1 is an introduction into this study, it also elaborates on the hypotheses and the aims of this study. In Chapter 2 a basic summary is given of the relevant literature regarding the toxicology of the HCS found in nail salons. These HCS include acetone, ethyl methacrylate, methyl methacrylate, formaldehyde, toluene and xylene. It also gives a brief account of previous studies done with regards to the exposure of nail technicians and the possible health effects thereof. The role of dermal exposure in the current occupational hygiene setting is also discussed as well as the importance of quantifying dermal exposure. Chapter 3 consists of a document written as an article in accordance with the format required by the journal: Annals of Occupational Hygiene to which it will be submitted for publication. The author instructions state that tables and figures should be on separate pages at the end of the text. However to improve readability the tables and figures in the article was placed in the text. The article is entitled *"*Respiratory exposure and potential dermal exposure to volatile organic compounds in nail salons: a pilot study*"*. Chapter 4 provides a final summary and conclusion. Recommendations for further studies are also included in this chapter.

The references used in the Preface, Chapter 1, Chapter 2 and Chapter 4 are provided according to the mandatory style stipulated by the North-West University (Harvard style). The relevant references of Chapter 3 are provided at the end of the chapter according to the author's instructions of the Annals of Occupational Hygiene (Vancouver style). As this chapter will be submitted to the Annals of Occupational Hygiene for peer reviewing and publication.

Author's contribution

The study reported in this dissertation was planned and executed by a team of researchers. The contribution of each of the researchers is depicted in Table 1.

Table 1: Research Team.

NAME	CONTRIBUTION
Ms. C. Spoelstra	▪ Designing and planning of study; ▪ Literature searches, interpretation of data and writing of the article; ▪ Recruiting nail technicians; ▪ Sampled exposure of nail technicians in salons..
Ms. A. Franken	▪ **Supervisor** ▪ Assisted with designing and planning of the study, approval of protocol, interpretation of the results and documentation of the study.
Mr. J. L. Du Plessis	▪ **Co-supervisor** ▪ Assisted with the approval of the protocol, interpretation of the results, reviewing of the dissertation and documentation of the study; ▪ Giving guidance with scientific aspects regarding dermal exposure sampling of the study.
Ms. N. Van der Merwe	▪ Assist with recruiting nail technicians; ▪ Helped with the personal exposure sampling

The following is a statement from the co-authors that confirms each individual's role in the study:

I declare that I have approved the above mentioned article and that my role in the study as indicated above is representative of my actual contribution and that I hereby give my consent that it may be published as part of Christa Spoelstra's M.Sc (Occupational Hygiene) dissertation.

_____ _____

Ms. A. Franken
(Supervisor)

Mr. J. L. Du Plessis
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Ms. N. Van Der Merwe

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After completing an enormous task like this, one looks back and think of all the hours of planning, hard work, tears and frustrations it took. I could however not have completed this study without help.

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Abstract

Objectives: The aims of this pilot study were to quantify respiratory and potential dermal exposure of nail technicians to acetone, formaldehyde, ethyl methacrylate, methyl methacrylate, toluene and xylene. Fifteen female nail technicians, working in different salons participated in this study. Products used for nail treatments differed between salons. Most salons used acrylate based nail products whereas others used UV-gel products exclusively. **Methods:** The participants were divided into two groups, those who used acrylate- and those who used UV-gel products exclusively. Eight hour personal respiration exposure to acetone, formaldehyde, ethyl methacrylate, methyl methacrylate, toluene and xylene were determined. The concentration of airborne volatile organic compounds in the salons was also determined with the use of a direct reading instrument (EntryRAE). Potential dermal exposure to the above mentioned solvents (excluding formaldehyde) was determined with the use of charcoal pads (surrogate skin method). During respiratory and dermal sampling, observations were made regarding work practices and control measures used in the salons. **Results:** It was found that the eight hour time weighed average exposure is well below the recommended occupational exposure limits of the individual chemicals and showed no additive effect. The highest mean respiratory exposures in both groups were acetone (27.22 mg/m^3 and 28.36 mg/m^3). EntryRAE results showed peak periods of exposure to volatile organic compounds during the day (322.16 ppm) that were much higher than the average eight hour exposure (0.21 ppm). The two groups' exposure levels were compared to determine if there is a significant difference between the exposures levels but no statistically significant difference was found. The dermal exposures on hand and neck to acetone, ethyl methacrylate and methyl methacrylate showed strong significant correlations to the concordant chemical's respiratory exposures. Correlations between air and dermal exposure was calculated once more after adjusting dermal exposure but the findings indicated only one statistically significant correlation of 0.42 in the case of ethyl methacrylate. **Conclusion:** Nail technicians are not at immediate health risk as the exposure in nail salons are well below recommended occupational exposure limits. However the unknown effects of chronic low level exposure to solvents and the large number of previous studies that reported increased health risks in nail technicians must also be considered. The use of methyl methacrylate in nail products sold in South Africa is also worrying as methyl methacrylate is banned by the FDA in the US due to its skin sensitisation potential that may lead to allergic contact dermatitis. The methods used to determine potential dermal exposure as well as adjusted dermal exposure remains problematic. This is due to the high percentage of adjusted dermal exposure values that had to be estimated and the fact that the activated charcoal pads have a higher absorption potential than human skin. Both methods must be improved to increase accuracy of results. Observations and EntryRAE results demonstrated the irregular nature of a nail technician's work shift as well tasks performed from day to day. This complicates gathering data that is representative of a nail technicians eight hour exposure. Therefore to further improve accuracy of results, sampling should in future be task specific.

Keywords: nail salon, dermal exposure, respiratory exposure, charcoal cloth pads, volatile organic compounds.

OPSOMMING

Doel: Die doel van hierdie studie is om die respiratoriese en potensiële dermale blootstelling van naeltegnici aan asetoon, formaldehied, ethylmethacrylaat, methylmethacrylaat, tolueen en xileen te kwantifiseer. Vyftien vroulike naeltegnici, wat in verskillende naelsalonne werk, het deelgeneem aan hierdie studie. Produkte wat gebruik is, het verskil van salon tot salon. Akriel-gebaseerde produkte is deur die meeste naelsalonne gebruik, waar die ander uitsluitlik UV-jel-produkte gebruik het. **Metodes:** Die deelnemers is in twee groepe verdeel afhangende van die naelprodukte gebruik (akriel of jel). Agt-uur persoonlike respiratoriese-blootstelling aan asetoon, formaldehied, ethylmethacrylaat, methylmethacrylaat, tolueen en xileen is gemeet. Die totale konsentrasie van vlugtige organiese verbindings in lug is ook vasgestel m.b.v. ŉ direkte-lees instrument (EntryRAE). Die potensiële dermale blootstelling aan bogenoemde chemikalieë (behalwe formaldehied) is bepaal deur die gebruik van koolstof-kussing-plakkers (surrogaat-dermale metode). Gedurende die respiratoriese en dermale monsterneming is werksprosedures asook die beskermende voorsorgmaatreëls wat in salonne getref is genoteer. **Resultate:** Daar is gevind dat die tydbeswaarde agtuur-blootstelling onder die voorgestelde beroepsblootstellinglimiete van die individuele chemikalieë was en dat blootstelling nie tot ŉ additiewe effek lei nie. Die hoogste blootstelling in albei groepe was aan asetoon (27.22 mg/m^3 en 28.36 mg/m^3). Die EntryRAE-resultate toon stiptye van blootstelling (322.16 dpm) aan vlugtige organiese verbindings gedurende die dag wat baie hoër was as die gemiddelde blootstelling oor agt ure (0.21 dpm). Die twee groepe se blootstellingsvlakke is met mekaar vergelyk, maar geen statistiese betekenisvolle verskil is gevind nie. Dermale blootstellings (hand en nek) aan asetoon, ethylmethacrylaat en methylmethacrylaat het ŉ betekenisvolle verwantskap met die ooreenstemmende respiratoriese blootstelling aan chemiese substanse gewys. Hierdie verwantskap is weer bereken nadat die dermale blootstelling op die hand aangepas is, die resultate wys dat slegs een betekenisvolle verwantskap van 0.42 in die geval van ethylmethacrylaat gevind is. **Gevolgtrekking:** Naeltegnici verkeer nie in onmiddellike gesondheidsgevaar nie, aangesien die blootstelling in naelsalonne beduidend laer is as die voorgeskrewe beroepsblootstellinglimiete. Nogtans moet die onbekende gesondheidseffekte van kroniese lae-vlak-blootstelling aan chemiese oplosmiddels asook die groot hoeveelheid soortgelyke studies wat verhoogde gesondheidsrisiko's van naeltegnici gerapporteer het, ook in aanmerking geneem word. Die verkoop van naelprodukte in Suid-Afrika wat methylmethacrylaat bevat is ook kommerwekkend aangesien die gebruik van methylmethacrylaat deur die FDA in die Verenigde State van Amerika verbode is. Dit is methylmethacrylaat se sensitiseringspotentiaal wat allergiese kontakdermatitis kan veroorsaak. Die metode wat gebruik is om potensiële dermale blootstelling te bepaal, sowel as die metode om die aangepaste dermale blootstelling te bepaal, bly problematies. Hierdie gevolgtrekking is gemaak na aanleiding van die hoë persentasie aangepaste dermale blootstellingswaardes wat geskat moes word, asook die feit dat die aktiewe koolstof-kussings ŉ hoër absorpsiepotentiaal het as die menslike vel. Beide metodes moet dus verbeter word om die akkuraatheid van resultate te verhoog. Waarnemings, tesame met die EntryRAE se resultate, demonstreer die onreëlmatigheid van naeltegnici se werkure asook wisselvalligheid van take wat daagliks verrig word. Dit is dus moeilik om betroubare data in te win wat verteenwoordigend is van ŉ naeltegnikus se agt uur blootstelling. Toekomstige monitering sal dus meer taakspesifiek moet wees.

Sleutelwoorde: naelsalon, dermale blootstelling , respiratoriese blootstelling, koolstof kussings, vlugtige organiese verbindings.

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List of Abbreviations

ASHRAE	American Society of Heating, Refrigerating and Air Conditioning Engineers
ATSDR	Agency for Toxic Substances and Drug Registry
BDL	Below Detection Level
CCOHS	Canadian Centre of Occupational Health and Safety
CIR	Cosmetic Ingredient Review
CNS	Central Nervous System
CSE	Chronic Solvent Encephalopathy
DNA	Deoxyribonucleic Acid
ERMA	Environmental Risk Management Authority
EMA	Ethyl methacrylate
FDA	Food and Drug Administration
FEV1	Fall in Forced Expiratory Volume in 1 second
FID	Flame Ionization Detector
GABA	γ-Aminobutyric Acid
HCS	Hazardous Chemical Substance
IARC	International Agency for Research on Cancer
IPCS	International Program on Chemical Safety
MMA	Methyl methacrylate
MPA	Methacrylate Producers Association, Inc
NICNAS	National Industrial Chemical Information Assessment Scheme
NIOSH	National Institute for Occupational Safety and Health
OEL	Occupational Exposure Limits
OEL-CL	Occupational Exposure Limits - Control Limit

OEL-RL	Occupational Exposure Limit – Recommended Limit
OSHA	Occupational Safety and Health Administration
PBZ	Personal Breathing-Zone
PEMA	Poly Ethyl Methacrylate
PID	Photo-ionisation Detector
PMMA	Poly Methyl Methacrylate
PPE	Personal Protection Equipment
PPM	Parts per Million
RTA	Renal Tubular Acidosis
SD	Standard Deviation
SK	Skin absorption
TNT	Tri-Nitro-Toluene
TWA	Time Weighed Average
USA	Unites States of America
UV	Ultra Violet
VOC	Volatile Organic Compound
VD	Vapour Density
VP	Vapour Pressure
WHO	World Health Organisation

CHAPTER 1

INTRODUCTION

1. Introduction

1.1 Problem statement

The desire for beautiful nails is a universal phenomenon under men and women (Heymann, 2007:1069). This is a rapidly growing industry that has tripled in the United States in the last two decades and has also grown likewise in the rest of the world (Molander *et al.*, 2006: 537-542). The result of this is a huge number of primarily women working as nail technicians in these salons.

Due to the range of products (adhesives, primers, nail polish, etc) used to apply artificial nails, nail technicians are exposed to a range of volatile organic compounds (VOCs) including solvents (Quach *et al.*, 2009:6; Molander *et al.*, 2006: 537-542). These chemicals include formaldehyde, toluene, acetone, ethyl methacrylate (EMA), methyl methacrylate (MMA), formaldehyde and xylene. The few preliminary studies done regarding exposure to VOCs in nail salons indicated a cause for concern not only for the nail technicians but also for the consumers (WVE, 2007:3). Most of these studies found self reported health effects namely musculoskeletal disorders, respiratory irritation and headaches. A higher prevalence of work related nasal and respiratory symptoms, including occupational asthma, in nail technicians when compared to a control group was reported (Adisesh *et al*., 2008:2; OHCOW, 2005:1). Skin problems including defatting and skin sensitisation that could lead to contact dermatitis was also found (Molander *et al.*, 2006: 537-542; OHCOW, 2005:1). Another concern is the correlation between spontaneous abortion and the number of hours worked per day as nail technician, the number of chemical services performed per week and work in salons where nail sculpturing was performed by other employees (John *et al*., 1994; Adisesh *et al.*, 2008).

Despite the reported health concerns, Regulations for Hazardous Chemical Substances (HCS) are not implemented in nail salons. It is also not unusual to find nail technicians working on clients' nails without any personal protection equipment (PPE) or proper ventilation (Capital health services, 2001:1).

