HEALTH RISK ASSESSMENT:
LEAD IN CHILDREN'S FACE PAINT

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GLOSSARY

Acute toxicity	1. *Adverse effects* of finite duration occurring within a short time (up to 14 d) after administration of a single *dose* (or *exposure* to a given *concentration*) of a test substance or after multiple doses (exposures), usually within 24 h of a starting point (which may be exposure to the *toxicant*, or loss of reserve capacity, or developmental change, etc.) 2. Ability of a substance to cause *adverse effects* within a short time of dosing or *exposure*
Adverse effect	A change in biochemistry, physiology, growth, development morphology, behaviour, or lifespan of an organism which results in impairment of functional capacity or impairment of capacity to compensate for additional stress or increase in susceptibility to other environmental influences
Benchmark response (BMR)	A specified change in biological response compared to background. For example, a 10% increase in the number of animals developing fatty liver compared with untreated animals
Dermal	Cutaneous, pertaining to the skin
Dose	Total amount of a substance administered to, taken up, or absorbed by an organism, organ, or tissue
Dose response	Association between dose and the incidence of a defined biological effect in an exposed population
Dose response assessment	Analysis of the relationship between the total amount of an agent administered to, taken up by, or absorbed by an organism, system, or (sub)population and the changes developed in that organism, system, or (sub)population in reaction to that agent, and inferences derived from such an analysis with respect to the entire population. Dose–response assessment is the second of four steps in risk assessment
Exposure assessment	Evaluation of the exposure of an organism, system, or (sub)population to an agent (and its derivatives). Exposure assessment is the third step in the process of risk assessment
Glomerular filtration rate	The flow rate of filtered fluid through the kidneys
Haematologic	Pertaining to or emanating from blood cells
Haematopoiesis	Formation of blood cellular components
Harm	An adverse effect. Damage or adverse effect to a population, species, individual organism, organ, tissue, or cell

Hazard identification	The identification of the type and nature of adverse effects that an agent has an inherent capacity to cause in an organism, system, or (sub)population. Hazard identification is the first stage in hazard assessment and the first of four steps in risk assessment
Hypertension	High blood pressure
Incidence	Number of occurrences of illness commencing, injury, or of persons falling ill, during a given period in a specific population usually expressed as a rate
Injury	Any physical harm or damage serious enough to warrant medical treatment by a health professional either at the scene or in a hospital or primary care practice
Margin of exposure (MOE)	Ratio between a defined point on the dose-response curve (eg. NOAEL) for the adverse effect and the estimated human exposure
Neurogenesis	Development of nervous tissue
No observed adverse effects level (NOAEL)	Greatest concentration or amount of a substance, found by experiment or observation, that causes no alterations of morphology, functional capacity, growth, development, or life span of target organisms distinguishable from those observed in normal (control) organisms of the same species and strain under the same defined conditions of exposure
Ocular	Pertaining to or via the eyes
Oral	Pertaining to or via the mouth
Permanent harm	An adverse effect from which the subject does not recover
Risk characterisation	The qualitative and, wherever possible, quantitative determination, including attendant uncertainties, of the probability of occurrence of known and potential adverse effects of an agent in a given organism, system, or (sub)population, under defined exposure conditions. Risk characterisation is the fourth step in the risk assessment process
Synaptic pruning	Neurological regulatory processes, which facilitate changes in neural structure by reducing the overall number of neurons and synapses, leaving more efficient synaptic configurations
Synaptogenesis	Formation of synapses between neurons
Toxicological endpoints	An observable or measurable biological event or chemical concentration (e.g. metabolite concentration in a target tissue) used as an index of an effect of a chemical exposure

SUMMARY

Children's face paints have been found to occasionally contain very high concentrations of lead (>10,000 mg/L). Children's face paints are classified as cosmetic products. In New Zealand, cosmetic products are regulated under the Cosmetic Products Group Standard 2006 under the Hazardous Substances New Organisms Act 1996. Under the group standard, cosmetic products must not contain lead and its compounds. It is unknown how common use of children's face paints is in New Zealand. Six children's face paint exposure incidents have been reported to the New Zealand hazardous substances surveillance systems in the period 2009-2012, including four cases of ingestion and two of ocular exposure.

