A Discussion of Some of the Scientific Issues Concerning the Use of PVC

An update of the CSIRO report "The environmental aspects of the use of PVC in building products, Second Edition, 1998"

A study
carried out for the
Vinyl Council of Australia

CSIRO Molecular Science
Australian National University

CSIRO

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A Study carried out for the Vinyl Council of Australia

Author: Dr Phillip Coghlan
Project Officer
CSIRO Molecular Science and
Australian National University

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STUDY BRIEF

Prepare a scientific overview of the main environmental issues and perceptions associated with the major products of the Australian PVC industry in order to assist the industry in the sound management of these issues.

In particular, provide a brief update of the principal conclusions of the CSIRO's June 1998 report *The environmental aspects of the use of PVC in building products*. Address each of the major issues identified in the Executive Summary of that report.

Examine peer reviewed literature and public reports from expert bodies, including expert overviews by recognised scientists, scientific authorities and international regulatory agencies. Where practical, consider scientific opinion on how the hazards and risks of PVC compare with those of alternative materials for similar end use products, and how they compare with the benefits bestowed.

Provide a brief overview of phthalate issues in relation to building and construction and other significant PVC product sectors. This synopsis should describe the concerns and views expressed by the public, international regulatory agencies, scientists and the international industry. It should describe in general terms the major technical issues (e.g. analytical methodology, availability of exposure data, definitive effects in humans, testing of replacement compounds, etc.). It is not intended that the toxicology of the phthalates be reviewed, though it may be necessary to provide contextual information in relation to the debate.

Vinyl Council of Australia
May 2000

EXECUTIVE SUMMARY

This study updates the principal conclusions of the CSIRO's June 1998 report "*The environmental aspects of the use of PVC in building products – second edition*". The issues identified in that report were the use of phthalates in plasticised PVC and their possible health and environmental effects, the fate of heavy metal-based heat stabilisers, PVC and its relation to fire and toxic emissions when combusted, the potential for recycling of PVC building products, and the appropriateness of alternatives to PVC in its building product applications.

The effects on human health and the environment from the release of phthalates present in plasticised PVC products have been the subject of much controversy. However, conclusive scientific evidence supporting the assertion that the most commonly used phthalates (DEHP and DINP) present a danger to humans is lacking. The perceived potential regarding the possibility that the toxicity of some phthalates may be the result of endocrine disruption is the principal cause for concern over phthalates, however there is a general lack of relevant information concerning possible adverse effects of endocrine disrupting chemicals on humans at environmental exposure levels.

The fate of the heavy metal-based thermal stabilisers used in PVC products has been the subject of recent investigations. In the case of the most commonly used stabiliser, lead compounds, the concerns relating to extraction are overstated. It has been found that the stabilisers are held within the PVC matrix and only limited, initial losses from the surface occur. Under landfill conditions the stabiliser loss is still slow and minor compared to other sources of lead in the environment. Likewise, the contribution made by PVC to the release of heavy metals into the environment as a result of building fires can be considered minor due to the rare occurrence of these events.

The behaviour of PVC during building fires has been the subject of much debate. Rigid PVC is inherently flame retardant, but plasticised PVC often requires flame retardant additives, most often metal hydrates and chloroparaffins, to reduce the fire hazard. Compared to its common plastic alternatives PVC products perform better in terms of lower combustibility, flammability, flame propagation, and heat release. However, in terms of smoke density and the release of corrosive gases, PVC performs poorly. The contribution made by PVC to the emissions of dioxin appears to be insignificant compared to other sources.

Even though waste PVC building products are suitable for recycling efforts have been low scale compared to other consumer plastics recycling programs.

There are alternatives to PVC in all building and construction applications. Nevertheless, the balance of available evidence indicates that PVC in its building and construction applications has no more effect on the environment than its alternatives.

It can be concluded from the evidence cited in this and the preceding reports that the possible adverse human health and environmental effects of using PVC in building products is not greater than those of other materials. However, there are several aspects which require additional study because the evidence is either unavailable, inconclusive or contradictory. Significant efforts are currently underway internationally to attempt to clarify some of the issues surrounding the use of PVC. These include the health and environmental effects of the phthalate plasticisers used in flexible PVC, the ultimate fate of the heavy metals used as heat stabilisers, and the toxicity of the emissions from accidental fires involving PVC.

1. INTRODUCTION

Poly(vinyl chloride) (PVC) is arguably the most versatile of all the plastics (more correctly termed polymers). It is resistant to corrosion and weathering, a superb electrical insulator, impact and scratch resistant, tends not to crack, can be made rigid or flexible, and under many circumstances, it does not catch fire readily. PVC is also cheap to produce and can serve its designed function for decades. Due to its high chlorine content (57 wt% in virgin PVC) PVC is inherently more energy conservative (less fossil carbon is utilised) compared to its polymer alternatives. However, when energy consumption during manufacture is included PVC is on parity with the other major plastics (UBA 1999). The uses of PVC are many and varied. The building products sector includes pipes (sewerage and potable water), cable and wiring covers, electrical switches and conduit, membranes, insulation, flooring, trim, and window frames. Consumer household uses include toys, blinds, wallpaper, furniture, shower curtains, electrical casings, imitation leather, and surface finishes. Packaging and medical disposables are also minor but important applications.

PVC, with a world-wide market second only to low-density polyethylene (Fauvarque 1996), is the plastic most widely used in building product applications. Virgin PVC is thermally and photochemically unstable and various additives are included in the formulation of PVC products to reduce these limitations. The classes of additives that can be used include inert fillers, heat stabilisers, plasticisers, flame retardants, lubricants, impact modifiers, smoke retardants, pigments, UV-radiation stabilisers, antistatic agents, bubbling agents, and fungicides.

The health and environmental effects of PVC and its additives have been the subject of intense debate in recent years (Hansen 1999). The focus of this debate was initially concerned with the manufacture of PVC but has now shifted to the use and disposal of the material. The problems that are most often associated with the use of PVC in building and other product sectors arise from a number of factors, namely the release or extraction of the heavy metal-based stabilisers, uncertainty surrounding the health implications of the phthalate plasticisers and other additives, the formation of dioxins and hydrogen chloride gas and other substances during building fires and incineration, the long-term consequences of landfilled PVC, and the poor recycling record of PVC waste.

2. SCOPE AND METHODOLOGY

This report is based on a scientific assessment of the information currently available on the environmental consequences of using and disposing of PVC. Extensive recourse has been made to the peer-reviewed scientific literature, reports by national and international government agencies and universities, and specific data obtained from industry sources. The amount of information specific to the Australian situation is very limited, therefore it is necessary in most cases to make recourse to the international resources and adapt them to the situation in Australia.

It is not intended to repeat here the overview of the manufacture of PVC discussed in the preceding report (Smith 1998), with the reader being directed to this and other comprehensive publications on the subject (Allsopp and Vianello 1992; Emsley 1994a; Smith 1996). The issues relating specifically to the manufacture of PVC resin, PVC compounds, PVC precursors (VCM, EDC, chlorine), plasticisers, stabilisers, flame retardants, and other additives, are regarded as primarily occupational health and safety concerns and therefore will not be covered in this report.