Because nail technicians are exposed to a mixture of chemicals it is difficult to determine which chemical in the mixture is the cause of an allergic reaction or other health symptoms (NIOSH, 1999). Exposure to one chemical can also cause different health effects depending on the route of exposure. In the past quantification of exposure to HCS was primarily done by measuring respiratory exposure. However in recent years it has become apparent that dermal absorption of chemicals is also a major contributing factor when it comes to quantifying total occupational exposure. The human body is almost completely covered with skin, this makes the skin the largest organ of the body. As a result the skin is exposed to a number of chemicals each day and must be considered as an important route of exposure (Lu and Kacew, 2002:209). The hands are involved in 90 - 95% of all reported cases of skin diseases each year. Exposure to organic solvents is one of the main causes of irritative contact dermatitis and allergic contact dermatitis (Niedner, 2008:334). Knowledge of dermal exposure is therefore fundamental to hazard

evaluation and control. Improvement in the techniques of dermal exposure assessment is an important goal for Occupational Hygiene research and is likely to lead to better health for worker populations (Fenske, 1993:687).

A study has been launched to quantify the respiratory and dermal exposure to different HCS in nail salons.

1.2 Hypotheses

- Nail technicians' respiratory exposure levels measured over 8 hours are below the TWA occupational exposure limits.
- Surrogate skin methods such as the charcoal pad (PERMEA-TEC pads®) can be used as a reliable method to quantify dermal exposure to chemicals in nail salons.

1.3 Research objectives

The objectives of this study were to:
Quantify nail technicians' personal respiratory exposure to acetone, ethyl- and methyl- methacrylate, formaldehyde, toluene and xylene.
Quantify dermal exposure to acetone, ethyl- and methyl- methacrylate, toluene and xylene with the use of charcoal pads (PERMEA-TEC pads®).

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CHAPTER 2

LITERATURE STUDY

2. LITERATURE STUDY

The literature study will focus on the potential exposure to Hazardous Chemical Substances (HCS) in nail salons and the potential health effects thereof on the nail technicians employed in these salons. The literature study will also include a summary of previous studies done with regard to occupational exposure to HCS in nail salons. Routes of exposure, namely air and skin exposure applicable to the exposure of nail technicians will also be discussed.

2.1 Products used in nail salons

According to Schoon (2005:171) nail enhancements (also known as extensions, false nails or artificial nails) are formed by coating the natural nail with a hard coating of acrylic. Typical nail enhancement systems consist of tip adhesives, wrap resins, liquid/powder systems, ultra violet (UV) gels and no-light gels. There are three main types of enhancements which all relay on acrylic monomers or oligomers (Schoon, 2005:171):
(a) Natural nail overlays – coatings that cover the nail plate but do not extend the nail;
(b) Tip and overlays – coatings which incorporate a plastic tip to extend the length of the nail; and
(c) Sculptured nails – coatings which extend the nail without using a plastic tip.

Schoon (2005:171) explained that there are a large variety of products that are used, but that they all share the characteristics that their basic ingredients (monomers or oligomers) undergo a polymerisation reaction to produce a hard acrylic polymer coating which forms the basis of the nail enhancement.

Energy is also required to stimulate the chemicals and trigger the polymerisation reaction. Some systems require light energy, for example UV light, while others need thermal energy. In some cases room temperature or the heat from the hand provides enough energy to start the reaction (Schoon, 2005:171; Newman, 2007 156-159).

Liquid and powder systems products are very commonly used in nail salons. This system involves combining a liquid monomer (such as ethyl methacrylate monomer (EMA)) with a powdered polymer (typically poly methyl and/or ethyl methacrylate (PMMA, PEMA) that contains the reaction initiator and other ingredients, such as colorants. The most commonly used polymerising enhancement products can be divided into three main categories according to the polymerising chemicals they contain (Schoon, 2005:171; Newman, 2007 156-159):
(a) Cyanoacrylates – wraps, no-light gels, tip adhesives;
(b) Methacrylates – monomer and polymer, UV nail enhancements; and
(c) Acrylates and Methacrylates – UV nail enhancements.

Ordinarily nail polishes (also known as nail varnishes, enamels, lacquers), topcoats and other nail treatments can be distinguished from enhancement systems because they form a hard coating by evaporation of solvents only and do not polymerise (Schoon, 2005:171).

2.2 Occupational exposure to HCS in nail salons

Table 1: Chemical ingredients in nail products (Roelofs and Tuan, 2007; OSHA, 1993; NIOSH Pocket Guide to Chemical Hazards, 2005)

Nail Products	Common Chemical Ingredients	CAS no.
Nail Polish	Ethyl acetate	141-78-6
	Butyl acetate	123-86-4
	Ethyl alcohol	64-17-5
	Isopropyl alcohol	67-63-0
	Acetone	67-64-1
	Methyl ethyl ketone	78-93-3
	Toluene	108-88-3
	Xylene	95-47-6
	Dibutyl phthalate	84-74-2
	Nitrocellulose	9004700
	Toluene sulphonamide	88-19-7
	Formaldehyde resin	50-00-0
	Titanium dioxide	13463-67-7
Nail polish removers	Acetone	67-64-1
	Ethyl acetate	141-78-6
	Butyl acetate	123-86-4
Artificial Nails (Includes acrylic polymers)	Ethyl methacrylate	97-63-2
	Methyl methacrylate	80-62-6
	Meth acrylic acid	79-41-4
	Methyl ethyl ketone	78-93-3
Nail Tip Adhesives	Ethyl cyanoacrylate	7085-85-0
Artificial Nail Removers	N-methyl pyrrolidone	872-50-4
	Acetone	67-64-1
	Acetonitrile	75-05-8
Disinfectants (Used in some salons)	Formalin (is an aqueous solution that is 37% formaldehyde by weight; inhibited solutions usually contain 6-12% methyl alcohol. Also see specific listings for Formaldehyde and Methyl alcohol.)	
	Isopropyl alcohol	67-63-0
	Bleach (sodium hypochlorite)	7681-52-9
	Ethanol (Hospital grade disinfectants)	64-17-5

It is abundantly clear from a perusal of Table 1 that a wide variety HCS are used during nail treatments. The exposure and health risks will however vary among the nail technicians depending on the frequency and type of nail treatment being performed in the specific nail salon.

The following HCS found in products used in nail salons will each be discussed with regards to their health effects: acetone, acrylic monomers (MMA and EMA), formaldehyde, toluene and xylene (Capital Health Services, 2007:1-4). All of the above mentioned chemicals can cause health problems after exposure to them and all have recommended- or control- occupational exposure limits as can be seen in Table 2. Each individual HCS will be discussed in more detail below.

Table 2: Occupational Exposure Limits (OEL) of Sampled Chemicals

	Regulations for hazardous chemical substances, 1995- Annexure 1.					**NIOSH**		**OSHA**	
HCS	**Notes**	**TWA OEL-RL**		**Short Term OEL-RL**		**TWA**		**TWA**	
		ppm	**mg/m^3**	**ppm**	**mg/m^3**	**ppm**	**mg/m^3**	**ppm**	**mg/m^3**
Acetone	-	750	1780	1500	3560	250	590	1000	2375
***Formaldehyde**	-	2	2.5	2	2.5	0.016	0.0197	0.75	0.923
MMA	-	100	410	125	510	100	410	100	410
EMA	No values have been given for this chemical.								
Toluene	SK	50	188	150	560	100	375	200	754
Xylene	SK	100	435	150	650	100	435	100	435

Note: TWA: Time Weighed Average
OEL-RL: Occupational Exposure Limit – Recommended Limit
SK: Skin absorption
* OEL-Control Limit
NIOSH: National Institute of Occupational Safety and Health, USA
OSHA: Occupational Safety and Health Administration, Department of Labour, USA

2.2.1 Acetone ($(CH_3)_2CO$)

Acetone also known as dimethyl ketone, ketone propane and 2-propanone is a colourless liquid with a fragrant mint-like odour (NIOSH, 2005).

2.2.1.1 Use

Dow chemical company (2006:2) reported that from the available acetone, 75% is used to produce other chemicals and only 12% is used as a solvent. Acetone is an ingredient used in a wide variety of products ranging from surface coatings, cosmetic products, films and adhesives to cleaning fluids and pharmaceutical applications. In nail salons acetone can be found in nail polish remover and in some nail varnishes. Pure acetone is also used to remove gel and acrylic nail overlays (Pearson, 2009).

2.2.1.2 Exposure

National Institute of Occupational Safety and Health (NIOSH) pocket guide (2005) names ingestion, respiratory and dermal absorption as routes of exposure. Examples of natural sources of acetone are decomposing vegetation and forest fires. These sources release acetone into the atmosphere and are responsible for roughly 97% of the acetone in the atmosphere. It is clear that acetone in the atmosphere due to man-made emissions (3%) are comparatively small.

The main route of exposure in the workplace is mainly through inhalation because of evaporation in various industrial and consumer product applications. In acetone manufacturing facilities the general exposure of workers to acetone is relatively low. This is because the process, storage and handling operation areas are enclosed (DOW chemicals, 2006:2).

2.2.1.3 Toxic effects in humans

According to the International Program on Chemical Safety (IPCS) (1998) acetone is one of three ketone bodies that occur naturally throughout the body. When acetone is inhaled or ingested it is rapidly absorbed via the respiratory and gastrointestinal tracts. Human studies showed approximately 50% of the inhaled amount of acetone is absorbed, by the body.

After absorption acetone does not accumulate in adipose tissues, but is evenly distributed among non-adipose tissues. Thereafter acetone is quickly cleared from the body by metabolism and excretion (IPCS, 1998).

2.2.1.4 Acute exposure

The New Hampshire Department of Environmental Health (2005) reported mild effects on the nervous system that abated soon after exposure stopped. These effects were seen in humans exposed to concentrations ≥ 500 ppm of acetone in air. Other symptoms included irritation of the eyes and respiratory system, mood swings and nausea. Tolerance to the effects of acetone can develop (CCOHS, Canadian Centre of Occupational Health and Safety, 1997).

Agency for Toxic Substances and Drug Registry (ATSDR) (1994:2) also reported that exposure to acetone can cause irritation in the nose, throat, lungs and eyes. Irritation can occur at levels of ≥100 ppm acetone in air. Exposure levels as high as 12,000 ppm can cause headache, light headedness, dizziness, unsteadiness, confusion and even unconsciousness, depending on how long exposure lasts. Other symptoms that have been linked to acetone exposure include a lack of energy, behavioural side effects and a premature period cycle in women.

Acute exposure through ingestion has been found to cause temporary unconsciousness and tissue damage in the mouth. In one case a man developed a limp which eventually cleared up and symptoms similar to diabetes such as excessive thirst and frequent urination (ATSDR, 1994:3).

Dermal exposure to acetone for more than thirty minutes may lead to skin irritation and even cause skin cell damage (ATSDR, 1994:3).

2.2.1.5 Target organs

The target organs for acetone are the eyes, nose, respiratory system, central nervous system (CNS) and the heart. In an animal study, rats were exposed to acetone which showed that target organs in male rats were the testis, kidneys, hematopoietic system and the liver, but in the female rats the liver was the only target organ affected (Dietz, 1991:3).

Direct contact to unbroken skin may result in redness and light swelling. The risk of developing health effects due to short term direct skin contact to acetone is very slim. Repeated or prolonged skin contact may however cause de-fatting of the skin area and also lead to contact dermatitis which can be identified by dryness, irritation, redness and cracking of the skin (CCOHS, 1997).

Concentrations of around 500 ppm acetone vapour will cause mild irritation to the eyes. These irritating effects become very apparent at 1000 ppm (CCOHS, 1997).

Respiratory system

According to the ATSDR (1994) respiratory exposure to acetone may cause irritation of the nose, throat, trachea and lungs. The irritating properties of acetone in humans have been noted in occupational settings and in controlled studies. Other symptoms that have been noted are the loss of ability to smell acetone.

In a pulmonary function testing study it was concluded that exposure to acetone caused no physical abnormalities (Specht *et al*. 1993). In related animal studies, respiratory side effects were observed, where exposure to acetone was much higher than those reported in human studies. Symptoms reported in these animal studies include pulmonary congestion, oedema, haemorrhage of the lungs and decreased respiratory rates (ATSDR, 1994).

Cardiovascular effects

ATSDR (1994) reported that there is currently limited information regarding the effect of acetone on the heart after respiratory exposure. Studies have shown that dermal and respiratory exposure may lead to an increase in pulse rates (120-160/minute). In earlier studies it was found electrocardiography of volunteers exposed to <1250 ppm acetone intermittently revealed no alterations when compared with the pre-exposure electrocardiograms (Stewart *et al.* 1975). It was also reported that there is no significant increased risk of death from circulatory system disease or ischemic heart disease due to acetone exposure.

In a more recent study it was reported that chronic exposure to acetone can lead to the same cardio toxic manifestation as toluene, namely, pro-arrhythmic effects due to its ability to lower parasympathetic activity, increase adrenergic sensitivity and altered ion homeostasis (Ramos *et al*. 2003:280).

In animal studies, reduced heart rates were observed in guinea pigs after exposure to very high concentrations of acetone for numerous acute durations. This may however be a consequence of the narcotic effects of acetone (ATSDR, 1994).

Reproductive/developmental effects

The CCOHS (1997) explained that no firm conclusion can be drawn regarding the effects of acetone on the reproduction system in either men or woman. This is mainly due to the lack of creditable human studies on this subject.

The New Hampshire Department of Environmental Health (2005) found that male rats exposed to very high concentrations of acetone in drinking water (3,400 milligrams per kilogram of bodyweight/day) had increases in malformed sperm and reduced sperm movement. Whether these effects would impair reproductive ability is however not known.

According to the CCOHS (1997) there is no human information with regards to acetone's effects on an unborn child. Animal information suggests that acetone would only cause effects in the presence of maternal toxicity.

2.2.1.6 Classification of acetone

The CCOHS (1997) reported that there is no information currently available on the carcinogenic effect of acetone on humans. Animal information suggests that acetone is not carcinogenic.

The Department of Health and Human Services and the International Agency for Research on Cancer (IARC) have not classified acetone as a human carcinogen.

