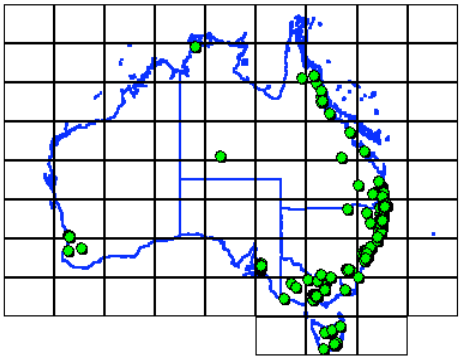
NATIONAL TOXICS NETWORK



RESPONSE TO AUSTRALIAN VINYL COUNCILS LETTER 29th January 2007

The Australian Vinyl Council in their letter of the 29th January raises three main points of contention; that is

1. PVC has been treated differently to other products.
2. There is no evidence that Phthalates have harmed humans.
3. PVC does not emit VOCs to a greater extent than other products.

This report will address these three issues.

1.'PVC has been treated differently to other building products.'

General Response:
It is appropriate for PVC to be treated differently from other building or consumer products for the following reasons:

- ***PVC uniquely produces dioxins both in its production and in its waste phase, through incineration and uncontrolled burning;***
- ***PVC does not share the reuse or recycling potential of other products;***
- ***The production of PVC results in significant air emissions of very toxic chemicals, eg vinyl monomer;***
- ***In its use phase PVC emits and exposes children to phthalates and bis-phenol A; both of which can affect sexual development; and***
- ***In its waste phase, the disposal of PVC may result in emissions of dioxins, furans and coplanar PCBs, and cadmium and lead.***

PVC leads to dioxin formation

The Green Home Guide correctly stated that 'dioxins and furans are by products of PVC' and are chemicals known to damage living things. The Vinyl Council's response acknowledges that dioxins are emitted in the PVC production process. However, it makes no reference to the fact that the production of ethylene dichloride (EDC) essential to the manufacture of PVC has been assessed as one of the world's largest sources of dioxin.[1]

In fact, PVC in its waste phase is the "…the single largest chlorine source in municipal waste, and has a significant role in the formation of dioxins and other toxic polychlorinated hydrocarbons in many countries."[2] As dioxins are a group of highly toxic substances that are capable of transboundary movement and bioaccumulate in living things, concerns for dioxin generation through the incineration of PVC are not restricted to national borders.

Similarly there is a clear relationship between PVC and dioxin emissions from incineration such as medical or quarantine incinerators. Many researchers have found a positive relationship between the input of PVC and other chlorine sources with dioxin outputs from incineration including fly ash.[3] Katami et al. (2002) concluded "PVC

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[1] Evers, E. 1989. *The formation of polychlorinated dibenzofurans and polychlorinated dibenzo-p-dioxins and related compounds during oxyhydrochlorination of ethylene*. Amsterdam: University of Amsterdam, Department of Environmental and Toxicological Chemistry.

[2] Choi et al., (2007). Suppression of Dioxin Emission in Co-Incineration of Poly(vinyl Chloride) with TiO2-Encapsulating Polystyrene. *Environ. Sci. Technol*. 41: 5833- 5838