No case reports or epidemiological studies were found of adverse health effects due to the use of lead-containing children's face paints.

Exposure modelling was carried out assuming either weekly or two-monthly use of face paint by young (2-3 years) or older (11-16 years) children. Modelling used either the highest lead concentration reported for children's face paints (31,795 mg/L) or the current Australian regulatory limit (25 mg/L). The Australian regulatory limit became mandatory on 1 January 2010. For young children it was assumed that up to 5% of the applied face paint may be directly ingested by transfer from face to fingers to mouth. Risks were assessed by a margin of exposure (MOE) approach, comparing exposures to a $BMDL_{01}$ (lower 95th percentile confidence interval for the benchmark dose giving a 1 IQ point decrement in cognitive ability in children) derived by the European Food Safety Authority. A MOE less than one indicates potential for adverse health effect, while interpretation of MOEs greater than one depends on the magnitude and nature of the dose metric on which the benchmark dose is based and the relevance of the population in whom the BMDL was determined.

For children aged 11-16 years, MOEs range from 0.26 to 4000. The MOEs less than one are associated with use of face paint containing very high concentrations of lead (31,795 mg/L). For face paints containing lead at the Australian regulatory limit of 25 mg/L, MOEs are in the range 320-4000.

For children aged 2-3 years, MOEs range from 0.015 to 6000. The lowest MOEs are associated with the use of face paint containing very high concentrations of lead and with ingestion of a proportion (5%) of the applied face paint. For face paints containing lead at the Australian regulatory limit of 25 mg/L, MOEs are in the range 19-6000, irrespective of whether face paint is ingested or not. However, it should be noted that, while for most scenarios exposure estimates predict blood lead levels well below the benchmark dose, no threshold for the impact of lead on child cognitive ability has been established.

These results suggest that frequent use of children's face paints containing very high concentrations of lead has the potential to cause adverse health effects. Use of paints complying with regulatory lead limits, such as those in effect in Australia, represents a low to negligible risk.

Direct ingestion of children's face paints during exploratory behaviour is unlikely to be fatal, even at the highest observed lead concentrations (31,795 mg/L) and

assuming ingestion of a complete large pack of face paint (100 mL). However, if a single ingestion event is averaged to one year of daily exposure, ingestion of 100 mL of face paint containing 520 mg/L or ingestion of 1.6 mL of face paint containing 31,795 mg/L of lead would potentially raise blood lead levels above the New Zealand notification concentration (100 μg/L).

1. INTRODUCTION

The purpose of this report is to develop a generic health risk assessment for children's face paint containing lead. This report will only consider domestic, non-occupational, routine and incidental exposure to lead-containing face paint. Exposure scenarios will be developed for the most common or likely exposure events.

1.1 Consumer Products Description – Lead-Containing Children's Face Paint

Face paints are special water-based cosmetic paints, formulated for application to the face. Face paints are commonly used for theatre, face-painting booths at fairs and other public events and everyday play, including children's birthday parties (Sarantis et al 2009).

1.1.1 Prevalence of use

No data were found on the prevalence of face painting in New Zealand or internationally. The harmonised system (HS), used to describe goods in international trade, including those imported into New Zealand, does not allow identification of import quantities of face paint.

1.1.2 Lead in face paint

It is uncertain how high concentrations of lead come to be present in face paint. However, it has been suggested that lead may be present as a contaminant of mineral pigments used to colour face paint (Sarantis et al 2009).

Two main sources of information are available on the lead content of face paint:

- Surveys, usually conducted by consumer advocacy or consumer protection groups and
- The European Commission rapid alert system for non-food dangerous products (RAPEX)[1]

It should be noted that none of the surveys of lead in children's face paint have been published in the scientific literature and only one has been published in a fully accessible format (Sarantis et al 2009). Information on other surveys comes from summaries on organisational websites or news reports in the media. These latter sources invariably include a less than ideal level of detail.