2.2.2 Acrylic monomers: Methyl methacrylate [$CH_2=C(CH_3)COOCH_3$] and Ethyl methacrylate [$CH_2=C(CH_3)COO(C_2H_5)$ or $C_6H_{10}O_2$]

Methyl methacrylate (MMA) and ethyl methacrylate (EMA) are colorless liquids with a harsh fruity odor. MMA is also known as methacrylate monomer, methyl ester, methacrylate acid or methyl-2-methyl-2-propenoate. EMA is also known as ethyl-2-methyl-2-propenoate, ethyl-2-methylacrylate, methacrylic acid, ethyl ester, rhoplex AC-33, 2-propenoic acid or 2-methyl-ethyl ester.

2.2.2.1 Use

The two common acrylic monomers, MMA and EMA are widely used in nail salons (NICNAS, 2007:1; Autian, 1975:141).

Lewis (1998) explained that after extensive investigations the FDA (U. S. Food and Drug Administration) concluded that liquid MMA is a poisonous and harmful substance and should therefore not be used in fingernail preparations. There are however still no specific regulation prohibiting the use of liquid methyl methacrylate monomer in cosmetic products. After these and other concerns regarding the adverse health effects of MMA

exposure arose, its use has for the most part been replaced by ethyl methacrylate (EMA). EMA is considered to have a lower toxicity when compared to MMA. These concerns about MMA in cosmetic products relate only to MMA monomer, the liquid MMA and not the MMA-based polymers as the MMA-based polymers do not have the same toxicity profile as MMA monomers (Lewis, 1998).

EMA is commonly found as base material in coatings, inks, paints and adhesives. It is also used in resins, solvents, oil additives, dental products, textile emulsions and in leather and paper finishing's (Andersen, 1995:452; Association of petrochemicals producers in Europe, 2005).

2.2.2.2 Exposure

During the application of nails in salons the most apparent exposures to EMA and MMA is through inhalation of vapour and short-term skin contact in the area of the fingernail. The exposure of MMA is more likely in cut-price salons because it is cheaper than other methacrylate monomers including EMA. The consumer and the technician would both be exposed to MMA vapour throughout the application process, making adequate ventilation a necessity as the level of MMA exposure is influenced by the quality of ventilation (NICNAS, 2007). MMA is well absorbed after inhalation, oral intake and dermal exposure (even through intact skin). Peak blood concentration occurs approximately one hour after exposure. MMA is sequestered in red blood cells which is slowly released into the plasma. MMA has a half life in whole blood of 3 hours at 20°C. MMA is metabolized by coenzyme A to form methacrylic acid which is found normally in the Krebs's cycle. The methyacrylic acid metabolite undergoes standard lipid metabolism and is ultimately converted to pyruvic acid. In the case of EMA it appears that degradation occurs only in the plasma (Dart, 2004:1363).

2.2.2.3 Toxic effects in humans

In the United States of America legislation was passed in approximately 30 states to ban the use of MMA-containing nail products due to its corrosive properties and skin sensitisation properties of the esters to humans (Methacrylate Producers Association, Inc. (MPA), 2002). The Environmental Risk Management Authority (ERMA) of New Zealand (2002) and the Canadian Healthy Environments (2007) and Consumer Safety Branch (2005) have also proposed banning use of MMA in cosmetics.

Regardless of the mounting evidence against the use of MMA in cosmetics, the CIR (Cosmetic Ingredient Review) has not banned the use of MMA in nail cosmetics. The CIR (2009) named EMA as safe with qualifications in the reference table that included a complete list of findings. They explained that it is safe when application is accompanied by directions to avoid skin contact because of its sensitisation potential.

2.2.2.4 Acute toxicity

The LD_{50} concentration of MMA for oral exposed rats is 7552-9440 mg/kg and dermal exposed rats are > 5000 mg/kg. After oral exposure of MMA in animals the main clinical sign after about 2-5 minutes is increased rate of respiration, this will be followed by motor weakness and decreased respiration after 15-40 minutes. Discoloration

and piloerection has also been observed. MMA is not considered to be acutely toxic, possibly due to its rapid metabolism (NICNAS, 2007; European Chemicals Bureau, 2002:70).

2.2.2.5 Irritation

Almost all acrylic acid derivatives are known dermal irritants and the acrylates used in cosmetic nail products are corrosive and have caused skin burns (Dart, 2004:1364). MMA and EMA is known to be severely irritating to rabbit skin and causes desiccation, blanching and eschar (dry scab or slough on the skin) formation, erythema and oedema. Skin reactions like dermatitis, erythema and eczema occur in humans after MMA exposure (NICNAS, 2007; European Chemicals Bureau, 2002:70; Dart, 2004:1362).

MMA have been found to be a mild irritant to rabbits causing irritation to the sclera (white part of the eye) and the conjunctiva. EMA is also an eye irritant (NICNAS, 2007).

Respiratory irritation after exposure to high concentrations MMA has been observed in both animal and human tests (Dart, 2004:1365). Human data for occupational exposure indicated that respiratory irritation by MMA occurred at concentrations as low as 112 ppm (NICNAS, 2007).

Like MME, EMA is also a known irritant. According to Andersen (1995:452) EMA caused ocular, nasal and respiratory tract irritation in rats and other animals after acute inhalation. The International Program on Chemical Safety (IPCS) (2003) found that EMA is also a human irritant and may cause coughing (inhalation), redness of the skin, watering and redness of the eyes.

2.2.2.6 Sensitisation

Many case reports of skin sensitisation exist in occupational settings where recurrent long-lasting unprotected skin contact with MMA monomer containing preparations was common practice. Undiluted MMA can also lead to skin sensitisation in susceptible persons after continuous exposure (Chemicals Bureau, 2002:73). There are also a number of human clinical reports of skin sensitisation reactions due to MMA exposure. One study concluded that MMA and EMA caused allergic contact dermatitis in persons working with dental prosthesis and persons working with photo bonded sculptured nails. Photo bonded sculptured nails caused hand and face dermatitis because of a number of (meth)acrylate compounds that were present in nail cosmetics (Kanerva *et al*, 1996:108-109).

MMA proved to be a moderate to strong sensitizer in experimental animals, with clear evidence of skin sensitisation after direct contact with MMA. Sensitisation also occurred after cross-sensitisation reactions between different methacrylate esters (Chung and Albert 1977:187; NICNAS, 2007).

The European Chemicals Bureau, (2002:83) found that MMA is a potential respiratory irritant after acute occupational exposure but found no evidence to indicate MMA as respiratory sensitiser.

A patch test study was done on one hundred and twenty-four patients with a history of exposure to acrylate compounds. Six patients exhibited an allergic patch test reaction caused by ethyl methacrylate (EMA). According to Kanerva *et al.* (1995:7) EMA can be considered as a significant human contact allergen. It was also noted that if the exposure to EMA increases, it will lead to more patients being sensitised to EMA and also to other acrylic compounds because of cross-reactivity (Kanerva *et al.* 1995:75). It is recommended that skin contact to EMA must be avoided or when used in salons, only be applied by a trained individual (Cosmetic Ingredient Review Expert Panel, 2002:63; Andersen, 1995:452).

2.2.2.7 Neurotoxicity

A connection between MMA exposure and neurotoxic effects was made for the first time in 1986. A dose response analysis among dental technicians with regards to organic dementia, showed a statistically significant increase in the prevalence of chronic symptoms with increased exposure (Steendahl *et al*, 1992:1481). According to Abou-donia *et al.* (2000:97) EMA has also been implicated in the development of neurologic impairment after occupational exposure. According to Harold (2008:202) EMA, as a human neurotoxin, is a contributing factor to symptoms like loss of cognitive efficiency, learning, memory and neurosensory changes, including changes in sense of smell (which is below normal levels in nail salon workers). In a questionnaire based study 34% of dental technicians who handled monomeric MMA resin with unprotected fingers, reported dermatitis and 25% reported finger numbing, feeling of coldness and whitening in areas after frequent contact with MMA monomer. These neurological complaints were more common among those with a longer career and increased exposure.

It was concluded that the effect on the nervous system could be an independent effect from the exposure or that it reflects the slower regeneration of the nervous system than the local skin lesion (Rajaniemi, 1986:56). The National Industrial Chemical Information Assessment Scheme (NICNAS) (2007) also found human clinical reports that indicated that finger numbness and other neurological problems may be related to MMA exposure, but mentioned

that it was not possible to connect the degree of MMA exposure with the severity of neurotoxicity from these case reports.

The neurotoxic potential of EMA was also investigated by Abou-Donia *et al.,* (2000) in two animal studies that used adult male Sprague-Dawley rats. The first study showed EMA exposure caused alterations in clinical parameters in the higher dose groups included lethargy, impaired breathing, decreased weight gain and increased mortality. Changes in motor activity were observed at 100 mg/kg. In the second experiment, animals which had ingested EMA in drinking water in different concentrations and exhibited a large number of changes in their nervous system. Sponge like texture similar in appearance or porosity, as the fibre tracts of the forebrain, brainstem and spinal cord was observed. Axonal swelling was also observed in the dorsal, ventral and lateral columns of the spinal cord clusters. Other changes included shrunken axons with separated myelin lamellae and large axons with thinner than normal myelin sheaths were apparent in the sciatic nerve. This study concluded that the observed effects of EMA on the nervous system of the animals are consistent with neurologic symptoms of workers exposed to EMA. It was however recommended that additional studies must be done to establish if the level and route of exposures associated with occupational use can cause similar impairments in humans as those seen in experimental animals (Abou-Donia *et al*., 2000).

2.2.2.8 Reproductive and developmental toxicity

Information regarding reproductive and developmental toxicity is conflicting. In an animal study embryo-foetal growth retardation and some foetal malformations occurred after parenteral administration of MMA (Nicholas *et al*, 1979:541). This study also established that, when compared with the control group, pregnant rats (after inhalation of MMA) showed significant differences in maternal weight and food consumption, foetal weight, crown-rump length, early foetal deaths and certain gross and skeletal anomalies. No difference could however be found in the number of foetuses per litter. More recently it was concluded that there are insufficient evidence available to indicate whether MMA shows developmental or reproductive toxicity (European Chemicals Bureau, 2002:100; NICNAS, 2007).

EMA was categorised as embryo toxic in 1972 by Singh *et al.* (1972:1632). Exposure of female rats to different methacrylate esters including EMA and acrylic acid caused incidences of death of foetuses, gross abnormalities, skeletal malformations and lower foetal weight. Food consumption also lowered but no maternal deaths were observed during these studies (Saillenfait *et al*, 1999:136; Singh *et al.* 1972:1632).
The Cosmetic Ingredient Review Expert Panel (2002) also explained that evidence of embryo toxicity and teratogenicity were seen in rats after being injected with 0.1223-0.4076 ml/kg EMA.

2.2.2.9 Mutagenicity and carcinogenicity

Chan *et al*, (1988:237) did a carcinogenesis inhalation study (of MMA) on male and female rats and mice. They found no cases of neoplasms but found that non-neoplastic lesions in the nasal cavity of MMA-exposed rats and mice had significantly increased. The olfactory epithelium was also inflamed and showed degeneration. After the

MMA exposure they found degeneration of the olfactory epithelium and inflammation, hyperplasia and cytoplasmic inclusions in respiratory epithelium.

The NICNAS (2007) explained that after bacterial tests MMA was not mutagenic but found evidence of chromosomal damage at high doses exposure in cultured mammalian cells. They concluded that the limited epidemiological evidence does not indicate a carcinogenic potential for MMA.

The CIR (2002) concluded that EMA is a chemical that showed both positive and negative mutagenicity in tests.

2.2.2.10 Cytotoxicity and genotoxicity

In an examination for genotoxic activity, exposure to a series of monomeric acrylate/methacrylate esters (methyl acrylate, ethyl acrylate, MMA and EMA) were examined in mouse lymphoma cells. All compounds induced concentration-dependent increases in mutation frequencies and also gross chromosome aberrations in mouse lymphoma cells (Moore *et al,* 1987).

The cytotoxicity of MMA has been demonstrated in many cultured cell lines. The cytotoxicity of MMA may be a contributing factor to its other toxic properties (NICNAS, 2007). According to Yang *et al,* (2003:2909–2914) MMA is not only a cytotoxic agent but also a genotoxic agent.

2.2.2.11 Cardiovascular effects

Administration of MMA and EMA to rats caused hypotension, drop in hart rate, reduction of cardiac output and stroke volume and increased peripheral resistance. It must also be noted that the effect of EMA on blood pressure decreased was weak compared with that of MMA (Waters *et al,* 1992:497; Shukan *et al,* 2002).

2.2.2.12 Other health effects

When MMA is used as a bonding layer in nails, mechanical damage may occur when a nail breaks and cause damage to the nail plate. This may result in infections around the nail plate.

2.2.2.13 Classifications of acrylic monomers

The IARC (1978) also concluded that there is not sufficient evidence to classify MMA as being carcinogenic.

2.2.3 Formaldehyde (HCHO)

Formaldehyde is a colourless gas with a sharp overpowering odour and is also known as methanal, methyl aldehyde and methylene oxide.

2.2.3.1 Use

Formaldehyde is commonly used in nail salons. It is used in the manufacture of plastic nail tips and in nail polish. Formaldehyde is also present in some of the disinfectants used in nail salons (WHO, 2001; IARC, 2006).

2.2.3.2 Exposure

The possible routes of exposure to formaldehyde are ingestion, inhalation and dermal absorption. According to McNary and Jackson (2007:573) secondary exposure to formaldehyde occurs from inhalation of motor exhaust and cigarette smoke, forest fires and fields burned in preparation for planting. Exposure in nail salons occurs through inhalation and skin absorption. Formaldehyde is well absorbed by the lungs, gastrointestinal tract and to a lesser extent the skin. At levels to which humans may be exposed, adverse effects are most likely to be observed primarily following inhalation. It has been shown experimentally that effects on organisms (e.g. mammals) are more closely related to concentration than to the accumulated total dose. This is due to the rapid metabolism and high reactivity and water solubility of formaldehyde. Dermal exposure largely affects the skin itself and little if any formaldehyde reaches the bloodstream. Formaldehyde exposure is elevated from the ingestion of food, but most of it is present in a bound and unavailable form (WHO, 2001).