The information presented here will update the considerations of the report "*The environmental aspects of the use of PVC in building products – second edition*" (Smith 1998). The following issues concerning the possible health and environmental hazards associated with the use of PVC have been identified:

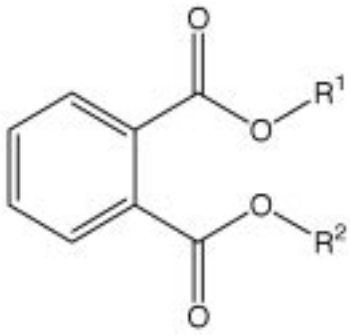
1. The use of phthalates in flexible PVC, and the consequent possibility of loss to the environment.
2. The ultimate fate of heavy metals used as heat stabilisers.
3. The toxicity of emissions from accidental fires and the inherent non-flammability of PVC as a positive attribute in a building fire situation.
4. The potential for recycling of rigid PVC products used in buildings.
5. The environmental effects of PVC compared to its alternatives.

A discussion of these points will attempt to put into perspective the risks and hazards associated with the use and disposal of PVC. As with many controversial subjects, there exist conflicting results and differing interpretations of the same data with regard to the issues relating to PVC. Dean (1995) summarises the situation precisely when he states, "*a large number of the perceived risks from e.g. PVC, dioxins, incineration, furnace ash, waste fuel oil, etc. are derived from assumptions that have been unsupported or refuted by later studies, but which remain strongly entrenched in the public mind*".

3. PHTHALATES

3.1 Properties and use

Phthalates are a compound class with the general structure shown below (where R^1 and R^2 can be various combinations of straight and branched alkyl chains). They are most commonly employed as plasticiser additives and typically comprise 10 – 50% of the weight of the finished flexible PVC product. The addition of a plasticiser to PVC introduces the problem of plasticiser migration. This is because the plasticiser is not bound to the polymer chain and is thus free to migrate out of the PVC product at its surface (Cadogan and Howick 1992; Smith 1998; Shashoua 2000). This phenomenon is in contrast to that found for the heat stabilisers, which do not migrate out of the PVC product (Smith 1998).



General structure of the phthalates

The most commonly used phthalate plasticiser is di-2-ethylhexyl phthalate (DEHP). The other common phthalate plasticisers are diisononyl phthalate (DINP) and diisodecyl phthalate (DIDP). Dibutyl phthalate (DBP), benzyl butyl phthalate (BBP), diisobutyl phthalate, diethyl phthalate, dimethyl phthalate, and diisoheptyl phthalate are used less frequently. In general, the commonly used phthalates have similar physical and chemical properties (Staples *et al.* 1997a). They also tend to have low acute and chronic toxicities (Cadogan and Howick 1992; David *et al.* 2000), however selected phthalates have shown a number of effects in *in vitro* studies that have caused concern. These are discussed below.

3.2 Environmental fate

The environmental fate of phthalates once they migrate from PVC products has been extensively examined (Staples *et al.* 1997a; Staples *et al.* 1997b;

Cadogan 1999; Staples *et al*. 2000). The current level of concern surrounding phthalates stems, in part, from the fact that they have low-level world-wide environmental distribution; are consumed in minute quantities by humans and animals alike; and in numerous *in vitro* and some *in vivo* laboratory studies have been shown, in large doses, to have potential for causing pronounced biological effects (Møller *et al*. 1995; Cadogan 1999). For these reasons they have been implicated as a possible contributor to a number of human disease states and ailments (Wilkinson and Lamb 1999; Sharpe 2000).

The commonly employed phthalate plasticisers generally have low water solubilities (although the values cited are variable). Phthalates are susceptible to a number of degradation pathways including hydrolysis, photochemical, and biodegradation (Staples *et al*. 1997a). Acidic and alkaline hydrolysis of phthalates firstly affords the mono-ester and an alcohol moiety followed by formation of phthalic acid and a second alcohol moiety. This pathway is most relevant to higher animals where ingestion of phthalates results in rapid formation of the mono-ester in the stomach. Phthalates also undergo rapid aerobic and slow anaerobic biodegradation. Dorfler *et al*. (1996) found that DEHP will degrade under soil conditions but that the rate is dependent on several factors that might not be present at all sites. Phthalates appear to persist in sediments where anaerobic conditions exist (VROM 1998). Cartwright *et al*. (2000) studied the degradation and impact of phthalates in a variety of soil conditions and found that in situations of poor bioavailability to microbes they were recalcitrant in the soil. However, they concluded that "*DEHP had no effect on the microbial community or membrane fluidity, even at 100 mg/g, and was predicted to have no impact on microbial communities in the environment*".

Mersiowsky *et al*. (1999) studied the landfill fate of PVC waste and made a number of observations and conclusions. They found that the vinyl chloride found in landfill gas does not originate from the PVC waste, the PVC polymer matrix does not degrade, loss of phthalate plasticiser from PVC certainly occurs but the extent is dependent on the surroundings and the plasticiser type, and phthalates are generally subject to biodegradation. In addition Webb *et al*. (1999) have found that "*plasticisers may accelerate the biodeterioration processes occurring on pPVC by enhancing fungal adhesion*".

3.3.1 Issues of human exposure

The potential health effects of phthalates have been the subject of much debate in the scientific community. Whilst there is still a large degree of uncertainty as to their role in certain health issues there are a number of established facts concerning phthalates. The overwhelming weight of evidence from

mutagenicity studies shows that phthalates are not genotoxic (Woodward *et al*. 1986; Fung *et al*. 1997; CERHR 2000a-g). DEHP and some other phthalates cause liver cancer in rodents *via* a mechanism known as peroxisomal proliferation (Latruffe *et al*. 1995; Parmar and Seth 1997; Qi *et al*. 2000). However, this mechanism is not considered relevant to humans because of the difference in the way that the human liver responds to phthalates compared to the rodent liver. Goll *et al*. (1999) found no peroxisomal proliferation of human liver cells in *in vitro* studies. In an *in vivo* study on marmosets conducted by Kurata *et al*. (1998) no peroxisome proliferation was detected after administering DEHP. Doull *et al*. (1999) concluded that "*the hepatocarcinogenic response of rodents to DEHP is not relevant to human cancer risk at any anticipated exposure level*". Based on studies of DEHP on cynomolgus monkeys Pugh *et al*. (2000) concluded that "… *phthalate esters do not appear to produce hepatic effects associated with peroxisome proliferation and hepatic carcinogenicity in humans*". An expert panel convened by the American Council of Science and Health (Koop *et al*. 1999) concluded that "… *there is no convincing evidence to date that early childhood cancers are caused by exogenous agents*", such as phthalates. On the basis of mechanistic studies involving peroxisomal proliferation, the World Health Organisation's International Agency for Research on Cancer has recently reclassified DEHP as "*not classifiable as to carcinogenicity to humans*" (IARC 2000). Some phthalates have been shown to have effects on the testis of laboratory animals (Sjöeberg *et al*. 1985; Parmar *et al*. 1986; Parmar *et al*. 1987; Mylchreest *et al*. 1998) and are thus suspected of contributing to the indications of reduced male fertility observed in some populations (Li *et al*. 1998; Sharpe 1998).

Concern over plasticiser migration from pPVC products has made phthalates the focus of considerable recent attention (Fiala *et al*. 2000; Safe 2000). The Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) has established standard protocols for the extraction of phthalates from plasticised PVC toys (CSTEE 2000). In 1999 the European Commission banned the use of DEHP in PVC toys and other easily mouthed items intended for children under 3 years of age as a precaution against the uncertain impact of phthalates on young children (EC 1999). Manufacturers of PVC toys have also voluntarily withdrawn the use of DEHP in many countries, including Australia.