[3] For example, see:
-- Oberg, T., Ohrstrom, T., (2003) Chlorinated aromatics from combustion: Influence of chlorine, combustion conditions and catalytic activity. *Environ. Sci. Technol*. 37: 3995-4000.
-- Hunsinger, H., Jay, K., Vehlow, J., (2000) Formation and destruction of PCDD/F inside a grate furnace. *Organohalogen Cpds.* 46:86-89.
-- Yamamura, K., Ikeguchi, T., Uehara, H., (1999) Study on the emission of dioxins from various industrial wastes incinerators. *Organohalogen Cpds*. 41: 287-292.
-- Tagashira, K., Torii, I., Myouyou, K., Takeda, K., Mizuko, T., Takushita, Y., (1999) Combustion characteristics and dioxin behavior of waste fired CFB. *Chemical Engineering Science* 54:5599-5607.
-- Costner, P.,(1998) Correlation of chlorine input and PCDD/PCDF emissions at a full-scale hazardous waste incinerator. *Organohalogen Cpds*. 36: 147 – 152.
-- Tuppurainen, K., Halonen, I., Ruokojarvi, P., Tarhanen, J., Ruuskanen, J., (1998) Formation of Hoenich, N. A, and Pearce, C.,'Medical waste production and disposal arising from renal replacement therapy', Advances in Renal Replacement Therapy, 9, 57 (2002)PCDDs and PCDFs in municipal waste incineration and its inhibition mechanisms: A review. *Chemosphere* 36: 1493-1511.
-- Manninen, H., Peltola, K., Ruuskanen, J., (1997) Co-combustion of refuse-derived and packaging-derived fuels (RDF and PDF) with conventional fuels. *Waste Manage. Res.*15:137-147.
-- Manninen, H., Perkio, A., Vartiainen, T., Ruuskanen, J., (1996) Formation of PCDD/PCDF: Effect of fuel and fly ash composition on the formation of PCDD/PCDF in the cocombustion of refuse-derived and packaging-derived fuels. *Environ. Sci. & Pollut. Res*. 3 (3): 129-134.
-- Wanke, T., Vehlow, J., (1996) The influence of flame retarded plastic foams upon the formation of Br containing dibenzo-p-dioxins and dibenzofurans in a MSWI. *Organohalogen Compounds* 28: 530-535.
-- Huotari, J., Vesterinen, R., (1996) PCDD/F emissions from co-combustion of RDF with peat, wood waste, and coal in FBC boilers. *Haz. Waste & Haz. Materials* 13(1): 1-9.
-- Vesterinen, R., Flyktman, M., (1996) Organic emissions from co-combustion of RDF with wood chips and milled peat in a bubbling fluidized bed boiler. *Chemosphere* 32(4): 681-689.
-- Moller, S., Larsen, J., Jelnes, J.E., Faergemann, H., Ottosen, L.M., Knudsen, F.E., "Environmental Aspects of PVC." Environmental Project No. 313. Denmark: Ministry of the Environment, Danish Environmental Protection Agency, 1995.
-- Thomas, V.M., Spiro, T.G., (1995) An estimation of dioxin emissions in the United States. *Toxicol. and Environ. Chemistry* 50:1-37., Katami, T., Yasuhara, A., Okuda, T. and Shibamoto, T. (2002) Formation of PCDDs, PCDFs and coplanar PCBs from polyvinyl chloride during combustion in an incinerator. *Environmental Science and Technology* 36(6): 1320-1324.

contributes significantly to the formation of PCDDs, PCDFs and coplanar PCBs from mixtures of plastics upon combustion."[4] Others (Hatanaka et al 2000), while noting that incinerator operating conditions can be the primary influencing factor in dioxin formation, also confirm a clear role for PVC, among other contributors to chlorine content of waste, in the formation of elevated quantities of dioxins.[5]

There is also a clear link between the presence of PVC and dioxin formation in the case of uncontrolled burning of domestic waste (eg bin-burning or backyard burning). While backyard burning is not prevalent in Australia, illegal or accidental fires in landfills are. Uncontrolled fires can lead to high emissions of polychlorinated dioxins and polychlorinated dibenzofurans into the surrounding environment. The presence of chlorine is necessary for the forming of dioxins, with PVC being a significant source of chlorine. The chlorine content of wood is just 0.028% compared to 57% for PVC; hence wood used in construction can be viewed as a relatively insignificant source of dioxins on a comparative weight basis. Uncontrolled fires such as those that occur in accidental building fires have high emissions comparable to the emissions of controlled incineration of combustibles.[6] The European Commission reported that the uncontrolled burning of 200 kilograms of PVC in Sweden resulted in 3 milligrams of dioxin.[7]

The DEH 2004 Report state that as the PVC level in the waste is increased from 0 to 1%, the dioxin emissions increase seven fold. The report also note that the mutagenicity of particulates from combustion of PVC is higher than those produced from the combustion of other commodity plastics such as PS, PET, and PE.

While the production of other building products also produces dioxins as evident in cement kiln emissions (particularly if they are burning hazardous waste) and some metal products based on copper, steel and aluminium, they do not have the same problem with dioxin emissions in their post use phase. We also note that there are many other toxic emissions from the production of PVC including, for example, Australian Vinyls'

--Hatanaka, T., Imagawa, T. Takeuchi, M. (2000) Formation of PCDD/Fs in artificial solid waste incineration in a laboratory-scale fluidised-bed reactor: Influence of contents and forms of chlorine sources in high temperature combustion. *Environmental Science and Technology* 34(18): 3920-3924

--Wikstrom, E., & Marklund, S. (2001) The influence of level and source on the formation of mono- to octa-chlorinated dibenzo-p-dioxins, dibenzofurans and coplanar polychlorinated biphenyls during combustion of an artificial municipal waste. *Chemosphere* 43(2): 227-234

--Yasuhara, A., Katami, T., Okuda, T., Ohno, N., & Shibamoto, T. (2001)
Formation of dioxins during the combustion of newspapers in the presence of sodium chloride and poly(vinyl chloride). *Environmental Science and Technology* 35(7): 1373-1378

[4] Katami, T., Yasuhara, A., Okuda, T. and Shibamoto, T. (2002) Formation of PCDDs, PCDFs and coplanar PCBs from polyvinyl chloride during combustion in an incinerator. *Environmental Science and Technology* 36(6): 1320-1324.