2.2.3.3 Acute toxicity

Many studies have assessed the health effects of inhalation of formaldehyde in humans. Most were carried out in unsensitised subjects and revealed consistent evidence of irritation of the eyes, nose and throat at exposure levels of 0.5–2.0 ppm. Symptoms are rare in concentrations below 0.5 ppm, however it becomes increasingly prevalent as concentrations increase in exposure chambers. Nose and throat irritation was the most sensitive response with an estimated threshold of 1 ppm. At concentrations between 0,03 ppm and 3 ppm mild-to-moderate eye and upper respiratory tract irritation occurred. The degree of irritation increased when concentrations increased. Tearing of the eyes occurred at exposure levels between 3.0–5.0 ppm; difficult breathing, nose and throat burning and heavy tearing of eyes occurred at concentrations between 10.0–20.0 ppm; and severe respiratory tract injury at 25.0–30.0 ppm exposure levels. Exposure levels of 100 ppm are immediately dangerous to life and health (IARC, 2006). The effects of inhaled formaldehyde on the airways of healthy people and unsensitised asthmatics have been reviewed (Liteplo and Meek, 2003). Exposure to 2–3 ppm formaldehyde for up to 3 hours did not provoke asthma in unsensitised asthmatics. High levels of formaldehyde cause asthmatic reactions probably by a hypersensitivity mechanism (IARC, 2006).

Krakowiak *et al.* (1998) reported on 10 healthy subjects and 10 asthmatics that were exposed to 0.5 mg/m^3 formaldehyde (range, 0.16–0.57 ppm) for 2 hours. During exposure to formaldehyde, all subjects developed sneezing, itching and congestion, with substantial resolution after 4 hours. An increase in total leukocytes and eosinophils was observed in both groups immediately after exposure, with resolution after 4 hours. An increase was also observed in the albumin : total protein ratio with a similar time course that was interpreted as an increase in nasal mucosal permeability. The authors suggested a non-specific, non-allergic pro-inflammatory effect when formaldehyde was inhaled at a low dose (0.14 ppm). An in-vitro study of human nasal ciliated epithelial cells showed reduced frequency of ciliary beat after exposure to 3.73 ppm formaldehyde for 2 hours, but no effect after

exposure to 3.73 ppm for 1 hour or 0.373 ppm for 2 hours (Schäfer *et al*., 1999). Formaldehyde is a well known cause of allergic contact dermatitis and is thought to act as a sensitiser on the skin (IARC, 2006).

2.2.3.4 Reproductive and developmental effects

Eleven epidemiological studies have assessed the reproductive effects of occupational exposures to formaldehyde, directly or indirectly. The outcomes observed in these studies included spontaneous abortions, congenital malformations, decreased birth weight, infertility and endometriosis. Inconsistent reports of higher rates of spontaneous abortion and lowered birth weights were reported among women occupationally exposed to formaldehyde (IARC, 2006).

2.2.3.5 Carcinogenic effect

A number of studies have found associations between exposure to formaldehyde and cancer at sites, including the oral cavity and hypo pharynx, pancreas, larynx, lung and brain (IARC, 2006).

Leukaemia

In 2004 suspicions arose that formaldehyde may cause leukaemia, but the evidence was inconsistent (IARC, 2006; Heck and Casanova, 2004). However, recent studies confirm that formaldehyde can cause blood cell abnormalities that are characteristics of leukaemia development (IARC, 2006).

Nasopharyngeal cancer

Findings from studies provided sufficient epidemiological evidence that formaldehyde causes nasopharyngeal cancer in humans (IARC, 2006; Hauptmann *et al*., 2004; Collins *et al*., 1997; Partanen, 1993; Blair *et al*., 1990).

Sinonasal cancer

The association between exposure to formaldehyde and the risk for sinonasal cancer has been evaluated (IARC, 2006; Olsen *et al*., 1984). Against these largely positive findings, no excess of mortality from sinonasal cancer was observed in other cohort studies of formaldehyde-exposed workers, including the three recently updated studies of industrial and garment workers in the USA and of chemical workers in the United Kingdom (Hauptmann *et al*., 2004; Coggon *et al*., 2003). Thus, there is limited epidemiological evidence that formaldehyde causes sinonasal cancer in humans (IARC, 2006).

2.2.3.6 Genotoxic effects

There is evidence that formaldehyde is genotoxic in multiple in-vitro models, in exposed humans and laboratory animals. Genotoxicity refers to an action on a cell's genetic material affecting its integrity. This effect may lead to mutagenic or carcinogenic effects. Studies in humans revealed increased DNA-protein cross-links in the peripheral lymphocytes of workers exposed to formaldehyde (Shaham *et al*., 2003). This is consistent with laboratory studies in which inhaled formaldehyde reproducibly caused DNA-protein cross-links in rat and monkey nasal mucosa (IARC, 2006).

2.2.3.7 Pulmonary function

Akbar-Khanzadeh and Mlynek compared 34 medical students and instructors who were exposed to 0.07–2.94 ppm (mean 1.24 ppm) formaldehyde and 12 control students and instructors who had no exposure to formaldehyde (Akbar-Khanzadeh et al., 1994). Pre- and post-morning sessions showed a 0.03% fall in forced expiry volume in 1 sec (FEV1) in the formaldehyde-exposed group compared with a 1% increase in the controls. The authors concluded that the reduction in lung function during the morning in the exposed group was most likely due to the exposure to formaldehyde (IARC, 2006).

2.2.3.8 Classification of formaldehyde

Due to the evidence that formaldehyde causes cancer, the International Agency for Research on Cancer in 2006, categorised formaldehyde in group 1 as a substance carcinogenic to humans (IARC, 2006).

2.2.4 Toluene ($C_6H_5CH_3$)

Toluene is an organic solvent and is derived from coal tar as well as petroleum. Toluene is a colourless liquid with a sweet, sharp and benzene like odours. It is also known as methyl benzene, methylbenzol, phenyl methane and toluol.

2.2.4.1 Use

Toluene can be found in gasoline and in many petroleum solvents. It is also used to produce a large number of products including tri-nitro-toluene (TNT), paint, thinners, dyes, drugs, detergents and also industrial solvents (McNary and Jackson, 2007:575). In nail salons toluene can be found in nail polish and also in nail polish removers (Patnaik, 1999:488; McNary and Jackson, 2007:575).

2.2.4.2 Exposure

Because gasoline contains 5-7 % toluene, it is a big source of atmospheric emissions and exposure to the general public (ATSDR, 2000). Inhalation is the main route of exposure in occupational settings including exposure in nail salons because of its rapid evaporation into the air. Exposure through skin contact is not uncommon (Bruckner, *et al*. 2007:1010; McNary and Jackson, 2007:573).

2.2.4.3 Toxic effects in humans

According to Bruckner *et al*. (2007:1010) after inhalation or ingestion of toluene it is very quickly absorbed from the lungs or GI tract into the blood stream. Because of the brain's high rate of perfusion and relative high fat content, the brain is negatively affected as toluene rapidly accumulates in the brain. Toluene is also deposited in other tissues, depending on the lipid content of the tissue. A portion of the inhaled or ingested toluene is well metabolized while the other portion is exhaled unchanged.

2.2.4.4 Acute toxicity

Acute toxicity of toluene is similar to that of benzene and may cause irritation of the nose, throat and eyes. These effects may be perceptible at exposure levels of 200 ppm in the air. Exposure can also produce excitement, euphoria, hallucinations, distorted perceptions and confusion. Higher levels may lead to depression, drowsiness, dizziness, light-headedness and unconsciousness. Concentrations of 10000 ppm may lead to death due to respiratory failure (Etkin, 1996:397; Patnaik, 1999:488).

2.2.4.5 Target organs

Bruckner *et al*. (2007:1010) reports that the CNS is the primary target organ of toluene. Other target organs include the kidneys, liver, skin and in some cases the heart.

Central nervous system

The symptoms that may occur after acute exposure range from slight dizziness and headaches to unconsciousness, respiratory depression and death. In some groups of occupationally exposed individuals subtle neurological effects have been described. Severe neurotoxicity has been reported in persons who have abused toluene for a prolonged period. Symptoms include inattention, apathy, memory dysfunction, lowered visuospatial skills, frontal lobe dysfunction and impaired psychiatric status (Bruckner *et al*. 2007:1010).

The effects that toluene have on the CNS are due to toluene's highly lipophilic characteristics. Toluene can therefore cross the blood-brain barrier and interact with several key brain neurotransmitters including, γ-aminobutyric acid (GABA), glycine and dopamine (McKeown, 2009).

Cardiotoxic effects

According to Ramos *et al*. (2003:280) chronic exposure to toluene can lead to a cardiotoxic manifestation namely proarrithmic effects due to its ability to lower parasympathetic activity, increase adrenergic sensitivity and altered ion homeostasis. Toluene also affects the cardiac automaticity and conduction negatively, that may lead to change in heart rate and heart rhythm. This may sensitise the myocardium to circulating catecholamines. Toluene abusers have been known to die of "Sudden sniffing death" which is secondary to cardiac arrhythmias (McKeow, 2009).

Kidney damage

Toluene can cause renal toxicity as it is a tubular toxin. Symptoms of this include, renal tubular acidosis (RTA), low concentration of potassium in the blood (hypokalemia), electrolyte disturbance, higher blood levels of urea or other nitrogen containing compounds in the blood, sterile pyuria, hematuria and proteinuria (McKeown, 2009; Kamijima *et al*. 1994:41). The toluene induced acidosis may be caused by a mechanism of decreased conductance of protons through the active transport pathway (Batlle *et al*. 1988:210).

In a recent court case in the United Kingdom a worker was compensated after being diagnosed with kidney disease because of occupational exposure to toluene. This worker was exposed to the solvent whilst working as a maintenance engineer and as a printer in a magazine printing factory (Thompsons Solicitors, 2009).

Liver effects

According to the Canadian Centre of Occupational Health and Safety (CCOHS) (2008) exposure to toluene must be very high before liver effects can be expected. Workers exposed to up to 500 ppm toluene and long-term toluene abusers show little evidence of liver damage. Some studies have reported that long-term exposure to 30-350 ppm may cause increased levels of liver enzymes that are known to be an early indicator of liver injury. Animal studies involving long-term high-level exposure (inhalation and ingestion) showed liver effects (CCOHS, 2008).

Skin effects

Dermal exposure to toluene may cause effects ranging in severity from dermatitis to extensive chemical burns with coagulation necrosis (McKeown, 2009).

2.2.4.6 Reproductive and developmental effects

Animal studies prove that toluene is a developmental toxicity hazard. Effects like reduced foetal weight, behavioural effects (effects on learning and memory) and hearing loss (in males) was evident in offspring of rats exposed (inhalation) to 1200 or 1800 ppm toluene (CCOHS, 2008).

In a review it was concluded that most studies only involved exposure to solvents in general or to certain solvent classes, with toluene exposure addressed as a co-exposure or identified as a common exposure in a sub-group. Only a few studies have specifically investigated toluene exposure and pregnancy outcomes (CCOHS, 2008). One of these studies reported that high levels of toluene exposure to pregnant woman showed growth and skeletal retardation in the offspring. The term "foetal solvent syndrome" was used to describe the dysfunctions in the children of toluene-abusing women. These dysfunctions include microcephaly and cranial facial features similar to children with foetal alcohol syndrome (Bruckner and Warren, 2003:10111).

2.2.4.7 Bone marrow damage

In an animal study the genotoxic effects of toluene on bone marrow cells of one group of male mice were studied using micronucleus test. Toluene exposure showed a dose-dependent increase in the frequency of micronucleated polychromatic erythrocytes. In a later study this genotoxic activity of toluene was confirmed in another group of the male mice (Mohtashamipur *et al*., 1958:106).

According to a review on the toxicology of toluene by OSHA (1989) it was stated that severe toluene exposure may cause a noticeable drop in the red blood cell count and partial destruction of the blood-forming elements of the bone marrow. However, other researchers report that toluene is not a bone marrow toxin.

2.2.4.8 Classification of toluene

There is no IARC classification for toluene.

2.2.5 Xylene ($C_6H_4(CH_3)_2$

Xylene, is a colourless liquid with an aromatic odour and is also known as 1,4-dimethylbenzene, para-xylene and p-xylol. Xylene is a known volatile organic compound (VOC). A VOC is an organic chemical compound that has high enough vapour pressures under normal conditions to significantly vaporize and enter the atmosphere (Bruckner and Warren, 2003:362).

2.2.5.1 Use

Xylene is a major component in gasoline and fuel oil but the primary uses of xylene in the industrial world are as solvents and synthetic intermediates (Bruckner *et al*., 2007:267). This is also the case in nail salons where xylene can be found in nail polish as a solvent.

2.2.5.2 Exposure

A large number of humans are exposed to xylene because of occupational exposure. Another source of exposure is environmental concentrations of xylene that humans are exposed to every day. The routes of exposure according to NIOSH pocket guide to chemical hazards are through inhalation, skin absorption, ingestion, skin and eye contact. In nail salons exposure to xylene is mainly through inhalation because this VOC evaporates into the atmosphere (Bruckner and Warren, 2003:267). Repeated exposure may cause accumulation in the blood and small amounts of xylene may remain stored in adipose tissue (Patnaik, 1999:491).

2.2.5.3 Toxic effects in humans

The acute toxicity of xylene is similar to other solvents including toluene and ethyl benzene. It can irritate the eyes, nose and throat and cause dermatitis. The irritation effects will occur in humans at a concentration of 200 ppm in the air. It can also cause headaches, nausea, vomiting, tiredness and lead to an upset stomach (Etkin, 1996:409; Patnaik, 1999:491).