As DEHP is used in PVC medical devices, patients are likely to be exposed to higher than environmental levels causing some experts to be concerned (CERHR 2000a; Rossi 2000). DEHP has long been known to leach from PVC blood bags and has been incidentally shown to have beneficial effects on

the quality of the blood products (Estep *et al*. 1984; Zhao and Courtney 1999; Yin *et al*. 1999). Loff *et al*. (2000) determined that newborns receiving parenteral nutrition *via* PVC-infusion systems are exposed to DEHP at a dose of 5 mg/kg body weight/day. Faouzi *et al*. (1999) found that adult hemodialysis patients were on average exposed to 75 mg of DEHP per session. Despite these findings scientists remain divided as to the risks associated with phthalates. Sharpe (2000) surmises that "*in both Europe and the US the weight of scientific opinion is that PVC toys and medical items are safe*". The Center for the Evaluation of Risks to Human Reproduction Expert Panel Review of Phthalates (CERHR) expressed "*serious concern that* intravenous *exposure* to DEHP *may adversely affect male reproductive tract development*" of critically ill infants undergoing intensive care treatment with PVC-based medical devices (CERHR 2000a) and "*low concern for potential health effects in children*" resulting from exposure to DINP in toys and other objects children may mouth (CERHR 2000b). Another expert panel convened by the American Council of Science and Health (Koop *et al*. 1999) found that "… *DEHP in medical devices is not harmful to even highly exposed* people …" and on DINP "… *the Panel concludes that much of this evidence has little relevance for humans and that DINP in toys is not harmful for children in the normal use of these toys*". Wilkinson and Lamb (1999) reviewed the potential health effects of the phthalate esters in children's toys and concluded that DINP "*does not present a significant risk to children*" and that "*the scientific evidence supports the continued use of DINP as a plasticiser in children's products*".

3.3.2 General aspects of endocrine disruption

An increasingly important area of concern regarding phthalates, and the major reason for the intense debate, is their implication as possible endocrine disrupting chemicals (Heinze and Adams 1997; Langer and Sang 1997). An endocrine disrupting chemical is defined as 'an exogenous substance that causes adverse health effects in an intact organism, or its progeny, consequent to changes in endocrine function' and a potential endocrine disrupter is 'a substance that possesses properties that might be expected to lead to endocrine disruption in an intact organism' (EC 1996). The complexity of the issues surrounding endocrine disruption necessitates discussion of the subject in detail in order to adequately explain its relevance to the issues surrounding phthalates.

Reviews on the subject of endocrine disruption published since the previous report (Smith 1998) are numerous and include those by Cheek and McLachlan (1998), Crisp *et al*. (1998), Keith (1998), NICNAS (1998), Solomon (1998), Sumpter (1998), Tyler *et al*. (1998), Adlercreutz (1999), Botham *et al*. (1999),

Cadogan (1999), Caldwell (1999), Depledge *et al*. (1999), Environment Canada (1999), Fritsche and Steinhart (1999), Gans (1999), Heffron (1999), Humfrey and Smith (1999), Koop *et al*. (1999), Menditto and Turrio-Baldassarri (1999), Phillips and Harrison (1999), Risebrough (1999), Rollerova and Urbancíková (1999), Sadik and Witt (1999), Safe (1999), Taylor and Harrison (1999), Younes (1999), Beresford *et al*. (2000), Juberg (2000), Maczka *et al*. (2000), Patrolecco *et al*. (2000), Sharpe (2000), and Vos *et al*. (2000). The range of natural and anthropogenic (mankind) chemicals that are known or suspected endocrine disrupters is wide and include those listed in the following table.

Known and Suspected Endocrine Disrupting Chemicals

Compound classes	Examples and occurrence
Natural hormones	Estradiol, estrone, estriol: natural human estrogens.
Synthetic hormones	Ethinyl estradiol (the Pill), diethylstilbestrol: medically prescribed synthetic compounds.
Phytoestrogens	Genistein, coumestrol, enterolactone: estrogen-like natural constituents of many foodstuffs including beans, sprouts and soybean.
Organochlorine pesticides	DDT (and metabolites), dieldrin: persistent environmental pollutants.
Polychlorinated biphenyl's	PCBs: widespread, persistent environmental pollutants.
Alkylphenol polyethoxylates	Nonylphenol, octylphenol: surfactants found in shampoos and some plastics.
Dioxins/furans	TCDD: unwanted products of combustion of many materials.
Plasticisers	Dibutyl phthalate, di-(2-ethylhexyl) phthalate, butyl benzyl phthalate, bisphenol A: added to plastics to afford flexible materials.
Polycyclic aromatic hydrocarbons	PAH's: products of combustion processes, especially prevalent in diesel exhaust.

Endocrine disrupting chemicals have been proposed as a potential cause of a number of human health problems including reduced sperm counts and semen quality, increases in cases of cryptorchidism (undescended testicles) and hypospadias (a congenital malformation of the penis), and rising rates of testicular and prostate cancer (Humfrey and Smith 1999). However, while there is some

evidence for these adverse trends, the evidence to link exposures to endocrine disruptors in the environment is either absent or equivocal. Human exposure to potential endocrine disrupting chemicals occurs even before conception and continues until death and the levels to which individuals are exposed over their lifetimes is highly variable. It is certain that everyone is exposed to low levels of known and suspected endocrine disrupting chemicals every day of their lives (we are 'living in a sea of estrogens') but it is not certain what the significance of this exposure is with regard to human health (Safe 1999; Juberg 2000).

In 1998 the International Union of Pure and Applied Chemistry, the International Union of Pharmacology and the International Union of Toxicology collaborated to produce perhaps the most comprehensive peer reviewed volume of information on the current state of scientific knowledge underpinning the risks of exposure to endocrine disrupting chemicals, both natural and anthropogenic (IUPAC 1998). The respective Presidents of these International Unions recommended that researchers in the endocrine disruption field ensure "*careful checking of experimental results*", that "*all scientific contributions should be peer reviewed*" and that "*care should be taken not to exaggerate the likely consequences of particular observations*". It is this author's opinion that this is the most prudent approach to the problems associated with the scientific uncertainty involved in the endocrine disruption debate.

A wide variety of assays for testing the endocrine disrupting potential of chemicals have been developed. The validity of the conclusions drawn from these tests remains a controversial topic in the scientific community (Jobling 1998; Safe 1998; Andersen *et al*. 1999; Purchase 1999; Elswick *et al*. 2000). Sadik and Witt (1999) believe that "*currently, no suitable characterisation method exists for evaluating most* (endocrine disrupting chemicals) *EDC's in biota or biological media*". Beresford *et al*. (2000) warn that "*considerable care and thought must be applied when interpreting results derived from any single assay*" and that "*only by using a suite of assays will we minimise the chances of wrongly labeling chemicals as endocrine disruptors*". This last point seems to be a prudent approach and is likely to be adopted by the US EPA in their endocrine disruption screening program.