Table 1 and 2 summarise available information on the lead content of face paint from these two sources.

[1] http://ec.europa.eu/consumers/safety/rapex/alerts/main/index.cfm?event=main.search accessed 21 November 2014

Table 1: Summary of surveys of lead in face paint

Survey country	Number of samples	Findings	Study reference
Australia	95	Products included finger, face and body paints, toy-style make up, modelling clay, play dough, plasticine, sticky 'goo' toys, art paints, crayons and pastels. One face paint contained excessive lead (>25 mg lead/kg). The country of manufacture of the high-lead item was not given	(Australian Competition & Consumer Commission 2011)
Canada	Not stated	Up to three samples contained excessive lead (>10 mg lead/kg). The country of manufacture of the high-lead item was not given	(Schmidt 2013)
New Zealand	15	One sample (from China) contained 15,200 mg lead/kg, the remaining 14 contained less than 10 mg lead/kg	(Consumer NZ 2014)
United Kingdom	Not stated	Lead levels in some face paints (from China) were reported to be as high as 16,900 mg lead/kg	(The Guardian 2012)
United States	10	Ten of 10 face paints[1] contained lead at concentrations in the range 0.08-0.65 mg lead/kg	(Sarantis et al 2009)

[1] Products were manufactured in USA (4), China (4), UK (1) and Spain (1)

Table 2: Summary of European Commission rapid alert system for non-food dangerous products (RAPEX) alerts for lead in face paint

Year	Product	Country of origin	Finding
2013	Face painting set	Unknown	Contained 41 (orange paint), 64 (yellow paint) and 39 (blue crayon/paint) mg lead/kg
2011	Children's face paint set	China	Yellow face paint contained 16,900 mg lead/kg
2009	Children's make-up - Face Painting Set	China	Contained 630 (yellow paint), 82 (red paint), 6830 (yellow crayon) and 1070 (orange paint) mg lead/kg
2009	Face paints	China	Contained 175 (yellow paint) and 270 (mustard paint) mg lead/kg
2009	Children's make up set	China	Green crayon contained 1747 mg lead/kg
2009	Face paint set	China	Black paint contained 25.2 mg lead/kg
2008	Face painting kit	China	Yellow face paint contained 3450 mg lead/kg
2007	Party Pack Face Colour	China	Several paints and pencils contained high concentrations of metals, especially the green paint, containing 31,795 mg lead/kg

1.2 Regulatory Situation in New Zealand

In New Zealand, regulation of cosmetic products is covered by the Cosmetic Products Group Standard 2006 under the Hazardous Substances New Organisms Act 1996.[1] Children's toy cosmetics and face paints are included in the scope of this group standard (Environmental Protection Authority 2014). Under the group standard, cosmetics in New Zealand must not contain lead and its compounds. The Standard does allow for the presence of 'trace' amounts of contaminants such as lead, as long as "such presence is technically unavoidable in good manufacturing practice, and the cosmetic product complies with clause 24 of Schedule 1". Clause 24 states that a substance "must not cause damage to human health when applied under normal or reasonably foreseeable conditions of use, taking account, in particular of the substance's presentation, its labelling, any instructions for its use and disposal as well as any other indication or information provided by the manufacturer or their authorised agent".

1.3 Regulatory Situation Overseas

In Australia, a maximum acceptable level for migratable lead of 25 mg/kg applies to children's 'finger paints' (Product Safety Australia 2014). This regulatory limit was made mandatory through Consumer Protection Notice No. 1 to the Trade Practices Act 1974 and came into effect on 1 January 2010.[2] The explanatory statement for the Notice states that, 'It is the view of the ACCC (Australian Competition & Consumer Commission) that children's face paints and cosmetics sold as children's toys should be included in the proposed mandatory standard for lead in children's toys, and the requirements should apply to all such products for children (0-14 years)'.