2.2.5.4 Cellular damage and oedema

Xylene is absorbed very quickly into the blood stream after inhalation and is then distributed to various organs and parts of the body including the liver, where it is bio-transformed. A fraction of the xylene re-enters the lungs by way of the circulation and may form reactive metabolites. Covalent bonds are formed between the metabolites and macromolecules in the lungs. This causes pulmonary cellular damage and oedema (Lu and Kacew, 2002:171).

2.2.5.5 Target organs

NIOSH pocket guide to chemical hazards (2005) names the eyes, skin, respiratory system, central nervous system, gastrointestinal tract, blood, liver and the kidney target organs of xylene. Exposure to xylene (regardless of route of exposure) primary affects the nervous system. The respiratory tract is affected when xylene is inhaled while higher concentrations of ingested xylene may cause hepatic, renal and body weight effects (ATSDR, 2007).

Neurological effects

ATSDR (2007) found that neurological effects that may follow after inhalation exposure of xylene have been evaluated in many experimental studies case reports and occupational studies. After studying the results of these studies the ATSDR concluded that acute respiratory exposure to mixed xylene may cause impaired short-term memory, increased reaction time, headache, nausea, difficulty concentrating, slurred speech, gross lack of coordination of muscle movements, dizziness, fatigue, agitation, confusion, tremors, laboured breathing and sensitivity to noise and lower performance regarding numerical ability was also reported. In several isolated case reports it was found that acute respiratory exposure to mixed solvents containing xylene may lead to unconsciousness, amnesia, brain haemorrhage and epileptic seizure. The International Program on Chemical Safety (IPCS) (1992) found that the major risk from exposure to high levels of xylene can cause progressive inhibition of nervous system function that may lead to a coma, respiratory depression and eventually death from lack of oxygen to the brain.

Respiratory effects

According to ATSDR (2007) the respiratory system showed symptoms of irritation and impaired performance in tests of pulmonary function after short periods of exposure to xylene. Chronic occupational exposure to an unspecified concentration of vapours of mixed xylene has also been associated with laboured breathing and impaired pulmonary function. Nose and throat irritation have also been reported after exposure to xylene. An autopsy was done on a paint worker who died following exposure to xylene fumes (10,000 ppm for several hours) reveal that the exposure had resulted in severe lung congestion with focal intra-alveolar haemorrhage and pulmonary oedema (Morley *et al*: 1970:442).

Gastrointestinal effects

According to Uchida *et al*. (1993:597) nausea, vomiting and gastric discomfort are symptoms that have been noted in workers after exposure to xylene vapours. These symptoms stopped after termination of the xylene exposure. In another study a patient admitted to the hospital after long term inhalation of paint fumes, containing xylene, showed symptoms of anorexia and vomiting (ATSDR, 2007).

Hepatic Effects

There is limited information available on the hepatic effects of xylene after exposure. Human studies suggest that hepatic toxicity might be a result of acute-duration exposure to high levels of xylene. Other data suggest that low levels of exposure in a occupational setting does not result in hepatic effects (ATSDR, 2007).

Renal Effects

NIOSH (1981) reported no adverse effects on the kidney after exposure to *p*-xylene at 100 ppm for 5 days or up to 150 ppm in a multi-week exposure paradigm. Other data showed that occupational exposure to mixed solvents containing xylene may be associated with adverse renal effects in humans, including increased blood urea and decreased urinary clearance of endogenous creatinine (Morley *et al*, 1970:442). Other studies suggest that low-level occupational exposure to mixed xylene does not result in kidney side-effects (ATSDR 2007).

2.2.5.6 Developmental effects

According to the ATSDR (2007) information regarding developmental toxicity from occupational exposure due to xylene exposure is not definitive because of the small number of subjects and/or concurrent exposure to other chemicals. Animal studies show adverse foetal effects at high concentrations that caused maternal toxicity. These effects include delayed ossification of the skeleton, reduced foetal body weight, postnatal neurobehavioral deficits and biochemical changes in foetal and maternal brain tissue.

Brown-Woodman *et al*. (1991:139) completed an in vitro study to determine the embryo-toxicity of xylene and toluene. They found no teratogenic effect to the foetus because of these solvents. However, both xylene and toluene were embryo toxic and caused a dose-dependent retardation of growth and development of the embryos. It is important to note that the levels of these solvents that lead to embryo toxic effects were higher than blood levels likely to occur in occupational exposure or recreational abuse.

2.2.5.7 Classification of xylene

Xylene is not genotoxic or carcinogenic and thus have no IARC classification.

2.3 Previous studies

There are a number of articles written with respects to the health hazards in nail salons. These articles raise concerns regarding the health of nail technicians including, irritation of the eyes, skin and respiratory tract, dermatitis, cognitive symptoms, central nervous system depression, occupational asthma and other respiratory symptoms, liver and kidney damage, miscarriages, birth defects and cancer (Heymann, 2007:1069-1070; Sole-Smith, 2007:21-22). Some studies have been done regarding occupational exposure and health hazards in nail salons in most parts of the developed world. There is currently however a shortcoming in information available regarding the quantification of exposure to HCS and occupational health risks in South African nail salons.

Alper *et al.* (2002:41) explains that long hours of low level exposure to chemicals including organic solvents, can increase the risk of infertility, miscarriages and foetus malformation. This might also be the case when it comes to nail technicians' exposure. Another study was done regarding maternal occupational exposure to solvents and the influence it has on the child's neuro-development. This study found that exposed children performed at a lower level than control groups in subtests that measure short-term auditory memory, general verbal information and

attention. The children who were exposed to organic solvents in utero showed reduced ability in recalling sentences (Laslo-Baker *et al.*, 2004:956-951).

Other studies focused only on the health of the nail technicians. Self-reported work related symptoms of nail technicians (71) were compared with a control group (64) in a study done by Adisesh *et al.* (2008) in Great Britan. They found that the nail technicians reported a significant, increased prevalence of work-related symptoms, including nasal, neck, shoulder, wrist/hand and lower back problems. They also found that the nail technicians reported increased prevalence of work-related lower respiratory symptoms, headaches, upper back and leg and foot problems (these were not statistically significant). A similar study was done by Quach *et al.* (2008:336-343) in the U.S California. A face-to-face survey was done with 201 nail technicians. Out of all the workers, 62% reported experiencing health problems. Forty seven percent reported health symptoms associated with solvent exposure, including skin irritations, breathing problems, numbness, eye and throat irritations and 42% reported chronic pain. The report stated that the symptoms began after they commenced working in the industry.

In 1981 NIOSH and OSHA declared that long-term exposure to acrylic acid and acrylic powders used in nail salons, including MMA (IPCS, 1997), has the potential to cause lung and kidney damage. Even though this information have been known for thirty years, Adisesh *et al.* (2008) reported that only half of the 71 nail technicians were aware of advice or information discouraging the use of acrylic nail products containing MMA. This study also found that some nail technicians still used products containing methyl methacrylate (MMA) while others did not know whether the products they used contained EMA or MMA. A study done by Hemmer *et al.* (1996:377) focused on identifying relevant allergens in commercial light-curing products that contain acrylates, similar to the products used in nail salons. This was done with the help of patch tests to evaluate the efficiency of "hypoallergenic" products. They found that the omission of irritant methacrylic acid in UV-curable products did not lower the high sensitisation potential of new acrylates. This contradicted the manufacturer's declaration on hypoallergenic products that continue to include acrylate functional monomers and still leads to allergic sensitisation (Hemmer *et al.*, 1996:377).

McNary and Jackson (2007:573) designed a study to quantify the formaldehyde and toluene exposure of professional nail technicians and their customers during the application of cosmetic nail products, in the USA. The results of this study showed that neither workers nor consumers are at any additional risk from exposure to formaldehyde or toluene in cosmetic nail products beyond daily exposure from commercial products in a work setting and in the home. This study used passive (diffusion) monitors to collect organic vapour samples. Formaldehyde sampling was done using a silica-gel absorption tube connected to a high-flow pump. This was attached to a stand on the worker's desk away from the worker as not to disturb normal work operation. The samples may however not be representative of the actual amount of personal respiratory exposure to formaldehyde as the sampling tube was not in the direct breathing zone of the worker (McNary and Jackson, 2007:573).

Recommendations for further studies in nail salons were given by some of the authors of related articles recommending that further investigation of nail technicians' exposure to potentially hazardous dust and vapours be done. Investigations should also assess the effectiveness of ventilation systems for reducing exposure (Adisesh *et al.,* 2008). There is also a critical need for further investigation into the risk for breast cancer of nail technicians, their prevalent health concerns about chemicals used in salons and the high level of acute health problems that is reported in this worker group (Quach *et al.,* 2008:336).

2.4 Dermal Exposure

To formulate an accurate exposure assessment, the exposure concentrations and related dose for specific pathways must be taken into account. The goal is therefore to use the best available information and knowledge to estimate health risks for a subject population (IPCS, 2000). The human body is almost completely covered with skin, this makes the skin the largest organ of the body. As a result the skin is exposed to a number of chemicals each day and must be considered as an important route of exposure. The chemicals include environmental pollutants, cosmetics, domestic products, topical medication and industrial pollutants and in some cases chemical exposure at certain workplaces, as is the case in nail salons (Lu and Kacew, 2002:209). It is thus fortunate that healthy skin is considered to be a very good barrier and thus serve as the body's first line of defence against bacteria and other foreign molecules. However, some compounds can be absorbed through the skin and cause systemic effects. These effects may occur after a worker's skin was exposed to harmful chemicals through direct contact with contaminants, contaminated surfaces, desorption of aerosols and immersion in, or splashes from liquids (NIOSH, 2005-2007).

According to Cohen and Rice (2003) the skin comprises of two major layers, the epidermis and the dermis. For absorption to take place a contaminant must pass through the epidermis or the appendages (hairs, arrector pilli, sebaceous glands, sweat glands and nails) and then through a number of cell layers before entering the blood and lymph capillaries in the dermis. The stratum corneum is the rate determining barrier of the skin. This layer is the top layer of the epidermis and is packed densely with keratinized cells.
The most apical layers of these cells are dead and lose their nucleus and cytoplasm, but contain alternatively a tough, resistant protein called keratin. This specialisation makes the stratum corneum the single most important barrier regarding prevention of fluid loss from the body while also serving as a major barrier to prevent absorption into the body (Rozman and Klaassen, 2003:59). The second phase of dermal absorption is diffusion of toxicant through the dermis. The dermis is situated beneath the epidermis and contains a porous, non-selective, aqueous diffusion medium. This layer also contains the blood supply and serves to carry absorbed compounds into the body. The dermis is therefore much less effective as a barrier than the stratum corneum (Lehman-McKeeman, 2007:131; Lu and Kacew, 2002:209). If an insult is severe or very intense it can overwhelm the protective function of the skin and acute or chronic injury becomes readily manifested in various ways (Rice and Mauro, 2007:741).

2.4.1 Health effect of dermal exposure

NIOSH named skin diseases as one of the most pervasive occupational health problems. Skin conditions resulting from exposure to consumer products or occupational exposure are not always reported and do not necessarily result in work time loss. Because of this, skin conditions are poorly recorded and tracked. The incidence of skin diseases therefore appears to be greatly underestimated. The recording of occupational skin diseases depends largely on the specific country's national regulation and compensation systems (European Agency for Safety and Health at Work, 2008:16; Rice and Mauro, 2007:741). According to OSHA (2008) dermal exposure in the occupational setting is a major problem in the United States of America. The number of cases and the rate of skin disease in the United States exceed recordable respiratory illnesses. Studies have shown that chemicals are absorbed through the skin without the worker noticing and are therefore sometimes overlooked as an exposure route. OSHA (2008) also explains that dermal absorption is a more significant route of exposure than the lung. This is mostly the case when working with non-volatile chemicals which are relatively toxic and remain on work surfaces for long periods of time.

According to NIOSH (2005-2007), skin diseases that may occur after dermal exposure is known as skin irritation and corrosion, both is confined to exposed areas. Irritant contact dermatitis, sensitisation of the skin and allergic contact dermatitis may occur. In some cases sensitisation of the respiratory tract may also be a result of skin exposure. It is now clear that dermal exposure and absorption can potentially contribute to systemic toxicity or to overall or total exposure to a chemical(s).

2.4.1.1 Irritant contact dermatitis

Occupational irritant contact dermatitis is an inflammatory reaction caused by direct contact of irritating chemicals with the skin in an occupational setting. Inflammation usually occurs either immediately or within a short period of time. Irritant contact dermatitis results in redness of the skin, blisters, scales or crusts. Not necessarily all exposed workers will have these symptoms and the symptoms do not necessarily occur at the same time. Irritating chemicals include acids, bases and fat-dissolving solvents (CCOHS, 2008; NIOSH, 2005-2007).

This condition can be treated with creams, ointments and skin cleansers. It is also recommended that the affected area should be protected from physical trauma, chemical irritation, excessive sunlight, wind and rapid temperature changes while the dermatitis is active (CCOHS, 2008).

2.4.1.2 Allergic contact dermatitis

According to NIOSH (2005-2007) this condition is an immunological mediated reaction of the skin caused by direct contact of the skin after sensitisation has occurred to a chemical that is an allergen.

Allergic contact dermatitis develops in stages. The first stage is the period during which an individual may be repeatedly in contact with the allergen without developing any skin reaction. This stage can last a lifetime or only a few days. During this stage the allergen is changing the composition of the skin and causes the skin to lose some of its "barrier" characteristics (NIOSH, 2005-2007).

The skin then becomes sensitised after the allergenic chemical has penetrated the outer layer of the skin. The penetrated allergenic substance then combines with the skin's natural proteins. This combination is then carried throughout the body by the lymphocytes in the blood. When re-exposure occurs, lymphocytes recognize the allergen and react with it. During this stage a tissue-damaging chemical called lymphokines are released which leads to itching, pain, redness, swelling and the formation of small wheals or blisters on the skin. The blisters may break, forming crusts and scales. Untreated, the skin may darken and become leathery and cracked. This reaction is usually confined to the area of exposure but it is not uncommon in severe cases to spread to cover large areas of the body (American Osteopathic College of Dermatology, 2009; Cohen *et al*, 2003:288-300).