[5] Hatanaka, T., Imagawa, T. Takeuchi, M. (2000) Formation of PCDD/Fs in artificial solid waste incineration in a laboratory-scale fluidised-bed reactor: Influence of contents and forms of chlorine sources in high temperature combustion. *Environmental Science and Technology* 34(18): 3920-3924.

[6] Dept of Environment and Heritage, 2004 End of Life Environmental Issues with PVC in Australia, authored by Dr John Scheirs, August 2003.

[7] Life cycle assessment of PVC and of principal competing materials, Commissioned by the European Commission, July 2004

emissions to air of significant amounts (i.e., tonnes per annum) of the very toxic and carcinogenic vinyl chloride monomer.[8]

With limited production of PVC in Australia, much of the manufactured PVC goods sold here are imported from overseas facilities. This is particular the case with home goods, toys and clothing, which is of relevance to the Green Home Guide. Little if anything is known about dioxin emissions from PVC production in the developing world including China.

PVC is not recycled

Since most PVC is not recyclable, much of the PVC waste finds its way into Australian landfills. According the Department of Environment and Heritage (DEH 2004) Report, the total consumption of PVC resin in Australia for 2001 was some 194,500 tonnes. In addition some 31,000 tonnes of PVC was imported in the form of finished PVC products. This gave an overall PVC consumption figure of around 225,500 tonnes of which approximately only 6,000 tonnes or 2.7% is recycled per annum. [9]

In addition to its own miniscule recycling rate, PVC has a negative impact on the recycling of other plastics. The DEH 2004 report acknowledges that there are major impediments to PVC recovery and recycling, such as the presence of various types of contamination and commingled (incompatible) polymers mixed with the PVC stream, the relative thermal instability of PVC and the resulting potential machinery corrosion due to hydrochloric acid attack.

PVC contains and leaches toxic metal stabilizers

In 2006, an investigation of soft plastic toys in India was presented to the Intergovernmental Forum on Chemical Safety.[10] The key findings were that 77 of 111 samples were of PVC plastic with lead and cadmium found in all tested PVC samples in varying concentrations. While the lead ranged from 0.65 ppm to 2104 ppm with an average of 112.51ppm, cadmium ranged from 0.016 ppm to 188 ppm with an average of 15.71ppm.

In August 2007, the US based Toys "R" Us Inc. announced it was removing all vinyl baby bibs from its stores after two bibs made in China showed lead levels that exceeded Toys "R" Us standards.[11]

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[8] Australian PVC manufacturers, Australian Vinyls reported estimated emissions to the Federal National Pollutant Inventory of 3,200 kg of the toxic vinyl chloride monomer to air in 2004-2005 and 2,300 kg in July 2005 to June 2006 Available at http://www.npi.gov.au

[9] Dept of Environment and Heritage, 2004 End of Life Environmental Issues with PVC in Australia, authored by Dr John Scheirs, August 2003. According to report Cryogrind Pty Ltd recycles approximately 2,500 tonne per year of PVC (Siganakis, 2002). The other major PVC recycler in Australia, Nylex SRM (Dandenong, Vic.) recycles approximately 3,000 t/a of PVC scrap and convert it to reformulated compounds. However, this includes 30 t/m of flexible PVC that they presently import from the UK. There are another 6 recycling facilities treating very small amounts

[10] See ttp://www.who.int/ifcs/documents/forums/forum5/toying_agarwal.pdf. Toying with Toxics, An investigation of lead and cadmium in soft plastic toys in three cities in India, Ravi Agarwal ,Toxics Link. The full report is available at http://www.toxiclink.org

[11] Toys "R" Us Recalls Bibs Over Lead Threat, Vinyl Baby Bibs Manufactured In China Found To Exceed Lead Levels, August 21, 2007. Available at http://www.cbsnews.com/stories/2007/08/17/health/main3180464.shtml

Organotins are also found in PVC products including food packing materials,[12] and other building materials. A study in Japan found organotins in children's PVC toys including face masks, balls, soft toys and food toys.[13] Elevated levels of organotins, particularly tributyltin, have also been found in PVC flooring and somewhat lower concentrations in carpets.[14] Organotins are toxic at low levels of exposure and are transported through the placenta, with demonstrated adverse developmental effects.[15]

Once in landfill, lead based stabilisers can be released from PVC[16] contributing to the heavy metals content in the municipal solid waste. Emissions of heavy metals from PVC are evident in the early acidogenic stage of the landfill development. The release of heavy metal stabilizers can be accelerated by high temperatures that can occur in large landfill sites under aerobic thermophilic conditions.[17]