Endocrine disrupting chemicals have been implicated as possible causative agents for a perceived reduction in male fertility as determined by studies indicating historically reduced sperm counts (Carlsen *et al*. 1992), although some question the methodology used in these studies (Weber and Vreeburg 1998). On the subject of the causative agents that might be responsible for these trends, the evidence is far from conclusive. Sharpe (1998) states that "*the possibility that exposure to environmental oestrogens has caused deleterious changes*

in male reproductive health remains a plausible hypothesis. However, we still lack any piece of direct evidence which would support this theoretical relationship." And most recently (Sharpe 2000) "*as yet there are very few data regarding the toxicity of phthalates in human populations; data on reproduction and development are non-existent*". Safe (1999) points out that "*recent studies have reported that … there are remarkable demographic variations in sperm counts, and some of these differences within the same country are higher than the reported decrease in sperm counts in the original meta-analysis by Carlsen and coworkers*". In a report for the Teratology Society, Barlow *et al*. (1999) concluded that "*the data presently available do not support a consensus view that chemicals present in the environment* (including phthalates) *are contributing to observed increases in human developmental disorders that are potentially endocrine related*". Ashby *et al*. (1997) are concerned that "*one should always keep in mind that the regulation of synthetic chemicals for endocrine disrupting properties may not alleviate the observed increases in human breast cancer and testicular cancer or the apparent decrease in human sperm counts and sperm quality reported in some countries*".

The effects of endocrine disrupting chemicals on wildlife and the extrapolation of these observations to possible human health problems has been debated extensively in the scientific literature. Much of the evidence for endocrine disruption of wildlife populations comes from sites with heavy environmental pollution (Tyler *et al*. 1998; Vos *et al*. 2000). There is little consensus regarding the actual effects of environmental endocrine disrupting chemicals on wildlife populations and the associated potential human health problems with some commentators expressing concern (Tyler *et al* 1998; Botham *et al*. 1999) while others remain cautiously skeptical (Sumpter 1998; Van der Kraak 1998; Risebrough 1999; Taylor and Harrison 1999).

Much of the controversy surrounding the issue of endocrine disruption is caused by the uncertainty over the relevance of *in vitro* and *in vivo* laboratory animal experiments to humans, which is the major driver for the current wave of investigation into the issue (Jobling 1998; Maczka *et al*. 2000; Risebrough 1999; Safe 1999; Juberg 2000). The issue of 'species differences', or the importance of differences in the way different species respond in studies, and the extrapolation of results to humans, has yet to be resolved (Hengstler *et al*. 1999). The issue of ultra low-dose effects of endocrine disrupting chemicals versus traditional toxicity tests is another aspect of the endocrine disruption area that is under intense scrutiny (Carpy *et al*. 2000; Fang *et al*. 2000).

The hazards associated with the naturally occurring endocrine disrupting chemicals, the phytoestrogens (plant derived compounds with estrogen-like activity), might be greater than those posed by environmental levels of

synthetic chemicals (Kuiper *et al*. 1998; Safe and Gaido 1998). Some phytoestrogens are known to be many orders of magnitude more potent endocrine disruptors than their synthetic counterparts, which in most cases are many orders of magnitude less active than β-estradiol, a natural estrogen. The possibility of adverse effects resulting from exposure to phytoestrogens is uncertain (Santti *et al*. 1998; Holmes and Phillips 1999). Adlercreutz (1999) notes that "*during pregnancy the endogenous estrogen load is very high and still the fetus can handle relatively high amounts of dietary phytoestrogens*". However, due to their low levels and poor bioavailability Fritsche and Steinhart (1999) claim that "*… hormonal effects cannot be expected from the ingestion of naturally occurring dietary steroid hormones*". Endogenous estrogens are a known risk factor for human breast cancer, however, the contribution that environmental levels of xenoestrogens (compounds with estrogen-like activity that are foreign to the body) make must form only a small component considering the overall dietary intake of phytoestrogens (Mazur and Adlercreutz 1998). Soy products are rich in phytoestrogens and have been associated with both positive and negative human health effects (Turner and Sharpe 1997; Humfrey 1998; Adlercreutz 1999; Davis *et al*. 1999; Holmes and Phillips 1999).

3.4 Phthalates in perspective

Concern about the potential for phthalates to have adverse health effects at the levels encountered during normal use, or at environmental exposure levels, now centres around the possibility that their toxicity may be mediated by disruption of normal endocrine control of reproductive tract tissue development. However, on the balance of current scientific evidence, the toxicity associated with some phthalates has not been shown to be the result of classical endocrine disrupting mechanisms i.e. interference of the interaction of hormones with their cellular receptors. Based on several factors (including conflicting results) Moore (2000) concludes that "*the oestrogenic activity of phthalates in* in vitro *studies is not relevant to humans or the environment*". Cadogan (1999) concludes that "*the information from* in vivo *studies clearly shows that phthalates possess neither oestrogenic nor anti-oestrogenic activity*".

Recent *in vitro* studies have not provided conclusive results concerning the possibility of adverse human health effects arising from phthalates. Paganetto *et al*. (2000) found that the phthalates studied did not have significant affinity for oestrogenic, progestinic and androgenic receptors. Nakai *et al*. (1999) found that some dialkyl phthalates bind to a recombinant human estrogen receptor but that "*the most potent di*-n-*butyl phthalate is much less potent that 17β-estradiol in the receptor binding assay*". Zacharewski *et al*. (1998) found that some of the less common phthalates exhibited weak estrogen receptor-mediated activity in some

in vitro assays at high concentrations but none elicited *in vivo* estrogenic responses. Blair *et al*. (2000) studied eight phthalates for *in vitro* human estrogen receptor activity and found no competitive binding.

A recent series of reports from the US Department of Health and Human Services' Center for the Evaluation of Risks to Human Reproduction concluded that the phthalates DEHP, DINP, DIDP, DBP and BBP were of minimal or no concern as regards their effects on the reproductive systems of human adults (CERHR 2000a-f). However, the expert panel expressed serious concern as regards the possibility of adverse effects on the developing reproductive tract of male infants exposed to very high levels of DEHP that might be associated with intensive medical procedures such as those used in critically ill infants (CERHR 2000a). Primarily because of data gaps some concern was also noted with regards to the exposure of pregnant women to environmental levels of DEHP and the possibility of adverse effects on the foetus. The Panel called for follow-up evaluation of reproductive system development and function of human populations who were heavily exposed to DEHP (i.e. premature infants).

It is relevant to note here that compared to many phthalates, the often recommended alternative to phthalate plasticisers, di-2-ethylhexyl adipate (DEHA), migrates from PVC products to a substantially greater extent (Petersen and Breindahl 1998; Wallace 1999), is a peroxisome proliferator, weakly binds to fish endocrine receptors (Jobling *et al*. 1995), and is a rodent teratogen in high doses (Singh *et al*. 1973; Hodge 1988). The health and environmental effects of alternatives to phthalate plasticisers need to be studied in far greater detail before they can be reasonably considered as appropriate replacements (CSTEE 1999).

In response to the possible adverse effects of phthalates migrating from plasticised PVC products there have been a number of reported advances that are designed to reduce or halt this occurrence. Lakshmi and Jayakrishnan (1998) have reported a method for the modification of flexible PVC that produces migration resistant PVC surfaces suitable for many medical devices. Bichara *et al*. (1999) reduced the rate of DEHP migration from PVC items using a simple two-step process that involves soaking in a liquid and then drying at various temperatures.