In the European Union (EU) face paints are regulated as cosmetics under Directive 76/768/EEC (Council of the European Communities 1976). This directive is similar in wording to the New Zealand Cosmetic Products Group Standard 2006, in including 'lead and its compounds' in a list of substances that 'must not form part of the composition of cosmetic products'.

1.4 Incident Surveillance in New Zealand

In New Zealand, information on hazardous substance exposure incidents is collated in the Hazardous Substances Surveillance System (HSSS) by the Massey University Centre for Public Health Research (CPHR). For the period 2009 to 2012, six incidents (two male, four female) were reported involving exposure to face paint (Helene Marsters, CPHR, personal communication). The incidents involved children aged from one to six years (mean = 2.3 years). All exposures were reported to be the result of exploratory behaviour or unintentional exposure. Four of the incidents involved ingestion of face paint, while the remaining two involved ocular contact.

[1] http://www.epa.govt.nz/Publications/Cosmetic%20Products%20Group%20Standard.pdf Accessed 22 July 2014

[2] http://www.comlaw.gov.au/Details/F2009L00223 Accessed 21 November 2014

No hospital discharges due to face paint exposure were reported during the same period.

2 HAZARD IDENTIFICATION

While other ingredients may be present in children's face paints, the current assessment only concerns effects due to the presence of lead in these products.

2.1 Health Effects – Lead

Lead is generally considered to be of very low acute toxicity, with lowest LD_{50} values for lead salts being greater than 2000 mg/kg body weight (EFSA 2010; JECFA 2011).

Lead has been implicated in a wide range of adverse chronic human health effects, including effects on the nervous system, cardiovascular effects, renal effects, immune system effects, haematologic effects, reproductive and developmental effects and cancer (US Environmental Protection Agency 2012). The US Environmental Protection Agency (USEPA) considered the strength of evidence for a causal relationship between lead exposure and various adverse health endpoints and concluded there was sufficient evidence for a causal relationship in relation to:

- Cognitive function decrements in children
- Attention-related behavioural problems in children
- Hypertension
- Coronary heart disease
- Decreased red blood cell survival and function
- Altered haem synthesis
- Development
- Male reproductive function

It should be noted that the material in the following sections relates to general exposure to lead, rather than specifically to exposure to lead through the use of lead-containing face paint.

2.1.1 Cognitive function decrement in children

Lead can impair cognitive function in children and adults, but children are more vulnerable than adults (ATSDR 2007). The greater impact of lead on the cognitive function of children than adults is partly due to their greater absorption of lead, but also due to the particular susceptibility of the developing nervous system to lead toxicity.

Meta-analysis of seven international population-based longitudinal cohort studies demonstrated a negative relationship between child blood lead concentration (PbB) and full scale intelligence quotient (IQ) score (Lanphear et al 2005). The associated dose-response relationship (see section 3 for more details) shows no lower threshold, that is, there is no PbB level where no decrement in IQ is observed. This health endpoint is considered to be the most sensitive for children and has been used as the basis for subsequent international risk assessments for the effects of lead on children (EFSA 2010; JECFA 2011).

While the exact mode of action leading to cognitive function decrement is unknown, lead induced impairment of neurogenesis, synaptogenesis and synaptic pruning, long term potentiation, and neurotransmitter function has been observed in animal studies (ATSDR 2007).

2.1.2 Attention-related behavioural problems in children

Several prospective studies have demonstrated associations of early childhood and lifetime average PbB levels or tooth lead levels with inattention, impulsivity, and hyperactivity in children 7-17 years and young adults ages 19-24 years (US Environmental Protection Agency 2012). Behaviour was assessed using both objective neuropsychological tests and parent and teacher ratings of behaviour (eg, Connors' scale). Similar findings have been reported in animals, for increases in impulsivity or impaired response inhibition with relevant prenatal and early postnatal lead exposures.