Cadmium stabilisers in PVC have been estimated to make up 30-40% of cadmium in municipal waste in Sweden.[18] Plasticizer loss from PVC also occurs in landfill [19] with plasticized products showing a higher inclination to release their stabilizers. [20]

PVC linked to other POPs

Besides dioxins and furans, PVC is linked to two new POPs currently proposed for addition to the Stockholm Convention; short-chained chlorinated parrafins (SCCPs) and pentachlorobenzene (PeCB). The Draft Risk Profile on Short Chained Chlorinated Paraffins (SCCPs) acknowledges that a major source of releases of SCCPs is the formulation and manufacturing of products containing SCCPs, such as PVC plastics. [21]

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[12] Fromme, H., Mattulat, A., Lahrz, T., Ruden, H., (2004) Occurrence of organotin compounds in house dust in Berlin (Germany). *Chemosphere*. 58(10), 1377-83.

[13] Ohno, H., Suzuki, M., Aoyama, T., Mitani, K., 2003. Determination of organotins in polyvinyl chloride toys. *Shokuhin Eiseigaku Zasshi* 44: 208-212. [Abstract]

[14] Allsopp, A., Santillo, D. & Johnston, P., 2001. Poison Underfoot: Hazardous Chemicals in PVC Flooring and Hazardous Chemicals in Carpets. ISBN 90- 73361- 68- 0. Amsterdam: Greenpeace International.

[15] Kergosien DH, Rice CD. 1998. Macrophage secretory function is enhanced by low doses of tributyltinoxide (TBTO), but not tributyltin- chloride (TBTC1). Arch Environ Contam Toxicol 34(3): 223- 228.

[16] Health Canada (1996) Lead Hazard posed by PVC Mini-blinds Press Release June 25

[17] Argus Study for European Commission DGXI.E.3, 'The behavior of PVC in Landfills, Final Report, February 2000, prepared by ARGUS in association with University Rostock-Prof. J. Spillmann, Carl Broa|s and Sigma Plan S.A., p. 33 (2000).

[18] W. Hogland and F. Tamaddon, (1993) Review of Cadmium in Plastic Waste in Sweden, *Waste Management & Research*, Vol. 11, No. 4, 287-295

[19] Monney L. Jamois-Tasserie M. Dubois C. Lallet P. Villa F. Renaud C., (2001) Plasticiser migration and structural changes in an aged poly(vinyl chloride) coating, *Polym. Degrad. Stabil*., 72, 459

[20] Mersiowsky I., '(2002) Fate of PVC polymer, plasticizers, and stabilizers in landfilled waste', *J. of Vinyl Technology*, 8, 36

[21] See http://www.pops.int/documents/meetings/poprc/drprofile/drp/DraftRiskProfile_SCCP.pdf

The Draft Risk Profile on Pentachlorobenzene notes that, "PeCB formation has been observed during combustion of PVC, a common component of municipal solid waste."[22]

2. 'Phthalates have not harmed humans'

General Response:
Phthalates are detected in the blood and urine of both adults and children. There is growing evidence of adverse effects at the normal levels estimated for an adult. Exposure to phthalates and their metabolites are associated with a broad range of health effects in humans, including asthma and other respiratory problems, rhinitis and eczema in children; premature breast development in female children; deteriorated semen quality and DNA damage in men; congenital genital malformations in boys; and reductions in reproductive hormones.

As the Vinyl Council has not challenged the overwhelming evidence that phthalates leach from PVC products providing a ready source of exposure, we will not specifically address this issue. We would however, like to acknowledge that phthalates such as di(2-ethylhexyl) phthalate (DEHP) can enter the environment *via* a number of different routes: during plasticizer production; during plasticizer distribution; during incorporation into PVC resin; during disposal in industrial and municipal landfills; leaching from PVC products during use (e.g. floor tiles, greenhouse film) and leaching from PVC products after use as with *in situ* disposal. DEHP has been shown to migrate into food from certain food wraps during storage. [23]

Phthalates are detected in the blood and urine of both adults and children,[24] with the US National Toxicology Program (NTP) expressing concern over the adverse development of babies born to pregnant women who are exposed to DEHP, at current levels estimated for an adult.

Exposure to phthalates and their metabolites have been associated with a broad range of health effects in humans, including:

- asthma and other respiratory problems, rhinitis and eczema in children;
- deteriorated semen quality in men;
- DNA damage;
- adverse male genital development; and
- reduction in reproductive hormones.