Despite all the studies performed to date there is a lack of scientific evidence showing that phthalates have an adverse effect on humans at the levels likely to be encountered either environmentally or during normal use of phthalate containing PVC products. However, the possibility that such a link will be established in the future should not be discounted. The scientific community

and the PVC industry must remain open to the possibility of change in the scientifically valid understanding of the endocrine disruption issue.

On the balance of available scientific evidence there is little justification for greater regulatory control than currently exists in relation to the use of DEHP or any other phthalate as a plasticiser of PVC. Phthalates are non-toxic at levels relevant for the vast majority of human exposure, non-genotoxic, and are not regarded as potentially carcinogenic to humans and have not been shown to be human endocrine disrupting chemicals. However, the endocrine disruption mode of action remains a plausible mechanism by which a variety of chemicals might cause adverse effects on humans. Although animals may accumulate phthalates from the environment, the phthalates do not bio-magnify through the food chain because they are rapidly metabolised by most organisms. Because available data do not implicate phthalates as contributors to human disease, and because there is a lack of research into the possible health and environmental effects of the alternatives to phthalate plasticisers it cannot at this time be recommended that the phthalate plasticisers be phased out in favour of the available alternatives.

4. STABILISERS

4.1 Use

Stabilisers are added to PVC to afford protection against thermal and UV degradation of the polymer chain during processing and use, respectively (Wolf and Kaul 1992). The commonly used stabilisers are compounds of lead (basic lead sulfate and lead stearate), tin (mono- and dibutyltin and tin thioglycolate), cadmium, and complex salt systems of barium/zinc and calcium/zinc.

4.2 Fate

The fate of these stabilisers has been extensively studied for the lead based compounds but much less so for the other systems. CSIRO's Burn and Sullivan (1993) showed that after the initial commission flushing, the rate of lead extraction from a 100 mm diameter PVC wastewater pipe system was 0.7 µg/L/day. Additionally, Burn and Schafer (1997) estimated that the PVC in a sewer system contributed 0.5 µg/L lead to the waste water. Both of these levels are insignificant when compared to other sources of lead released into the environment, for example Wilkie *et al*. (1996) reported mean lead concentrations in Melbourne domestic sewerage at 13 mg/L. Mesch and Kugele (1992) investigated the risk associated with the use of organotin stabilisers in PVC and concluded that there was minimal risk due to a number of factors including low toxicity resulting from minimal exposure, limited

leaching from the finished PVC products and very low ecotoxicity due to rapid degradation.

The environmental fate of the stabilisers used in PVC products in the context of landfill situations has been studied. The general view expressed in studies investigating the release of stabilisers from PVC products is that because the stabilisers are encapsulated in the PVC matrix, the migration rate is extremely low and would only affect the surface of the PVC product, not the bulk of the material (Swedish EPA 1996). Tukker (1995) found that "*landfill of PVC is believed not to cause environmental problems since the material is inert*". Mersiowsky *et al*. (1999) add that "*landfill leachate possesses an inherent toxicity … PVC waste in landfills does not contribute significantly to the toxicity of the landfill leachate*" and that "*the contribution of PVC waste to the ubiquitous contents of heavy metals in municipal solid waste* (MSW) *is negligible*".

One aspect regarding the fate of the heavy metal stabilisers used in PVC products that appears to have been neglected somewhat, is their potential for release into the environment during fires and incineration. Meharg (1994) studied the inputs of pollutants into the environment from large-scale plastics fires (including PVC) and found that "… *considerable amounts of the heavy metals Cd* (cadmium), *lead, antimony and zinc (Zn) were released into the aquatic and terrestrial environments …*", adding that the "*fire fighting water run-off from chemical fires often causes severe environmental damage*" due to the pollutants the water carries. He concluded that plastics fires "*have the potential to release large amounts of heavy metals into the environment*".

The introduction of heavy metals into the environment is a process that cannot be reversed once it occurs. For this reason efforts must be taken to reduce such contamination of the environment. In a report by the Swedish EPA concern over cadmium is made with the statement that "*the National Environmental Protection Agency's view is that cadmium must be removed from the eco-cycle, which means plastics containing cadmium must not be recycled*" (Swedish EPA 1996). In Sweden it has been recommended that "*the use of lead stabilisers in Swedish manufacturing of PVC products from new, non-recycled plastic raw materials should be reduced* (90% of 1994 levels by 2005)" (KEMI 1996). The Ca/Zn stabiliser system contains co-stabilisers such as diketones, epoxies, organophosphites, phenols, and polyols making the formulations more complex and less readily classifiable as to their possible health and environmental effects. The German Federal Environment Agency (UBA 1999) highlight that "*as far as pipes are concerned, the required long-term stability with the addition of Ca/Zn is still being examined*". It appears likely that efforts

will be made to limit the spread of heavy metals into the environment. Thus, it is inevitable that PVC will contribute its share of the efforts with a shift away from the heavy metal stabilisers currently being used. It is noted that cadmium based stabilisers are not used by the principal Australian manufacturers, although there is some use of cadmium-based pigments in Australia (Faulkner 2000).

4.3 Health Effects

The health effects of the metals used in PVC stabilisers are well known. All forms of lead are extremely toxic to humans due to their cumulative effects, causing anemia, neurological effects (especially on children), kidney damage, sterility and miscarriages, and possibly cancer. Metallic tin is generally considered harmless, however, organotin compounds can be toxic to the central nervous system and the liver. Cadmium is known to cause kidney damage in humans and may also cause hypertension and anemia. With chronic inhalation this metal may result in cancer of the respiratory tract. Calcium and zinc are both essential elements that are considered non-toxic to humans.

In an isolated instance Ward *et al*. (1997) reported in the Medical Journal of Australia a single case of lead poisoning of an adult male as a result of the chewing (over a 10 year period) of lead stabilised electrical cable.

Finally, there is the potential for the replacement of metal based stabilisers with organic based stabilisers with a pyrimidinedione system recently passing a commercial feasibility study, including recyclability, technical performance, leaching and toxicology tests (Hopfmann 1998).

The fate of the heavy metal stabilisers used in PVC products is dependent on a number of complex factors. Losses of the stabiliser to the environment are limited during the use phase of the product. Because the stabiliser is held within the PVC matrix, only limited losses from the surface of the PVC product will occur. Under conditions where plasticiser leaching occurs, some loss of the stabiliser can occur, but the contribution that PVC makes to the levels of heavy metals in landfill waste is negligible.

5. PVC AND FIRE

5.1 Combustion

Rigid PVC is regarded as inherently flame retardant and will not burn without an external heat source, thus offering a degree of safety not always available from its polymer alternatives. In most cases plasticised PVC will burn without an external heat source and for this reason a variety of flame retardants are often

included in the formulation to reduce this hazard. Flame retardants employed include metal hydrates, such as antimony trioxide and aluminium or magnesium hydroxide, phosphate esters and chloroparaffins. According to the Swedish EPA (1996) the "*chloroparaffins are persistent, bioaccumulative and toxic for various animal species*" and should be avoided.

A distinction must be made between the risk of occurrence of a fire and the risks associated with the consequences of a fire. Due to its low flammability PVC rates highly on its potential to prevent a fire from occurring. The German FEA (UBA 1999) state that "*the main influencing factors for assessing the fire conduct of materials are combustibility, flammability, flame propagation, heat emission and smoke development*". In the event of a fire occurring PVC has advantages and disadvantages in relation to its fire behaviour. The German FEA (UBA 1999) found that "*as far as the fire conduct of PVC is concerned it can be said that the high chlorine content favourably affects the inflammability, flame propagation as well as heat release in the sense of low combustibility, but on the other hand noticeably worsens smoke gas density, corrosiveness and toxicity of the smoke gases*". Compared to its alternatives plasticised PVC will not drip molten material and its heat of combustion is lower (Emsley 1994a).