The allergic sensitisation may stay with the individual through his/her life but the level of sensitivity may lower if there is no further contact with the allergen (CCOHS, 2008; American Osteopathic College of Dermatology, 2009).

Treatment of allergic contact dermatitis includes symptomatic control of itching with oral antihistamines. Medium- and high-potency topical steroids may also be prescribed for rashes occurring on the extremities or trunk and mild-potency topical steroids will be prescribed for thinner skin like on the face and skin fold areas. In severe cases involving large body areas it may be necessary to take a 14-day course of an oral steroid (prednisone) (Skinsight, 2008).

2.4.1.3 Systemic Toxicity

Systemic toxicity may occur after dermal absorption of toxic chemicals which are then transported to other sites in the body where their toxic effects occur (NIOSH, 2005-2007).

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CHAPTER 3

ARTICLE:

Respiratory exposure and potential dermal exposure to volatile organic compounds in nail salons: a pilot study

Respiratory exposure and potential dermal exposure to volatile organic compounds in nail salons: a pilot study

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Abstract

Objectives: The aims of this pilot study were to quantify respiratory and potential dermal exposure of nail technicians to acetone, formaldehyde, acrylic monomers, toluene and xylene. Fifteen female nail technicians, working in different salons participated in this study. Most salons used acrylate based nail products whereas others used UV-gel products exclusively. **Methods:** The participants were divided into two groups, those who used acrylate- and those who used UV-gel products exclusively. Eight hour personal respiration exposure to acetone, formaldehyde, ethyl methacrylate, methyl methacrylate, toluene and xylene were determined. The concentration of airborne volatile organic compounds in the salons was also determined with the use of a direct reading instrument (EntryRAE). Potential dermal exposure to the above mentioned solvents (excluding formaldehyde) was determined with the use of charcoal pads (surrogate skin method). During respiratory and dermal sampling, observations were made regarding work practices and control measures used in the salons. **Results:** It was found that the eight hour time weighed average exposure is well below the recommended occupational exposure limits of the individual chemicals and showed no additive effect. The highest mean respiratory exposures in both groups were acetone (27.22 mg/m^3 and 28.36 mg/m^3). EntryRAE results showed peak periods of exposure to volatile organic compounds during the day (322.16 ppm) that were much higher than the average eight hour exposure (0.21 ppm). The two groups' exposure levels were compared to determine if there is a significant difference between the exposures levels but no statistically significant difference was found. The dermal exposures on hand and neck to acetone, ethyl methacrylate and methyl methacrylate showed strong significant correlations to the concordant chemical's respiratory exposures. Correlations between air and dermal exposure was calculated once more after adjusting dermal exposure but the findings indicated only one statistically significant correlation of 0.42 in the case of ethyl methacrylate. **Conclusion:** Nail technicians are not at immediate health risk as the exposure in nail salons are below recommended occupational exposure limits. However the unknown effects of chronic low level exposure to solvents and the large number of previous studies that reported increased health risks in nail technicians must also be considered. The use of methyl methacrylate in nail products sold in South Africa is also worrying as methyl methacrylate is banned by the FDA in the US due to its skin sensitisation potential that may lead to allergic contact dermatitis. The methods used to determine potential dermal exposure as well as adjusted dermal exposure remains problematic. This is due to the high percentage of adjusted dermal exposure values that had to be estimated and the fact that the activated charcoal pads have a higher absorption potential than human skin. Both methods must be improved to increase accuracy of results. Observations and EntryRAE results demonstrated the

irregular nature of a nail technician's work shift as well tasks performed from day to day. This complicates gathering data that is representative of a nail technicians eight hour exposure. Therefore to further improve accuracy of results, sampling should in future be task specific.

Keywords: nail salon, dermal exposure, respiratory exposure, charcoal cloth pads, volatile organic compounds.
Word Count: 4,166

Introduction

The desire for beautiful nails is a universal phenomenon amongst men and women giving rise to a rapidly growing industry (Heymann, 2007). According to Schoon (2005) nail enhancements are formed by coating the natural nail with a hard coating. Typical current nail enhancement systems consist of liquid and powder acrylic systems, ultra violet (UV) gels and no-light gels. Schoon (2005) explained that there are an assortment of products that may be used in salons. They all share the characteristic that the basic ingredients (monomers or oligomers) undergo a polymerisation reaction to produce a hard acrylic polymer coating, the nail enhancement. Energy is required to stimulate the chemicals and trigger the polymerisation reaction. Some systems require light energy (e.g. UV light) while others need thermal energy (e.g. room temperature or body heat.). Liquid and powder systems involve combining a liquid monomer (such as ethyl methacrylate monomer, EMA) with a powdered polymer (typically poly methyl and/or ethyl methacrylate) that contains the reaction initiator and other ingredients, such as colorants. The most commonly used polymerising enhancement products can be divided into three main categories by the polymerising chemicals they contain, namely: cyanoacrylates (wraps, no-light gels, tip adhesives), methacrylates (monomer and polymer, UV nail enhancements) and acrylates together with methacrylates UV nail enhancements. Nail polishes (also known as nail varnishes, enamels, lacquers), topcoats and other nail treatments differ from enhancement systems because they form a hard coating by evaporation of solvents and do not polymerise (Schoon, 2005; Newman, 2007).

It is well known that nail technicians are exposed to several hazardous chemical substances (HCS) during the application of artificial fingernails (Roelofs and Tuan, 2007; OSHA, 1993). These HCS include formaldehyde, toluene, acetone, EMA, methyl methacrylate (MMA), xylene and several phthalates (Quach *et al.*, 2008). To date, a number of articles have been published about the health hazards in nail salons and the associated health effects, these are summarised in Table 1.

Table 1: Hazardous chemical substances present in nail salons and their associated health effects
(Heymann 2007; Sole-Smith 2007; LoSasso *et al.*, 2001; Oregon OHSA 2008; Alper 2002).

Health effects	**HCS responsible**
Eye, skin and respiratory tract irritation	Formaldehyde, EMA, MMA, Phthalates, Acetone, Toluene, Xylene, Methyl ethyl ketone
Skin Irritation and dermatitis	EMA, MMA, Acetone, Toluene, Xylene, Methyl ethyl ketone
Cognitive symptoms (affecting memory and learning)	Acetone, Toluene, Xylene, Methyl ethyl ketone
Central nervous system depression (headaches, nausea, dizziness and irritability)	Acetone, Toluene, Xylene, Methyl ethyl ketone
Occupational asthma	Formaldehyde, MMA
Liver and kidney damage	Mixed solvents
Miscarriages	Dibutyl Phthalate, Toluene
Birth defects	Toluene
Infertility	Dibutyl Phthalate

In the past, occupational exposure to volatile chemicals was only measured by means of respiratory exposure. In recent years it has however become apparent that dermal absorption of chemicals may also be a major contributor to exposure (Fenske, 1993). Niedner (2008) explained that the hands are the primary area of the body affected by occupational hazards, including exposure to solvents. Occupational skin conditions on the hands account for 90-95% of all occupational related skin diseases registered each year. It is therefore important to quantify skin exposure in order to optimally prevent the occurrence of skin diseases (Niedner, 2008). Initially, quantification of dermal exposure to volatile organic compounds was done indirectly through bio- monitoring of urinary metabolites. This method is sufficient when estimating systemic exposure but not adequate when evaluating different exposure pathways (Vermeulen *et al*., 2006). More recently, dermal exposure to volatile chemicals has been measured with the use of activated charcoal patches as first described by Cohen and Popendorf in 1989. According to Semple (2004), exposure measured by patches must be referred to as "potential dermal exposure". The reason for this is because patches measure the total amount of material deposited over a selected area and therefore provide an estimation of the total dermal exposure. This method has been used in a number of occupational settings and has proven to be a useful method for determining potential dermal exposure to chemicals including benzene, toluene, styrene, terpenic resin acids and monoterpenes (Vermeulen *et al*., 2006; Van Wendel De Joode, *et al*., 2005; Lindsay *et al.*, 2006; Eriksson and Wiklund, 2004a). However, there are currently no dermal occupational exposure limits (OEL) for any hazardous chemical substances (Du Plessis *et al*., 2010) while numerous respirable OEL values are available. The OELs of the airborne hazardous chemical substances (HCS) quantified in this study can be seen in Table 2. These OELs are specified by the Occupational Health and Safety Act, 1993 (Regulations for Hazardous Chemical Substances), the National Institute of Occupational Hygiene (NIOSH) and the Occupational Safety and Health Administration (OSHA).

Table 2: Occupational Exposure Limits of Hazardous Chemical Substances found in nail salons

	Regulations for HCS, 1995-Annexure 1.					**NIOSH**		**OSHA**	
	Notes	**TWA OEL-RL**		**Short Term OEL-RL**		**TWA**		**TWA**	
		ppm	**mg/m^3**	**ppm**	**mg/m^3**	**ppm**	**mg/m^3**	**ppm**	**mg/m^3**
Acetone	-	750	1780	1500	3560	250	590	1000	2375
***Formaldehyde**	-	2	2.5	2	2.5	0.016	0.0197	0.75	0.923
MMA	-	100	410	125	510	100	410	100	410
EMA	-	-	-	-	-	-	-	-	-
Toluene	SK	50	188	150	560	100	375	200	754
Xylene	SK	100	435	150	650	100	435	100	435

SK = Skin absorption

*OEL-Control Limit

A NIOSH study conducted personal and area air sampling of methyl and ethyl methacrylate, acetone, and benzene in a nail salon in Ohio, Springdale. They found no methyl methacrylate or benzene vapours in the salon but found low

concentrations of ethyl methacrylate (ranged from non-detected to 7 ppm) and acetone vapours (6 and 10 ppm) (Decker and Beasley, 1992). Another study, quantified exposure to HCS's in nail salons with the use of eight hour general-area air sampling of HCS's, formaldehyde, and methacrylates, along with personal breathing-zone (PBZ) air sampling for methacrylates. The results indicated that acetone, *n*-butyl acetate, ethyl acetate, ethyl methyacrylate, toluene, and 1,1,1-trichloroethane were the major chemical compounds present in the air. Samples collected specifically for airborne methacrylates detected, ethyl methacrylate on all nine samples collected but no methyl- and isobutyl methacrylate. Short-term PBZ air samples collected during the application of acrylic monomer liquid and polymer powder showed ethyl methacrylate concentrations of 128 mg/m^3 (7-minute sample) and 78.9 mg/m^3 (14-minute sample) (Almaguer and Blade, 1992). Both of these studies found the exposure to be below the recommended NIOSH OEL's.

Other studies reported a statistically significant increased prevalence of work-related nasal symptoms and elevated levels of work-related lower respiratory symptoms and headaches in nail technicians compared to control groups. A correlation between spontaneous abortion and the number of hours worked per day as nail technician, the number of chemical services performed per week and work in salons where nail sculpturing was performed by other employees have also been found (John *et al.,* 1994; Adisesh *et al.,* 2008). Despite this and other possible health effects of long-term low level exposure to organic solvents (Bruckner and Warren, 2003), Regulations on HCS are seldom implemented in salons and it is not unusual to find nail technicians in salons working on a client's nails without proper ventilation or any personal protection equipment (Porter, 2009). This is due to the perception that nail salons are not an industrial setting.

Currently there is no information regarding the assessment of exposure to HCS and occupational health risks in South African nail salons. The purpose of this pilot study is to quantify respiratory and potential dermal exposure of nail technicians to acetone, formaldehyde, ethyl methacrylate (EMA), methyl methacrylate (MMA), toluene and xylene.

Methods

Participants. Fifteen female nail technicians between the ages of 20 and 50 years, working in different salons situated in Potchefstroom (five salons) and Klerksdorp (four salons) participated in the study. All of these participants gave informed consent.

Air sampling. Eight hour (full shift) airborne exposure to acetone, formaldehyde, EMA, MMA, toluene and xylene of the volunteers were assessed. Formaldehyde was sampled by means of NIOSH method 2541, using solid sorbent tubes (SKC 226-118). Acetone, EMA, MMA, toluene and xylene were sampled by using one solid sorbent tube (SKC 226-01) according to NIOSH method 1501. Personal air sampling pumps were attached to the nail technicians' waist belt. Flexible tubing connected the pumps to both sorbent tubes using dual adjustable low flow tube holders (SKC 224-26-02). The pumps and the dual adjustable low flow tube holders were calibrated prior to measurements, at flow rates of

0.1 L/min (SKC 226-118) and 0.2 L/min (SKC 226-01) for the sorbent tubes respectively. The sorbent tubes were placed in the breathing zone of the nail technicians.

Formaldehyde sorbent tubes were stored at room temperature while the other sorbent tubes were stored at 5°C. All sorbent tubes (including field blanks) were sent to an accredited analytical laboratory for analysis, by using a Gas Chromatograph equipped with a Flame Ionization Detector (FID).

Area samplings. EntryRAEs (RaeSystems, USA) were setup to use the modular photoionisation detector (PID) sensor for continuous detection of volatile organic compounds (VOC) using a 10.6eV lamp (range 0-999 ppm). According to the manufacturer the sensor is able to detect a large number of VOCs including acetone, toluene and xylene but cannot detect EMA or MMA. The EntryRAEs were connected to flexible tubing and then placed on a stand, located on the working surface near the technician approximately 1.5 m above floor level. EntryRAEs detected VOC concentraions during the same eight hour shift as the air and dermal sampling. After sampling, the results were studied and the fifteen minutes out of every eight hour shift showing the highest average level of exposure to VOCs (ppm) were selected.