[22] See http://www.pops.int/documents/meetings/poprc/drprofile/drp/DraftRiskProfile_PeCB.pdf

[23] Dept of Environment and Heritage, 2004 End of Life Environmental Issues with PVC in Australia, authored by Dr John Scheirs, August 2003

[24] Second National Report on Human Exposure to Environmental Chemicals (January 2003), Department of Health and Human Services, Centers for Disease Control and Prevention

Two studies in the late 1990s (Jaakkola et al. 1999 [25] and Øie et al 1997[26]) found that the presence of plasticizers in surface materials indoors can increase the risk of bronchial obstructions, asthma, and perhaps the susceptibility to respiratory infections. The later noted that indoor inhalation of DEHP-adsorbed particulate matter could be as or more important than inhalation of vapor phase DEHP.

These findings were supported by a Swedish study (Bornehag et al 2004), [27] which has linked a range of phthalates to asthma, allergic rhinitis (hay fever), and eczema. Researchers took dust samples from the moulding and shelves in the children's bedrooms. Samples containing higher concentrations of phthalates were associated with symptoms of asthma, hay fever, and eczema. Notably, PVC flooring in the children's bedrooms was associated with symptoms. The Swedish study found that children exposed to household dust with the greatest concentrations of DEHP were 2.9 times as likely to have asthma as were children exposed to the lowest concentrations of that phthalate. Similarly, children in homes with the greatest concentrations of butyl benzyl phthalate were 3.0 and 2.6 times as likely as the other children to have rhinitis and eczema, respectively.

In 2002, researchers[28] explored whether general levels of phthalates in the U.S. population were associated with altered semen quality and found suggestive evidence of an association between high mono-benzyl phthalate (MBzP) levels and low sperm counts and between high mono-methy phthalate (MMP) and poor sperm morphology. Mono-n-butyl phthalate (MBP), MBzP and MMP were associated with altered semen quality.

In a related study, in 2003[29] it was found that urinary monoethyl phthalate (MEP), at environmental levels, is associated with increased DNA damage in sperm. Rozati et al. (2002)[30] had found that the concentration of phthalate esters was significantly higher in infertile men compared with controls and concluded that they may be instrumental in the deterioration of semen quality in infertile men without an obvious etiology.

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[25] Jaakkola, J.J.K., Øie, L., Nafstad, P., Botten, G., Lødrup-Carlson, K.C., Samuelsen, S.O. and Magnus, P. (1999) Interior surface materials in the home and the development of bronchial obstruction in young children in Oslo, Norway, *Am. J. Public Health*, 89, 188–192

[26] Øie, L., Hersoug, L.G. and Madsen, J.Ø. (1997) Residential exposure to plasticizers and its possible role in the pathogenesis of asthma, *Environ. Health. Perspect.*, 105, 972–978.

[27] Carl-Gustaf Bornehag, Jan Sundell, Charles J. Weschler, Torben Sigsgaard, Björn Lundgren, Mikael Hasselgren, and Linda Hägerhed-Engman (2004) The Association between Asthma and Allergic Symptoms in Children and Phthalates in House Dust: A Nested Case–Control Study *Environmental Health Perspectives* Volume 112, Number 14, 1393-1397

[28] Duty, S., Silva, M., Barr, D., Brock, J., Ryan, L., Zuying, C., Herrick, R., Christiani, D., Hauser, R., (2002). Urinary phthalate monoesters at general populations exposure levels are associated with altered semen quality. *Epidemiology* July 2002, Volume 13 Number 4 Supplement

[29] Duty, S., Singh, N., Silva, M., Barr, D., Brock, J., Ryan, L., Herrick, R., Christiani, D., Hauser, R., (2003). The relationship between environmental exposures to phthalates and DNA damage in human sperm using the Neutral Comet Assay. *Environ. Health Perspect*. 111: 1164-1169.

[30] Rozati, R., Reddy, R., Reddanna, P., Mujtaba, R., (2002) Role of environmental estrogens in the deterioration of male factor fertility. *Fertility & Sterility* 78: 1187-1194

Impacts on women had been suggested in 2003 study [31], which linked environmental contamination with DEHP, through its metabolite MEHP, and the suppression of estradiol production in the ovary, leading to anovulation.

A 2004 study [32] demonstrated that the daily exposure to DEHP of 3–30 ug/kg body weight/day comes close to the TDI of 37 ug/kg body weight/day of the EU Scientific Committee for Toxicity, Ecotoxicity and the Environment (CSTEE). The study found that the RfD (reference dose) of the US Environmental Protection Agency (EPA) of 20 ug/kg body weight/day is even exceeded and the 3–30 ug/kg body weight/day may be increased by 2–3 orders of magnitude for infants undergoing intensive therapeutic interventions. Thus, the actual exposure to DEHP is higher than previously believed and the TDI can be exceeded within the general population considerably. In particular, it was shown that nursery school children aged 2–6 years are probably exposed to twice as much DEHP as adults. The authors argue that the available toxicity data and the limited, but suggestive human exposure data are cause for serious concern that DEHP exposure may be detrimental to human fertility and reproduction. In particular, since the blood-testis barrier forms just before puberty in humans, permeability of the blood-testis barrier is increased in children and particularly in newborns, whose testicles are still developing. As a consequence, male newborns are thought to be at the greatest potential risk.