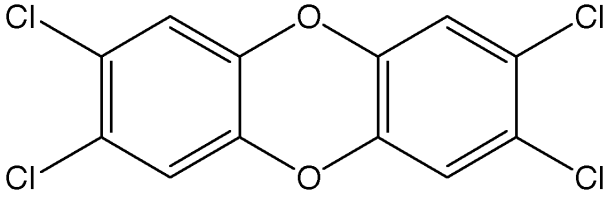
Irvine *et al*. (2000) compared the fire hazards of some common polymers (uPVC, PC, PP, PS and PMMA) with the following conclusions: in terms of time to ignition and the rate of heat release uPVC ranked best, however, in terms of rate of smoke and toxic gas and irritants released uPVC ranked second worst behind PS. Ansari (1997) found that "*the fire behaviour of extruded PVC structural foam is superior to wood in respect to fire propagation index and surface spread of flame classification, but inferior in respect of specific optical density of smoke generated*". There are continuing efforts within the PVC industry and academia to improve the flame retardancy and reduce smoke production and toxicity of plasticised PVC products (Thomas and Harvey 1999).

PVC yields a number of combustion products of which carbon dioxide, carbon monoxide (CO), water and hydrogen chloride (HCl) are the most important. It is established that the single most significant contributor to fire related death is carbon monoxide poisoning. The German FEA (UBA 1999) state that "*the acute fire gas toxicity is largely determined by carbon monoxide (CO)*". Hirschler (1994) states that "… *PVC smoke toxicity is such that … it does not add toxic hazard to that already inevitable due to the carbon monoxide present in very large fires*". The contribution of hydrogen chloride to smoke toxicity is minor with the German FEA (UBA 1999) considering that "*another, although slight, increase in the acute toxicity results from the formation of HCl during fires involving PVC*". Hydrochloride gas has a characteristic strong smell which

enables its detection at very low concentrations whereas carbon monoxide is odourless and therefore potentially more deadly (Perena Conde 1997).

5.2 Dioxins

In addition to the products of combustion discussed above, PVC has also come under some criticism because of the perception it has potential to add to the release of dioxins into the environment during the incineration of domestic and hospital waste. 'Dioxin' is the commonly used name for a group of 210 compounds more strictly termed the polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The 75 PCDDs and 135 PCDFs are naturally occurring, persistent, bioaccumulative organic compounds that are produced during combustion processes, natural and anthropogenic, and during a number of chemical manufacturing processes (Emsley 1994b; Rappe *et al*. 1999). Since their identification in the 1970's as highly toxic environmental pollutants measures have been taken, rather haphazardly in many cases, to curb their formation and release into the environment. Rappe (1996) emphasises that "*it is generally accepted that only 17 out of the 210 dioxin and dibenzofuran congeners are toxic*" with the most toxic being 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). A risk assessment of the complex mixtures of PCDD/Fs can be accomplished with the use of the Toxicity Equivalency Factor (TEF), which ranks the relative toxicity of the 17 toxic PCDD/F congeners compared with TCDD, allowing conversion of quantitative analytical data of PCDD/F mixtures into a single Toxic Equivalency Quotient (TEQ or TEq) measure.[∞]



2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)

It is widely accepted that the toxicologically active PCDD/Fs exert their effects by a common mechanism involving binding to a cytoplasmic receptor protein called the Ah (aryl hydrocarbon) receptor whose physiological role is not yet fully understood (Okino and Whitlock 2000; Poellinger 2000). Chronic levels of PCDD/Fs have been linked to a number of human ailments. According to Neuberger *et al.* (1999) "*chloracne is the most frequent but not the only chronic disease from TCDD exposure*". Grassman *et al.* (1998) add that "*studies of highly*

exposed human populations show that dioxins produce developmental effects, chloracne, and an increase in all cancers and suggest that they may also alter immune and endocrine function. In contrast, the health effects of low-level environmental exposure have not been established".

In fact the possible human health effects of dioxins is the subject of intense debate centred around the interspecies differences with respect to the effects of dioxins on animals, including humans (Boening 1998; Mukerjee 1998; Neubert *et al*. 1999). For example Geyer *et al*. (1997) surmise that "*this question of species differences is one of the central issues of the controversies that have emerged on the validity of risk assessment strategies for TCDD …*". Zimpleman (1999) asserts that "*a fairly substantial body of evidence supports the conclusion that TCDD does not pose a threat to human health in terms of cancer or birth defects despite the danger it poses to experimental animals such as mice*". Finally, Ames and Gold (1997), the former the discoverer of the 'Ames mutagenicity test', find that "*TCDD … is an unusually potent rodent carcinogen but seems unlikely to be a significant human carcinogen at the levels to which the general population is exposed*".

Dioxins are ubiquitous global contaminants to which everyone is exposed. The main route of exposure is *via* food consumption where it is estimated that people are exposed to 1-3 pg (i)-TEQ per kilogram body weight per day (Liem *et al*. 2000). Estimates for atmospheric levels of dioxins in several cities have been found to range from 16-460 fg/m^3 TEQ (Taucher *et al*. 1992; Lohmann and Jones 1999).$^{\Delta}$ Wagrowski and Hites (2000) recently estimated that between 2 and 15 tonnes of PCDD/F is deposited onto the earth's surface each year. Since measures were taken to reduce emissions, dioxin levels in the environment and in human tissues have been falling from their peak which occurred in the late 1960's early 1970's. Van Leeuwen *et al.* (2000) state that "*there is now clear evidence of a decrease in PCDD/PCDF concentrations in human milk over time in almost every region for which suitable data exist*". Wittsiepe *et al.* (2000) studied PCDD/F blood levels in the German population and found that "*over the examined time period a continuous decrease of the PCDD/F concentrations in human blood was observed. The mean levels found were about 42.7 pg I-TEq/g (lipid basis) in 1991 and 20.7 pg I-TEq/g (liquid basis) in 1996 …*". The World Health Organisation (WHO) have established a Tolerable Daily Intake (TDI) range of 1-4 pg TEQ/kg body weight (WHO 1998).

Although the human health effects resulting from exposure to the low environmental levels of dioxin are unknown, there may be no notable effect due to the manner in which the body sequesters dioxin into the fatty tissues.

Emsley (1994b) states that "*dioxins are resistant to breakdown in animals, and tend to collect in fatty tissue*" and "*we may even have evolved to be immune to low levels of dioxins …*". Geyer (1997) adds that the "*… adipose tissue serves as a protective reservoir against the toxic effects of TCDD and related persistent lipophilic compounds*". According to Mukerjee (1998) "*no studies are available determining the impact of daily background exposure to this persistent and potent carcinogen on cancer incidence in the general population*". In a report by the United Nations Environment Programme (UNEP 1999) this uncertainty and the associated concerns are confirmed with the finding that "*although no adverse health effects could be causally linked so far with background exposures of PCDD/PCDF in human milk, for reasons of preventative health care, the relatively high exposure of breast-fed infants must still be considered a matter of concern*". The relatively low incidence of adverse human health effects associated with environmental exposure to dioxins was highlighted by Schecter and Olson (1997) with the estimation that only "*between 0.009% and 0.09% (111-1114 cases/year) of all cancer cases in the US might be directly linked to dioxin intake in food*".