Dermal sampling. A surrogate skin method was implemented for dermal sampling. PERMEA-TEC pads® (SKC 769-3050) were used to determine potential dermal exposure to solvents. These charcoal pads included both direct-reading colorimetric detectors (normal sensitivity range, from 0.5 to 5 mg) and a micro-encapsulated solvent indicator system. Sampling took place during the same eight hour shift as the air sampling. One PERMEA-TEC pad® was placed on top of the technician's right hand and a second pad was placed on the neck, representing the breathing zone. Two PERMEA-TEC pads® were used as blanks. After sampling, the charcoal pads were removed from the backing and stored in tightly sealed glass vials at 5°C before analysis by an accredited lab. A Gas Chromatograph equipped with a FID was used to analyse the charcoal pads. PERMEA-TEC pads® may also capture airborne vapours, therefore, dermal exposure on the hand was calculated by subtracting the amount of HCSs measured on the neck area, from the amount of HCSs measured on the hand. This is referred to as adjusted dermal exposure (Vermeulen *et al.*, 2006:1148). Adjusted dermal exposure values at or below zero were set at two-thirds of the smallest positive adjusted dermal exposure value as suggested by Van Wendel De Joode *et al*., (2005).

Observations. During sampling, observations were made regarding work practices in use, ventilation and also control measures used in the different salons.

Statistical analysis

The fifteen nail technicians were divided into two groups. The Acrylate Group consisted of nine technicians who worked with acrylic and gel nail products. The Non-Acrylate Group included six nail technicians who worked with gel nail products exclusively. A substitution method (Ogden, 2010) was used to replace the results below the level of

detection. Non-Acrylate Group's exposure to EMA and MMA were replaced with zero except in the case of the two technicians who had been exposed to acrylates indirectly, in all other cases the results below detection level (BDL) was replaced with D/2 (D = lower analytical detection limit of the chemical).

Statistical analysis of both groups' exposure was performed using Statistica Version 9.0 (Statsoft Inc., 1984-2009). Spearman rank order correlations were obtained for dermal and respiratory measurements. The correlations was used to investigate the relation between dermal and respiratory exposure, as well as between the hand and neck exposure areas. The Mann-Whitney-U test was used to determine if the exposures of the two groups differed significantly from each other.

Results

During observations it was noted that all of the technicians worked in a seated position with the respiratory zone ± 30 cm – 40 cm from the source (chemicals used) of exposure while doing nail sculpturing. Time spent on nail sculpturing differed greatly between participants. Some nail technicians had a fully booked day whilst others had cancelations and time off. Not all of the salons who participated in this study are exclusive nail salons with some participants spending time on other beauty-related treatments for clients during the sampling period. These treatments did not involve the use of HCSs.

Only one of the fifteen participants used gloves to protect her hands from potential dermal exposure. This was due to a previous allergic reaction she experienced whilst working with acrylic nail products. This participant however chose, out of free will, not to wear the gloves on the day of sampling. No other form of PPE was found to be in use at any of the other salons.

The dermal and respiratory exposure samples of both groups (Acrylate- and Non Acrylate- Groups) showed detectable levels of acetone, EMA, MMA, toluene and xylene (excluding exposure to acrylates in salons working exclusively with gel products). The HCS with the highest mean respiratory exposure in both groups was acetone with concentrations of 27.22 mg/m^3 in the Acrylate Group and 28.36 mg/m^3 in the Non-Acrylate Group. Acetone was also the HCS with the highest mean potential dermal exposure, except in the case of hand exposure in the Acrylate Group where EMA had the highest exposure (73.82 μg/cm^2). The respiratory- and potential dermal exposure of the two groups is summarised in Tables 3 and 4. Each of the nail technicians' eight hour respiratory exposure levels to the HCS's were below the TWA Occupational Exposure Limits of the South-African Regulations for Hazardous Chemical Substances (Table 2).

Table 3: Acrylate Group- TWA exposure to HCS (n=9)

Respiratory exposure	Exposure hand	Exposure neck

	(mg/m^3)	(µg/cm^2 8h)	(µg/cm^2 8h)
HCS	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**
Acetone	27.22 (24.54)	49.80 (48.08)	38.10 (36.16)
EMA	7.09 (5.90)	73.82 (67.58)	35.06 (28.94)
MMA	0.05 (0.06)	0.44 (0.53)	0.22 (0.23)
Toluene	0.09 (0.06)	1.96 (0.10)	3.00 (3.84)
Xylene	0.02 (0.02)	0.42 (0.25)	0.81 (1.42)
Formaldehyde	0.19 (0.12)		

SD – Standard Deviation

Table 4: Non-Acrylate Group - TWA exposure to HCS (n=6)

	Respiratory exposure (mg/m^3)	Exposure hand (µg/cm^2 8h)	Exposure neck (µg/cm^2 8h)
HCS	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**
Acetone	28.36 (22.42)	74.85 (77.4)	61.66 (42.37)
EMA	0.01 (0.01)	0.02 (0.02)	0.02 (0.02)
MMA	0.002 (0.003)	0.03 (0.05)	0.02 (0.04)
Toluene	0.06 (0.02)	1.26 (0.88)	1.49 (1.01)
Xylene	0.02 (0.02)	0.79 (0.56)	0.55 (0.23)
Formaldehyde	0.21 (0.06)		

SD – Standard Deviation

Both groups' potential dermal exposure levels of acetone, MMA and Acrylate Group's exposure to EMA were higher on the hand but dermal exposure levels of toluene were higher on the neck. The Non-Acrylate Group's exposure to EMA on hand and neck were both 0.02 $\mu g/cm^2$. The dermal levels of exposure to xylene differed between groups, with the Acrylate Group's exposure on the neck being higher than their hand exposures, but the inverse was true for the Non-Acrylate Group.

Most threshold limit values and OELs are developed for a single chemical substance but, when two or more HCSs in a mixture have similar toxicological effect on the same target organ or system, their combined or "additive" effect should also be considered (Molander *et al*., 2006). According to the Regulations for HCS, 1995-Annexure 1 this could be done with the help of the following equation:

If the sum of $C_1/L_1 + C_2/L_2 + ... C_n/T_n > 1$ the threshold of the mixture is exceeded (where C_1 indicates the measured TWA concentration and L_1 the corresponding exposure limits). Because all of the HCS's measured in this study are known to cause eye, skin and respiratory tract irritation the additive effect was calculated. The sum of C_n/L_n fractions equalled 0.11 in the Acrylate Group and 0.1 in the Non-Acrylate Group. Therefore neither of the two groups' exposures showed any additive effect.

The EntryRAEs' results also showed detectable concentrations of VOC's in all the salons. Fifteen minutes with the highest average concentration out of the eight hour VOC air concentrations measured were selected for each of the fifteen nail technicians. The average eight hour exposure in the Acrylate Group ranged from 0.092 ppm to 46.12 ppm and the fifteen minute average exposure ranged from 1.93 ppm to 322.16 ppm. The Non-Acrylate Groups' average eight hour exposure range from 0.01 to 13.32 ppm and the fifteen minute average exposure ranged from 0.21 ppm to 77.83 ppm. Fig 1 (a) and Fig 1 (b) show that the average eight hour VOC concentration in the air is lower than the fifteen minutes of highest exposure of each nail technician. The figures also show that the Acrylate Group's minimum and maximum 8 hour and fifteen minute exposures are higher than those of Non-Acrylate Group

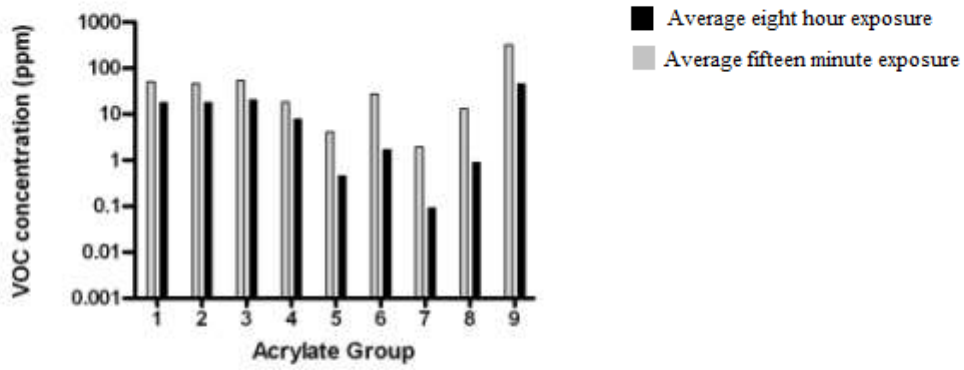

Fig 1 (a) : Average 8 hour and highest 15 minutes VOC air concentration according to the EntryRAE results for each of the nail technicians.

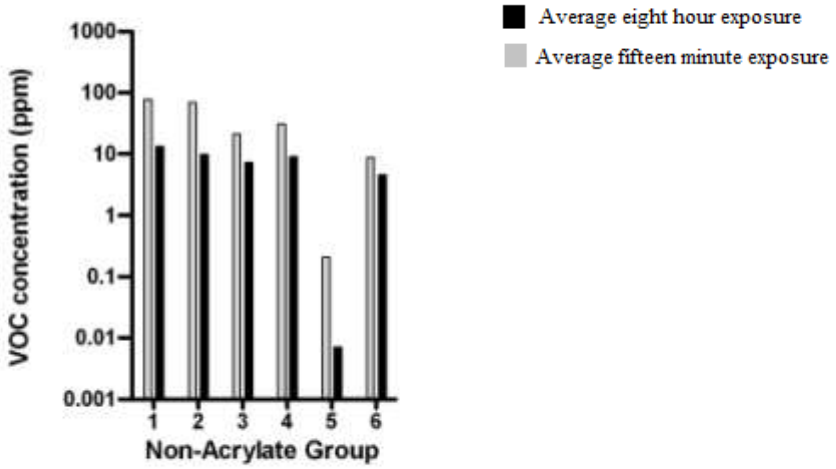

Fig 1 (b) : Average 8 hour and highest 15 minutes VOC air concentration according to the EntryRAE results for each of the nail technicians.

To determine if the two groups' exposures to the HCSs differed significantly from each other, a statistical method, Mann-Whitney U tests, was performed. The results of this test in Table 5 show that there were no significant differences between the groups' exposure levels to acetone, toluene, xylene or formaldehyde. Significant differences were found between the two groups' exposure to acrylates.

Table 5: Mann-Whitney-U test with Acrylate and Non-Acrylate Groups as variables to determine significant difference between exposures of groups

Marked tests are significant at $p < 0.05$

HCS	Exposure Air	Exposure Hand	Exposure Nek
Acetone	0.96	0.53	0.46
EMA	**0.00**	**0.00**	**0.00**
MMA	**0.01**	**0.02**	**0.01**
Toluene	0.22	0.27	0.53
Xylene	0.95	0.18	0.27
Formaldehyde	0.07	-	-

To establish the strength of the general relationship between the two routes of exposure (respiratory and dermal hand and neck) to each chemical Spearman's rank order correlation coefficients was used (Table 6). This statistical analysis will not distinguish between the two groups. A strong statistically significant relationship was found in the case of EMA followed by acetone and then MMA. Xylene has the weakest correlations between all the routes of exposures followed by toluene.

Table 6: Spearman rank order correlations between routes of exposure

Marked values are significant at $p < 0.05$

	r-values of correlations between:		
	Hand and Neck	**Neck and Air**	**Hand and Air**
Acetone	**0.86**	**0.89**	**0.74**
EMA	**0.94**	**0.93**	**0.91**
MMA	**0.67**	**0.77**	**0.86**
Toluene	**0.65**	0.24	-0.02
Xylene	0.03	**0.40**	-0.27

To determine the possible contribution of non-vapour exposure sources coming into contact with skin, such as chemicals in dust and drops of liquid products used in salons, the potential dermal exposure levels was adjusted. Adjustments were done by subtracting individual HCS' exposure measured on neck from the corresponding HCS' exposure measured on hand. Values at or below 0 after adjustment, were set at two-third of the smallest positive adjusted dermal exposure value (Van Wendel De Joode *et al.*, 2005). Of the adjusted dermal exposure values 38.6 % of the values were below zero and had to be given an estimated value. After adjusting the dermal exposure levels at the hand it was found that the Non-Acrylate Group had the highest adjusted dermal exposure to acetone, toluene and xylene as seen in Table 7.

Table 7: Adjusted dermal exposure values

	Acrylate Group (µg/cm^2 8h)	**Non-Acrylate Group (µg/cm^2 8h)**
HCS	**Mean (SD)**	**Mean (SD)**
Acetone	13.04 (12.65)	26.17 (41.72)
EMA	43.50 (49.54)	*0.0 (0.0)
MMA	0.30 (0.33)	0.03 (0.04)
Toluene	0.50 (0.46)	0.79 (0.15)
Xylene	0.16 (0.23)	0.52 (0.42)

Note:

* Adjusted dermal exposure of EMA in the Non-Acrylate Group is 0.021

After adjusting the dermal exposure the relationship between the two routes of exposure (respiratory and dermal) was determined for a second time by using Spearman's rank order correlation coefficients. As seen in Table 8 the adjusted dermal exposure and the respiratory exposure to EMA shows a statistically significant correlation of 0.42.

Table 8: Spearman rank order correlations between respiratory and adjusted dermal exposure

Marked values are significant at $p < 0.05$

	Correlation r-values
Acetone	0.26
EMA	**0.42**
MMA	0.38
Toluene	-0.45
Xylene	-0.35

Discussion

After quantifying exposure in nail salons it is clear that the TWA eight hour exposure is well below the Recommended OEL and action levels of the individual HCSs. It was also determined that exposure to the mixture of HCSs will not cause an additive effect. The average formaldehyde respiratory exposure of both exposure groups exceeded the NIOSH TWA-OEL (0.0197 mg/m^3) but is never the less below the Regulations for HCS (1995-Annexure 1) and OSHA TWA-OELs. The nail technicians are therefore not at immediate risk due to respiratory exposure. There is however an ongoing debate regarding the effects (if any) of chronic low-level exposure to virtually any solvent or mixed solvents. It is suspected that it might be the cause of neurologic dysfunctions, also known as: painter' syndrome, organic solvent syndrome, psycho-organic syndrome and chronic solvent encephalopathy (CSE) (Bruckner and Warren, 2003).