A 2005 [33] study showed how the urinary concentrations of four phthalate metabolites [monoethyl phthalate (MEP), mono-n-butyl phthalate (MBP), monobenzyl phthalate (MBzP), and monoisobutyl phthalate (MiBP)] were inversely related to anogenital index (AGI). The authors note that the associations between male genital development and phthalate exposure seen are consistent with the phthalate-related syndrome of incomplete virilization that had been reported in prenatally exposed rodents. These data further supports the hypothesis that prenatal phthalate exposure at environmental levels can adversely affect male reproductive development in humans.

In 2005, the National Toxicology Program (NTP) Center for the Evaluation of Risks to Human Reproduction (CERHR) convened an expert panel to re-evaluate the reproductive and developmental toxicities of di(2-ethylhexyl)phthalate (DEHP). The expert panel expressed concern that DEHP exposure can adversely affect reproductive development in infants less than one year old. Where DEHP/MEHP exposure is high due to medical procedures in infants the expert panel had serious concern that such exposures may adversely affect male reproductive tract development and function.[34]

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[31] *Tara Lovekamp-Swan and Barbara J. Davis (2003)* Mechanisms of Phthalate Ester Toxicity in the Female Reproductive System, *Environ Health Perspect* 111:139–145

[32] Latini, G., De Felice, C., Verrotti, A., (2004) Plasticizers, infant nutrition and reproductive health. *Reproductive Toxicology* Vol. 19:1, 27-33.

[33] Shanna H. Swan, Katharina M. Main, Fan Liu, Sara L. Stewart, Robin L. Kruse, Antonia M. Calafat, Catherine S. Mao, J. Bruce Redmon, Christine L. Ternand, Shannon Sullivan, J. Lynn Teague, and the Study for Future Families Research Team, (2005) Decrease in Anogenital Distance among Male Infants with Prenatal Phthalate Exposure, *Environmental Health Perspectives* Vol/ 113 : 8

[34] October 14, 2005 MEETING SUMMARY NATIONAL TOXICOLOGY PROGRAM CENTER FOR THE EVALUATION OF RISKS TO HUMAN REPRODUCTION EXPERT PANEL RE-EVALUATION OF DEHP OCTOBER 10–12, 2005

A 2006 study[35] supported these concerns concluding that data on reproductive hormone profiles and phthalate exposures in newborn boys are in accordance with rodent data and suggest that human Leydig cell development and function may also be vulnerable to perinatal exposure to some phthalates. Their study supported other recent human data showing incomplete virilization in infant boys exposed to phthalates prenatally and supports previous findings that DBP, DEHP and its metabolite mono-2-ethylhexyl phthalate (mEHP), and di-isononyl phthalate (DiNP) show antiandrogenic effects diminishing fetal testosterone production.

In January 2006, many of these concerns were acknowledged by the European Commission in their News Alert Issue No 7, (January 2006) 'Phthalates May Affect Baby Boys'. The alert reviewed further evidence from another 2006 study,[36] which demonstrated that exposure to phthalates through lactation can trigger the reduction of reproductive hormones in baby boys. The contamination by six phthalate monoesters was measured in 130 breast milk samples from Danish and Finnish mothers included in a cohort study conducted from 1997 to 2001. The blood samples of their 3 months old sons were analyzed for sex-hormones. The results of the study showed that 3-months old boys exposed to higher concentrations of phthalate monoesters in breast milk, showed slight, but significant, decrease in levels of reproductive hormones, including the main male sex-hormone - testosterone. *Importantly, the range of concentrations of phthalates in breast milk samples appeared to be below the estimates of the tolerable daily intake levels (TDI).* However, they note that a direct comparison to TDI values was not possible in this study since exposure through lactation is only one of the possible routes of exposure to phthalates in children.

A very recent study (Ren-Shan Ge et al 2007) [37] concluded that contemporary epidemiological evidence indicates that boys born to women exposed to phthalates during pregnancy have an increased incidence of congenital genital malformations and spermatogenic dysfunction, signs of a condition referred to as testicular dysgenesis syndrome (TDS).