With the exception of the manufacture of the PVC intermediate ethylene dichloride, no PCDD/Fs are produced during the manufacture of PVC products (Baldwin 1997). The issue of dioxins and PVC arises from the combustion or incineration of the latter during disposal or accidental fire. PCDD/Fs are formed during most combustion processes where a source of chlorine, including an inorganic source, is present (Katami *et al*. 2000). The United Nations Environment Programme reported that dioxin inventories have been established in 17 countries in which the main global source of PCDD/Fs is waste incineration (UNEP 1999).

The significance of incineration on the emission levels of dioxins has in recent years led to improvements in the technology used in municipal waste incineration in most of the countries where this method of waste disposal is employed. Eduljee and Gair (1997) consider that "*… it can be stated that the current PCDD/F release limits of 0.1-1 ng I-TEQ* Nm^{-3} *pertaining to PCDD/F release from MSW incinerators in Europe are sufficiently protective of human health relative to the current TDI of the WHO*". The contribution of PVC in the waste stream to dioxin emissions from municipal waste incineration is considered minor due to technical advances and the fact that at up to about 1% chlorine, dioxins levels are not influenced by the chlorine load (Naturvårdsverket 1996). This is exemplified by Rigo and Chandler (1999) who found that "*changing the amount of chlorine in waste streams does not have a discernible impact on PCDD/F emissions from waste combustors*". Olie *et al*. (1997) found that "*formation of PCDD/Fs during incineration of municipal*

waste is promoted by the catalytic properties of metals which are found in the waste itself" and "*prevention of the formation can be done by poisoning the catalysts with complexing compounds*". Yli-Keturi *et al.* (1999) confirmed this finding that "*PCDD/F levels can be reduced in waste incineration by using urea*". Interestingly, Takasuga *et al*. (2000) found that dioxin-free fly ash is a catalyst for the formation of dioxins during the combustion of samples both in the absence and presence of chlorine sources.

In Australia, the only sanctioned incineration of PVC products occurs during the destruction of hazardous PVC medical waste. According to an Environment Australia report (EA 2000), PVC typically comprises only 3% of the clinical waste stream entering Australian medical waste incinerators which collectively contribute only 2.5 – 9.7 g I-TEQ/year to Australia's estimated 150 – 2300 g TEQ/year air emissions of dioxins (EA 1998).

Although limited by a significant lack of Australian source data, a 1998 report on dioxin emissions by Environment Australia estimated that the largest contributor in Australia is prescribed fires and bush fires (EA 1998). The largest anthropogenic source is estimated to be the cement production industry, closely followed by residential wood combustion. Because it is not employed in Australia, unlike the situations in Europe, North America and Japan, municipal solid waste incineration is not a dioxin source in Australia.◊ Due to a lack of available data Connell *et al*. (1999) could not determine the extent of PCDD/F contamination in Australia.

The total contribution to dioxin emission levels resulting from fires involving PVC can be considered minor (Møller *et al*. 1995; Carroll 1996), although there is a high variability in the amounts of dioxin released during PVC fires. The German FEA (UBA 1999) consider "*the generation of dioxin in the case of a real PVC fire can therefore vary greatly because of differences in; the amount of PVC involved in the fire, the prevailing fire conditions, and the spread of fire gases*". Citing Smit *et al*. (1994) Tukker (1995) states that "*an uncontrolled burning of 200 kg PVC in Sweden was reported to result in 3 mg* dioxin. *This is in the same range as for the uncontrolled burning of wood*". Ruokojärvi *et al*. (2000) found that the PCDD/F concentration in the combustion gas of simulated house fires ranged from 1.0 to >7.2 ng/m^3 I-TEQ and observed that "*no additional pure PVC plastic is required for PCDD/F formation in house fires*". The risk of adverse health effects from dioxins produced in PVC fires is considered insignificant compared to the higher risk from other pollutants such as polycyclic aromatic hydrocarbons (PAHs) (UBA 1999). Spindler (1997) concludes that "*in all fires PAHs are the most significant carcinogenic substances in soot compared to dioxins by a factor between 100 to 500*".

The inherent physical and chemical properties of PVC offer both advantages and disadvantages with respect to the fire behaviour of PVC products in building fire situations. With respect to the toxicity of emissions from burning or incinerating PVC the indications are that it is no worse in most respects than other materials, apart from releasing hydrogen chloride. PVC's contribution to dioxin emissions appears to be insignificant compared to other sources.

6. PVC RECYCLING

PVC is a thermoplastic material and as such is readily recyclable (Menges 1996; Scheirs 1998). The reason for the low recycling rate of PVC compared to the amounts produced is largely because it is generally used in long-term applications, building and construction accounting for approximately 90% of Australian consumption (AVC 2000) and therefore does not enter the MSW stream as rapidly as other plastics. This is further reflected in the fact that PVC ranks last in terms of tonnages used in plastic packaging (a short-term application) (Truss and O'Donnell 1998). The barriers to the widespread recycling of PVC are contamination, relative thermal instability, its multicomponent nature, and low collection volumes. In all these cases appropriate technologies are available, however often the economics for recycling PVC are unfavourable (primarily due to the low recycling rate of PVC from building and construction wastes). The Australian PVC manufacturers practice a high level of recycling of industrial waste (Truss and O'Donnell 1998; AVC 2000). In Australia, recycled PVC bottles are cryogenically ground, mixed with virgin PVC and molded into non-pressure pipe and flooring.

In the previous report (Smith 1998) it was noted that the PVC products used in the building sector (pipes, wire insulation etc.) offer the ideal opportunity for reuse or recycling as a means of reducing the overall environmental impact of these materials. Unfortunately the amount of scientific information pertaining to the recycling of PVC building products is extremely limited.

If PVC waste is not recycled then two options are available for its disposal; landfill and incineration. Landfill disposal of PVC has been partly covered in the earlier sections of this report on Phthalates and Stabilisers, where the respective concerns were addressed. Incineration of PVC waste was partly covered in the Dioxin section of this report. It is noted that incineration with energy recovery extracts some of the inherent energy value stored in the PVC

product which is otherwise lost if landfill disposal is used. In terms of greenhouse gas emissions Dean (1995) points out that "*plastics, e.g. polyethylene, polystyrene, PVC, etc., are made from fossil carbon which will persist 'forever' if buried in a landfill, thus locking up the fossil carbon*".

PVC is often criticised for its inertness and persistence in the environment, and landfill situations in particular. However, it should be noted that there are numerous technologies currently available for the conversion of PVC waste into less troublesome materials, some of which might find commercial use in the future (Jaksland *et al*. 2000; Yoshioka *et al*. 2000). Alternatively, PVC could prove to be a cheap source of chlorine for the recovery of metals in wastes (Fray 1999).

Both rigid and flexible PVC products are suitable for recycling. PVC building products in particular offer the ideal opportunity for reuse/recycling, given their easy identification and increasing availability. Significant measures are needed to improve the recycling record of PVC.