McNary and Jackson (2007) designed a study to quantify the formaldehyde and toluene respiratory exposure of professional nail technicians during the application of cosmetic nail products, in the USA. They also found the exposure levels to be below the Recommended TWA exposure levels. Lower average levels of exposure to formaldehyde (0.03 mg/m^3) and higher levels of exposure to toluene (0.89 mg/m^3) were found compared to this study. The differences between the exposures found could be attributed to the differences in procedures and ingredients used during the manufacturing processes of the products used in salons. Two similar investigative studies regarding exposure in nail salons were done by NIOSH (Almaguer and Blade,1992; Decker and Beasley, 1992). Both studies found no respiratory exposure to MMA vapours. This is ascribed to the ban on MMA by the Food and Drug Administration (FDA in the USA), in all artificial nail preparations because of its inclination to cause allergic contact

dermatitis. Regardless of the health risk that MMA impose, it is still allowed to be used in South African products. After the ban on MMA in nail products in the USA it was replaced with EMA (Lewis, 1998). The first NIOSH study led by Decker and Beasley (1992) found EMA concentrations ranging from BDL to 32.67 mg/m^3. This NIOSH studies found higher concentrations of EMA compared to this study's results of BDL to 24.3 mg/m^3 whilst Almaguer and Blade (1992) detected concentrations ranging from 10.31 mg/m^3 to 14.1 mg/m^3. Decker and Beasley (1992) also detected lower levels of acetone vapours ranging from 14.42 mg/m^3 to 23.75 mg/m^3 compared to 0.4 mg/m^3 to 66.48 mg/m^3 found in this study.

EntryRAE results in Figure 1 (a) and (b) showed that there are peak periods of exposure during the day that are much higher than the average 8 hour exposure. This can be attributed to the fact that there are occasionally long periods in the day that the technician has no exposure due to cancelations, time off or time performing other beauty related treatments offered by salons. This explains the very low minimum eight hour HCS exposure of nail technician no. 5 (Fig 1b).

Observations showed that this specific technician had only one nail sculpturing client during that day. The high maximum fifteen minute exposure of nail technician no. 9 to VOC's (Fig 1a) can be explained by the specific task performed during those fifteen minutes. Namely the technician was soaking her client's nails in a bowl of acetone to remove the old acrylic-overlays from the client's natural nails.

No statistical difference in exposure levels (neck, hand and air) between the Acrylate and non-Acrylate Group was found, except in the case of EMA and MMA as expected. This was also the case in a study done by Molander *et al.*, 2006.

Dermal exposure results proved to be a contributing factor when quantifying the total exposure to HCS. The potential dermal exposure on hand and neck to acetone, EMA and MMA showed strong correlations to the concordant chemical's respiratory exposure. It can thus be concluded that if the respiratory exposure increases, the dermal exposure will also increase. The vapour pressure (VP) and vapour density (VD) were suspected to be cause of the weak correlation of xylene and toluene between the routes of exposure. After investigation it was found not to be the case. The VPs of the HCSs that showed strong correlation between air and dermal sampling are between 2 and 24 kPa at 20°C and their VDs are between 2 and 3.9 (where air = 1). The VPs and VDs of the HCSs with weak correlations between air and dermal sampling also lay within the above mentioned ranges, except for the VP of xylene that is slightly below range (1.19 kPa at 20 °C).

Weak correlations between adjusted dermal exposure and respiratory exposure to acetone and MMA were found. However a statistically significant correlation was found with EMA exposure. Toluene and xylene showed weak inverse correlations between the routes of exposure. These weak correlations can be explained by the accidental or random manner in which adjusted dermal exposure occurs. The adjusted dermal exposure is the result of spillage and spatter of the products used in nail salons and is not influenced by concentrations of HCSs in air.

Perfume used by nail technicians may also have influenced the potential dermal exposure results, as it contains a number of VOCs including acetone (Boudreau *et al*., 1992). During observations it was noted that nail technicians washed their hands between clients while wearing charcoal pad and some applied moisturiser on hands thereafter. These actions may also have influenced potential dermal exposure results.

The high percentage of adjusted dermal exposure values that had to be estimated show that a large part of the unadjusted dermal exposure on the hand can be attributed to vapours and not only direct contact to HCS (in dust and in spattered drops of products used by the nail technicians). The difference in proximity from the source could also contribute to the difference in the exposure measured on the hand and neck. Adjusting the dermal exposure might therefore lead to an underestimation of the contribution of dermal exposure to HCSs (Van Wendel De Joode *et al.*, 2005:49).

Conclusion

After measuring the HCS exposure of nail technicians working with acrylate and UV gel products it can be concluded that nail technicians are not at immediate health risk as the exposure in nail salons are well below recommended OELs and action levels. Regardless of this, it cannot be concluded that nail technicians are at no health risk due to occupational exposure. The unknown effect of chronic exposure to low levels of solvents and the large number of previous studies that reported increased health risks in nail technicians must also be considered (John *et al*., 1994; Adisesh *et al.*, 2008, Bruckner and Warren, 2003). Another worrying fact regarding the health of South African nail technicians is the use of MMA in nail products as it was detected in dermal and air samples. MMA has been banned by the FDA in the USA due to its potential to cause skin sensitisation that may lead to allergic contact dermatitis (Lewis, 1998). The use of gloves while working with cosmetic nail products that contain MMA is proposed to prevent sensitisation to MMA as well as the occurrence of contact dermatitis.

NIOSH named skin diseases as one of the most pervasive occupational health problems. Absorption of compounds through skin does not only lead to skin conditions but can also cause systemic effects (NIOSH, 2005 to 2007). It is therefore important to quantify the occupational exposure of skin. When taking the high percentage of adjusted dermal exposure values that had to be estimated (38.6%) into account and the fact that the activated charcoal pads has a higher absorption potential than human skin (Eriksson and Wiklund, 2004b; Vermeulen *et al.*, 2006) it becomes apparent that the use of carbon pads as method for measuring potential dermal exposure remains problematic. This method as well as the method used to adjust the results must also be improved to increase accuracy of results.

To improve accuracy of results in future studies, focus must shift to short term exposure for the duration of different tasks performed during nail sculpturing. Task specific sampling is important to establish worst case scenario exposure in nail salons. Observations and EntryRAE results verified the irregular and unpredictable nature of a nail technician's work shift as well as the variability in tasks performed from one day to the next.

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CHAPTER 4

CONCLUDING CHAPTER

General Summary

The nail enhancement industry is rapidly growing throughout the world (Molander *et al.*, 2006: 537-542). It is abundantly clear from previous studies that a wide variety of hazardous chemical substances (HCS) (mostly solvents) are used during nail treatments (Roelofs and Tuan, 2007; OSHA, 1993; NIOSH Pocket Guide to Chemical Hazards, 2005). Consequently a large number of, primarily women, working as nail technicians in nail salons are exposed to a range of hazardous chemicals each day.

To understand the potential health risks involved when working as a nail technician, the toxicology of selected HCS found in cosmetic nail products were researched (Capital Health Services, 2007:1-4). These HCS include acetone, acrylic monomers (MMA and EMA), formaldehyde, toluene and xylene. It was found that all of these chemicals have the potential to cause irritation to the nose, throat, lungs and eyes. Respiratory exposure to high levels of MMA is known to cause occupational asthma, whilst dermal exposure to MMA, EMA and solvents have the potential to cause skin sensitisation that could lead to contact dermatitis. Another target organ of these solvents is the central nervous system (CNS) (ATSDR, 1994; FDA, 1974; Liteplo and Meek, 2003:6; Bruckner, *et al*. 2007:1010; McNary and Jackson, 2007:573; Etkin, 1996:409; Patnaik, 1999:491). Formaldehyde is the only one of these chemicals that has been associated with cancer. Studies show that exposure to formaldehyde causes cancer at sites including the oral cavity, hypo pharynx, pancreas, larynx, lung and brain (IARC, 2006). Because of this, the International Agency for Research on Cancer categorised formaldehyde in group 1 as a substance carcinogenic to humans (IARC, 2006).

A number of articles raised concerns regarding the health risks of nail technicians including, irritation of the eyes, skin and respiratory tract, dermatitis, cognitive symptoms, central nervous system depression, occupational asthma and other respiratory symptoms, liver and kidney damage, miscarriages, birth defects and cancer (Heymann, 2007:1069-1070; Sole-Smith, 2007:21-22). Two studies investigated these concerns and found significant increased prevalence of self-reported work related symptoms in nail technicians. These symptoms include lower respiratory symptoms, headaches, skin eye and throat irritations and numbness (Adisesh *et al.*, 2008; Quach *et al.*, 2008:336-343).

Leading from these concerns a study was launched to quantify the exposure of nail technicians working in nail salons. In the past, occupational exposure to HCS was only measured by means of respiratory exposure. To quantify exposure comprehensively dermal exposure must also be measured as chemical absorption through the skin is a major contributor to the total exposure (Fenske, 1993:687 ; Niedner; 2008:334).

For that reason this study focused on respiratory- as well as the potential dermal exposures of 15 nail technicians working in different salons (situated in Potchefstroom and Klerksdorp) to acetone, acrylic monomers (MMA and EMA), formaldehyde, toluene and xylene.

The results showed that the nail technicians are not subjected to immediate health risks as the TWA exposure levels of each chemical was well below the OELs and Action levels (50% of OEL). The possibility of an additive effect resulting from exposure to the mixture of HCS was also rejected. However, it cannot be concluded that nail technicians are at no health risk due to long term occupational exposure. The unknown effect of chronic low level exposure to solvents along with the large number of previous studies that reported increased health risks in nail technicians must also be considered (John *et al*., 1994: 147-155; Adisesh *et al.*, 2008, Bruckner and Warren., 2003: 360-371). Furthermore, nail technicians are at risk of developing skin sensitization and contact dermatitis as MMA was detected in respiratory and dermal samples. This is despite the fact that MMA has been banned by the FDA in the USA due to its potential to cause skin sensitization (Lewis, 1998). The detection of MMA is therefore also a cause for concern.

Dermal exposure results proved to be a contributing factor when quantifying the total exposure to HCS. Charcoal pads as a method for measuring potential dermal exposure proved to be very user friendly and took very little time to apply and remove from the sampling area. Charcoal pads were designed to be worn under personal protection gloves on fingers and palm of hands to determine the brake through thereof. The charcoal pads were also simple to transport as it is very small and lightweight. These physical qualities as well as the position of the charcoal pads on the hand (top of hand) made it possible for nail technicians to go about their normal tasks without restrictions. The pads could not be placed on the fingers or on the palms of the nail technicians' hands as some of the technicians performed massages as well as facials during the day. The oil, soap and water used during these tasks could have compromised the absorption of the charcoal pads and therefore lead to unreliable results.

The potential dermal exposure (before adjustments) to acetone, EMA and MMA showed strong correlations to the concordant chemical's respiratory exposure. After adjusting the dermal exposure, only one significant correlation was observed in the case of EMA. The weak correlations between the adjusted dermal- and respiratory- exposure can be explained by the accidental or random manner in which adjusted dermal exposure occurs. Adjusted dermal exposure represents spillage or spatter of the products used in nail salons and is thus not influenced by concentrations of airborne HCSs. The high percentage of adjusted dermal exposure values that had to be estimated shows that a large part of the unadjusted dermal exposure on the hand can be attributed to vapours and not direct contact to HCS.

The difference in proximity from the source could also contribute to the difference in the exposure measured on the hand and neck. Adjusting the dermal exposure on the hand might therefore lead to an underestimation of the contribution of dermal exposure to HCSs (Van Wendel De Joode *et al.*, 2005:49)

In conclusion, taking the uncertainties surrounding the adjustment of dermal exposure into account, as well as the fact that the activated charcoal pads has a higher absorption potential than human skin, it becomes apparent that the use of charcoal pads as method for measuring potential dermal exposure remains problematic (Eriksson and Wiklund, 2004b: 563-568; Vermeulen *et al.*, 2006: 1143-1148). This method as well as the method used to adjust the results must be improved to increase accuracy of results.

The first hypothesis that we investigated that is: "Nail technicians' respiratory exposure levels measured over 8 hours are below the TWA occupational exposure limits" can consequently be accepted. Conversely, the second hypothesis namely: "Surrogate skin methods such as the charcoal pad (PERMEA-TEC pads®) can be used as a reliable method to quantify dermal exposure to chemicals in nail salons" must be rejected.

Limitations during this study included the use of perfume and hand moisturiser by nail technicians while wearing the charcoal pads. The nail technicians also washed their hands between clients while wearing charcoal pads. These actions may have influenced the potential dermal exposure results. The use of three charcoal pads instead of two, with the third patch also placed on the hand, could have given a more accurate representation of direct exposure, close to the source. Another limitation was the irregular and unpredictable nature of a nail technician's work shift as well as the variability in tasks performed from one day to the next.
The daily tasks and the shift duration of nail technicians cannot be predicted because it depends on client bookings. As the clients' demand for nail treatments fluctuate so will the daily tasks and shift duration of a nail technician change.

Future studies must, therefore, shift their focus to short term exposure for the duration of different tasks performed during nail sculpturing. This will improve accuracy of results and is imperative to establish worst case scenario exposure in nail salons. Increasing the dermal area that is sampled by increasing the number of charcoal pads used will also improve accuracy of results.

It is recommended that nail technicians working with acrylate nail products must wear gloves. This is to prevent skin sensitisation and resulting contact dermatitis as MMA is still present in cosmetics used in South Africa. The use of Latex gloves is not recommended as it can also lead to an allergic reaction. Secondly, because health effects of long term low level exposure to solvents are not yet fully understood, increased ventilation is suggested to lower exposure. This can be achieved by opening windows and doors during working hours. If is not practically possible to improve natural ventilation the area can be artificial ventilated by means of an extraction system.

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