While DEHP remains the most prevalent plasticizer in PVC formulations and is also the dominant phthalate found in the environment, it is not the only phthalate of concern to human and animal health. Rats and mice fed di-isononyl phthalate (DINP) also showed an increase in liver cancers [38] and in the 2003 study[39] linking urinary MEP, at

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[35] Katharina M. Main, Gerda K. Mortensen, Marko M. Kaleva, Kirsten A. Boisen, Ida N. Damgaard, Marla Chellakooty, Ida M. Schmidt, Anne-Maarit Suomi, Helena E. Virtanen,2 Jørgen H. Petersen, Anna-Maria Andersson, Jorma Toppari, and Niels E. Skakkebæk, (2006) Human Breast Milk Contamination with Phthalates and Alterations of Endogenous Reproductive Hormones in Infants Three Months of Age, Vol 14:2 2006 • *Environmental Health Perspectives*

[36] Main, M.K. et al. (2006) "Human Breast Milk Contamination with Phthalates and Alterations of Endogenous Reproductive Hormones in Infants Three Months of Age", *Environmental Health Perspectives* 114 (1).

[37] Ren-Shan Ge, Guo-Rong Chen, Cigdem Tanrikut and Matthew P. Hardy, (2007) Phthalate ester toxicity in Leydig cells: Developmental timing and dosage considerations, *Reproductive Toxicology* Volume 23 : 3, 366-37

[38] Jacqueline H. Smith*, Jason S. Isenberg, George Pugh, Jr.*, Lisa M. Kamendulis, David Ackley, Arthur W. Lington* and James E. Klaunig (2002) Comparative *in Vivo* Hepatic Effects of Di-Isononyl Phthalate (DINP) and Related C_7–C_{11} Dialkyl

environmental levels, with increased DNA damage in sperm, the authors noted that previous *in vitro* studies had found di-*n*-butyl phthalate (DBP) and di-isobutyl phthalate (DiBP) to be genotoxic in human epithelial cells of the upper aerodigestive tract as well as in mucosal cells and lymphocytes.

It has been shown that phthalates can be hormone disruptors,[40] immunotoxins,[41] cancer promoters and/or reproductive and developmental toxins.[42] DEHP has been classified as a "probable human carcinogen" by the USEPA.

The 2006 EU announcement quoted by the Vinyl Council was based on the 2003 EU Risk Assessments of DINP and DIDP. In assessing DINP, risk assessors noted their difficulty in assessing the level of consumer exposure to DINP, "as DINP is not chemically bound to PVC, it can be released during the entire cycle of life of end products that are used by consumers." These end products are building materials (cables, floor covering, paints, etc.), car undercoating, clothes, gloves, shoes and boots, toys and child care articles. [43]

In relation to di-isodecyl phthalate (DIDP) they note that the calculations are based on the assumption that it is not used in toys and "in case DIDP should be a substitute for other phthalates in toys in the future, margin of safety (MOS) of 18.8, derived from hepatic toxicity in dogs, would not be considered sufficient to protect infants." They conclude that owing to the uncertainty on the applicability of the NOAEL (no observable effect level of 16.5 mg/kg bw/d) for reduced offspring survival and the significance of the MOS (83 and 41, respectively without and with toys), no formal conclusion could be drawn."[44]

In assessing the impacts on humans, particularly children, NGOs have been vocal in their rejection of standard risk assessment methodologies that are based on single Tolerable

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Phthalates on Gap Junctional Intercellular Communication (GJIC), Peroxisomal Beta-Oxidation (PBOX), and DNA Synthesis in Rat and Mouse Liver, *Toxicological Sciences* 54, 312-321

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[43] EUROPEAN COMMISSION JOINT RESEARCH CENTRE Institute for Health and Consumer Protection European Chemicals Bureau I-21020 Ispra (VA) Italy "1,2-BENZENEDICARBOXYLIC ACID, DI-C8-10-BRANCHED ALKYL ESTERS, C9-RICH and DI-"ISONONYL" PHTHALATE (DINP) CAS Nos: 68515-48-0 and 28553-12-0 EINECS Nos: 271-090-9 and 249-079-5 Summary Risk Assessment Report 2003 Special Publication I.03.101

[44] EUROPEAN COMMISSION JOINT RESEARCH CENTRE Institute for Health and Consumer Protection European Chemicals Bureau I-21020 Ispra (VA) Italy "1,2-BENZENEDICARBOXYLIC ACID, DI-C9-11-BRANCHED ALKYL ESTERS, C10-RICH and DI-"ISODECYL" PHTHALATE (DIDP) CAS Nos: 68515-49-1 and 26761-40-0 EINECS Nos: 271-091-4 and 247-977-1 Summary Risk Assessment Report 2003 Special Publication I.03.103