7. ALTERNATIVES

The usefulness of the available scientific information concerning alternatives to PVC in its building product applications is questionable. Comparative laboratory studies are often limited to a small range of materials and comparisons between the findings of various laboratories usually difficult to make. Nevertheless, PVC products in general rank average overall on the criteria under examination. For example, "*in the majority of assessment methods, PVC pipe ends up between the other alternatives, ie. there are better and worse pipe materials from an environmental point of view*" (KEMI 1996). And "*Prognos sees more favourable prospects for PVC and HDPE in the short and medium term than for their competitors made of vitrified clay or cast iron*" (Prognos 1999).

Other examples include window frames where the Prognos Study (Prognos 1999) found that "*PVC windows do not currently have any price advantages, but in the short and medium term they offer overall more favourable prospects than the alternatives of wood and aluminium windows*". The German FEA (UBA 1999) agreed stating that "*the life-cycle assessments available today on the basis of existing method developments show that wood and PVC window frames are roughly equivalent and that aluminium profiles have disadvantages*". For packaging film, such as transparent fresh meat packaging, "*PVC is practically without*

competition due to its specific technical properties … thus making a positive contribution to sustainability" (Prognos 1999). For PVC cables "*Prognos only gives them favourable prospects in the short term*" with identified disadvantages including "*potential risks such as the consequences of fires, the use of plasticisers and, sometimes, flame protection agents, as well as the greater weight of the material*" (Prognos 1999).

Several attempts have been made to apply the newly emerged method of 'life cycle analysis' (LCA) to PVC. The problems with LCA are that it requires reliable data, which is often lacking for PVC, and often makes use of inappropriate toxicity data for emissions. Tukker (1998) summarises the uncertainty in LCA with the warning that "*until approaches have been worked out that deal with paradigmatic, modelling and data uncertainty, all current methods for toxicity impact assessment should be very carefully applied*" concluding that all should "*… refrain from publishing anything that looks like a guide for impact assessment, as far as it concerns themes that probably suffer from high uncertainties …*". In the findings of a report on the life cycle assessment of various pipeline systems Reusser (1998) stated, amongst other things, that "*a preference for certain materials is not justified on the basis of the LCA study findings*". For these reasons, meaningful outcomes from the various LCA's of PVC products are difficult to obtain.

In all its applications PVC can be replaced with existing materials, such as concrete, polyethylene, polypropylene, PET, ductile iron, vitreous clay, latex, wood, steel, and fibreglass, which under certain circumstances might be considered more environmentally sound. However, few of the alternatives to PVC have undergone as extensive an evaluation (if somewhat flawed) as the material they are to replace. The Swedish National Chemicals Inspectorate find that "*PVC pipes can be replaced with concrete, polyethylene, polypropylene, ductile iron, fibreglass-reinforced and rust-resistant pipes. The choice of pipe is controlled by price, internal and external influences of pressure, chemicals and biological activity. In acid soil cement pipes and iron-based pipes can corrode. Polyolefins do not cope with high temperatures. Heavy pipe materials are worse from a working environment viewpoint*" (KEMI 1996). In their study on the ecological and environmental aspects of PVC the German FEA (UBA 1999) have recommended that alternatives to plasticised PVC be investigated while substitution of rigid PVC "*… would not result in a substantial reduction of environmental risks …*".

There is little conclusive evidence, including LCA information, available that shows that PVC in its building and construction applications has significantly more effect on the environment than its alternative materials.

8. CONCLUSIONS

PVC has for a number of years been the subject of intense and often heated debate in some quarters over its possible detrimental health and environmental effects. Initially the fact that PVC contains a large amount of the element chlorine prompted calls for its banning by some. The issue was not the chlorine itself but rather its contribution to the formation of toxic chemicals, namely dioxins, during its combustion or incineration. It has been shown in this report that the relative contribution of PVC to the world-wide formation of PCDD/Fs is insignificant. However, the potential exists for PVC to add to the formation of these compounds and also the release of other pollutants, namely heavy metals and chlorinated paraffins, into the environment.

The scientific evidence concerning adverse health and environmental effects of phthalates is far from conclusive. This report shows that for the most part phthalates are non-toxic to humans at the levels encountered during the normal use of plasticised PVC items and in the environment. However, the unresolved issue of the toxicity of some phthalates being mediated by endocrine disrupting mechanisms remains. At present there is no scientifically valid reason for the banning or limiting of phthalates in their present uses.

It is noted that the landfill disposal of PVC is not detrimental to the environment. However, the use of heavy metal stabilisers and certain flame retardants in PVC products needs to be seriously examined in order to reduce the potential for their release into the environment. Finally, efforts need to be increased, particularly in the building and construction industries, to improve the recycling rates of PVC products.

9. RECOMMENDATIONS

The scientific basis for recommendations concerning PVC and its use in relation to building and construction is whether the product poses a risk to human health or the environment. For this purpose the known hazards associated with PVC are considered and the following recommendations made:

1. The use of PVC additives with known high toxicity be discontinued (in particular cadmium).
2. The scientific issues surrounding phthalates and the potential for their toxicity to be mediated by endocrine disruption mechanisms should be closely monitored to enable rapid response to community concerns.

3. There is the possibility that future studies will show that phthalates are hazardous therefore steps should be taken now into researching and developing alternatives to phthalate plasticisers.

4. Measures be taken to significantly improve the recycling of PVC building products.

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12. GLOSSARY

BBP	Butyl benzyl phthalate
CERHR	Center for the Evaluation of Risks to Human Reproduction
Ca	Calcium
CO	Carbon monoxide
CSIRO	Commonwealth Scientific and Industrial Research Organisation
DBP	Dibutyl phthalate
DDT	Chlorinated insecticide
DEHA	Di-2-ethylhexyl adipate
DEHP	Di-2-ethylhexyl phthalate
DIDP	Diisodecyl phthalate
DINP	Diisononyl phthalate
EDC	Ethylene dichloride or 1,2-dichloroethane
FEA/UBA	German Federal Environment Agency
HCl	Hydrogen chloride
HDPE	High-density polyethylene
LCA	Life cycle analysis
MSW	Municipal solid waste
PAH	Polycyclic aromatic hydrocarbon
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo-*p*-dioxin
PCDF	Polychlorinated dibenzofuran
PET	Polyethylene terephthalate
PMMA	Polymethyl methacrylate
PP	Polypropylene
pPVC	Plasticised or flexible PVC
PS	Polystyrene
PVC	Poly(vinyl chloride)
TCDD	2,3,7,8-Tetrachlorodibenzo-*p*-dioxin
TDI	Tolerable Daily Intake
TEF	Toxic Equivalence Factor
TEq or TEQ	Toxic Equivalency Quotient (unit of toxicity for a dioxin mixture)
uPVC	Unplasticised or rigid PVC
US EPA	United States Environment Protection Agency
UV	Ultra-violet radiation
VCM	Vinyl chloride monomer
WHO	World Health Organisation
Zn	Zinc

∞ Changes in the understanding of the relative toxicity of individual PCDD/F congeners has led to the situation where several different TEQs definitions have been employed over the years. Thus TEQ-(NATO), (i)-TEQ or I-TEQ (International) and the new WHO TEQ have been used at different times (van den Berg *et al*. 2000; van Leeuwen *et al*. 2000).

Δ Taucher *et al*. use the NATO TEQ whilst Lohmann and Jones use the international TEQ.

◊ Recently, the NSW EPA licensed an Australian company to commission a MSW-type incinerator in Wollongong which utilises new technology that significantly reduces dioxin emissions.