More recent studies have used a formal diagnosis of attention deficit/hyperactivity disorder (ADHD) as the marker of effect in studies on this topic (Ha et al 2009; Nigg et al 2008; Nigg et al 2010; Wang et al 2008). ADHD involves three aspects of behaviour; inattention, hyperactivity and impulsivity (EFSA 2010). There is some evidence to suggest that lead exposure predominantly affects impulsivity and that dysfunction in this domain could contribute to cognitive impairment (Cory-Slechta 2003).

It is plausible that the physiological mechanisms noted in relation to cognitive function decrements could also contribute to attention-related behavioural problems in children.

2.1.3 Hypertension

Evidence from epidemiologic and toxicological studies demonstrates consistent effects of lead exposure on hypertension (US Environmental Protection Agency 2012). Longitudinal prospective studies consistently support the association of biomarkers of lead exposure with hypertension incidence and increased blood pressure, while a meta-analysis across three prospective studies and five cross-sectional studies reached similar conclusions (Navas-Acien et al 2008).

This health endpoint is considered to be the most sensitive for adults and has been used as the basis for subsequent international risk assessments for the effects of lead on adults (EFSA 2010; JECFA 2011).

A number of potential mechanisms have been suggested for the impact of lead exposure on hypertension, including impairment of renal function, oxidative stress, effects on the renin-angiotensin system, suppression of nitric oxide and induction of increased levels of homocysteine (EFSA 2010; JECFA 2011).

It should be noted that increases in blood pressure associated with increases in lead exposure are consistent, but relatively modest.

2.1.4 Coronary heart disease

Biomarkers of lead exposure (PbB or bone lead content) have shown consistent associations with an increased risk of mortality due to coronary heart disease (CHD) (Schober et al 2006; US Environmental Protection Agency 2012; Weisskopf et al 2009). However, studies examining associations between biomarkers of lead exposure and clinical cardiovascular endpoints have shown mixed results (EFSA 2010; JECFA 2011).

Uncertainty remains regarding the level, timing, frequency, and duration of lead exposure contributing to CHD in adult populations with higher past exposure (US Environmental Protection Agency 2012).

Elevated blood pressure is a risk factor for cardiovascular disease (JECFA 2011) and may contribute to the association between lead exposure and CHD. Oxidative stress, caused by lead, has also been suggested as a possible mode of action for CHD associated with lead exposure (US Environmental Protection Agency 2012).

2.1.5 Decreased red blood cell survival and function

Epidemiological studies in human adult and child cohorts have demonstrated alterations in several haematological parameters, increased measures of oxidative stress and altered haematopoiesis (US Environmental Protection Agency 2012). While many of these studies are judged to lack rigorous methodology and consideration for potential confounding they are supported by toxicological findings.

2.1.6 Altered haem synthesis

A number of studies in animals and humans have demonstrated induction of anaemia due to lead exposure, by inhibition of haem synthesis and reducing erythrocyte survival (ATSDR 2007; US Environmental Protection Agency 2012). Lead interferes with haem synthesis by inhibiting the activity of the enzymes δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase (ATSDR 2007). Population studies suggest no threshold to the inhibition of ALAD by PbB, although there does appear to be a threshold for decreases in haemoglobin due to PbB.

2.1.7 Development

A number of epidemiological studies have consistently shown associations between PbB and delayed onset of puberty in males and females (ATSDR 2007; US Environmental Protection Agency 2012). The evidence of delayed pubertal onset among males and females from epidemiological studies is consistent with evidence from toxicological studies at relevant exposure levels.

2.1.8 Male reproductive function

Toxicological studies in rodents, non-human primates, and rabbits have shown detrimental effects on semen quality, sperm, and fecundity/fertility, while epidemiologic studies have reported detrimental effects on sperm (ATSDR 2007; JECFA 2011; US Environmental Protection Agency 2012).

Toxicological studies suggest that oxidative stress is a major contributor to the effects of lead on the male reproductive system.