Daily Intakes. This approach ignores the need for assessment of cumulative exposure to groups of 'like' chemicals (e.g., all phthalates, all reproductive toxins) and fails to acknowledge the importance of pulse exposure both prenatally through mother to foetus and postnatally through breast milk. The unique vulnerability of children to hazardous chemicals and the concept of "windows of susceptibility"[45] is well recognised by the World Health Organisation (WHO), the United Nations' Children's Fund (UNICEF) and the United Nations Environment Program (UNEP).[46] The 2003 EU Risk Assessments of DINP and DIDP fails to take into account these factors or the synergistic impacts of the many hundreds of man made chemicals present in umbilical cord blood, breast milk, adult fat and blood.

Phthalates are not the only plasticizers and additives used in PVC that are of concern. Bis-phenol A (BPA) used in plasticizers for PVC has been shown to leach from PVC products.[47] BPA is a bioaccumulative toxin and low doses are linked to adverse impacts on fetal prostate, breast, testicle, mammary glands as well as brain development in animals.

These studies demonstrate that phthalates and other PVC additives have harmed human health and continue to represent a significant risk to the health of children and newborns.

3. 'PVC does not emit VOCs to a greater extent than other products'.

General Response:
There are real world published case studies that have measured volatile organic compounds (VOCs) emissions from PVC materials and in some cases, to a greater extent than other building materials. There is a wide variability of emission rates and reduction factors among the numerous chemicals that make up the total VOCs.

A 3-year Finnish research project[48] has confirmed VOCs emissions from PVC. Material emissions were measured in seven buildings during the time of construction, in the newly finished, and in the 6 and 12 month-old buildings. The emission rates for volatile organic compounds (VOCs), formaldehyde, and ammonia were determined. The highest total VOCs (TVOC) emissions were measured in the newly finished buildings from the ceiling structure and from some of the PVC floor coverings.

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The authors noted that while some emissions levels went down over the 12 months period but others did not. Some emissions even increased in buildings during the follow-up period indicating the difference between emissions measured in the laboratory and on site from real structures.

In a 1999 Danish study,[49] new PVC (unfixed) flooring materials were assessed for emission rates of TVOCs. The highest emission rate for one material was 780 mg/(m2 ¡ h) and the lowest is 16 mg/(m2 ¡ h). The emission rates of TVOC decrease on the average approximately 60% from 4 to 26 weeks. However, there were differences in emission rates of individual chemicals. For a number of individual compounds the emission rate decreased little over 26 weeks. The authors conclude that based on the combination of high frequency, high initial emission rate and low reduction factor, this should prompt an evaluation of health and comfort effects. These compounds include T4 2-(2-Butoxyethoxy) ethanol, Butoxyethanol, 2-Ethyl-1-hexanol, Phenol, Hepta- and Octa-decane, hexadecane, Tri-methylbenzenes, Texanol-isobutyrate (TXIB), Cyclohexanone, 2-(2-Ethoxyethoxy) ethylacetate, Propylene glycol mono-methyl ether, Undecane.

A more recent 2003 Finnish study[50] investigated the presence of VOCs in an office building where workers complained of several respiratory, conjunctival and dermal symptoms, including an incidence of adult-onset asthma approximately nine-times higher than similar Finnish workers. They concluded the most probable single cause of the indoor air problem was the degradation of the PVC plastic floor coverings.

This was supported by the detection of emissions in both the indoor air and floor material samples indicating the degradation of PVC floor coverings. The plastic floor coverings, adhesives and the leveling layers were removed from 12 rooms and the underlying concrete slabs warmed to remove the diffused VOCs from these areas. After repairs, the concentrations of the VOCs were reduced, as was the prevalence of the employees' symptoms and several asthma patients' need for medication.

Phasing out PVC

Many responsible companies are already moving to eliminate PVC from their products, including the giant computer firm, Microsoft, which has long used PVC packaging. Companies such as Honda, Johnson and Johnson, Mattel, Nike, Sony and Walmart have developed PVC phase-out policies. Others including General Motors and telecommunications companies like German Telekom, Nippon Telegraph and Telephone of Japan have announced their intention to phaseout PVC.[51]

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[51] PVC-Free Future: A Review of Restrictions and PVC free Policies Worldwide, A list compiled by Greenpeace International, 9th edition, June 2003 Also see http://www.besafenet.com/pvc/companypolicies.htm

Conclusion

Based on the considerable published evidence of the adverse impacts of PVC production, use and disposal, and taking into account children's unique vulnerability to toxic chemicals and the Precautionary Principle, it is recommended that members of civil society reduce as far as possible their and their children's exposure to toxic man made chemicals such as PVC.

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