2.2 Absorption

Due to the dermal application of face paint, a key issue in assessing risks is the degree of dermal absorption of lead. Dermal absorption of lead compounds is generally considered to be much less than absorption by inhalation or oral routes of exposure (EFSA 2010; JECFA 2011). Dermal absorption has been estimated to be 0.06 % during normal use of lead-containing preparations (Moore et al 1980), although few studies have provided quantitative estimates of dermal absorption of lead in humans.

A study was carried out to determine rates of absorption of lead in a Franz cell, using skin harvested from nude mice (Pan et al 2010). Following application of 12 mg lead as lead acetate or lead nitrate, the absorption rate was approximately 0.02 μg lead/cm^2/hour, measured over a 10-hour observation period (0.001%). In *in* vivo studies in nude mice, absorbed lead was detected in liver and kidney, following a 120-hour occluded dermal application of approximately 14 mg lead, as either lead acetate or lead nitrate. Uptake of lead into the skin at the site of application was greater when lead acetate was applied compared to lead nitrate. However, liver and kidney lead concentrations at the conclusion of the study were not significantly different for the two lead compounds.

The chemical nature of the compound has a significant impact on dermal absorption (Hostýnek 2003). An organolead compound (tetrabutyl lead) was absorbed through a 2 cm^2 piece of human skin at a rate of 20 μg/cm^2/hour. Lead salts of organic counter-ions (oleate, naphthenate, acetate) had absorption rates in the range 0.2-4.2 μg/cm^2/hour, while the absorption rate of lead oxide was less than 0.03 μg/cm^2/hour.

It is uncertain what the chemical form of lead is in face paints. If lead is present as a pigment it is likely to be in the form of a chromate or carbonate salt. The available evidence suggests that lead salts including an inorganic anion (chromate and carbonate are inorganic anions) have absorption rate at the lower end of the range discussed above, although no data are available on absorption of lead chromate or carbonate salts.

2.3 Case Reports

No case reports of adverse health effects due to lead in children's face paint were found.

2.4 Epidemiological Studies

No epidemiological studies of adverse health effects due to lead in children's face paint were found.

3 DOSE-RESPONSE INFORMATION

Risk assessments carried out by EFSA and JECFA concluded that decrements in full scale IQ was the most sensitive adverse health effect associated with lead exposure for children, while increases in systolic blood pressure was the most appropriate endpoint for adults (EFSA 2010; JECFA 2011). In addition, the EFSA assessment considered the impact of lead exposure on chronic kidney disease, based on reductions in the glomerular filtration rate (GFR) (EFSA 2010).

3.1 Oral Health-based Exposure Limits

The available dose-response information for these endpoints relate PbB or bone lead measures to effect levels. For example, a blood lead concentration of 10 μg/dL (100 μg/L) equates to an approximate IQ decrement of four points. In both the EFSA and JECFA risk assessments existing models were used to equate PbB to dietary lead exposure. In the case of the JECFA risk assessment this involved equating a range of effect levels to corresponding dietary exposure levels (JECFA 2011). The EFSA assessment utilised a margin of exposure (MoE) approach and defined a benchmark dose (BMD) giving a 1% change in effect compared to baseline (EFSA 2010). Dietary exposures were derived that would equate to the lower 95th percentile confidence limit of the BMD ($BMDL_{01}$). The dietary (oral) doses derived by JECFA and EFSA are summarised in Table 3.

The JECFA assessment used six different statistical models to describe the observed relationship between PbB and decrements in FSIQ (JECFA 2011). The Hill and bilinear models provided the best fit to the observed data. A combined output from these two models and the bilinear model only were used in the JECFA assessment.

Table 3: Dietary (oral) doses and blood lead levels (PbB) equating to various effect levels for lead

Population group	Endpoint	Effect level	PbB level (μg/L)	Estimated oral exposure dose (μg/kg body weight/day)
European Food Safety Authority (EFSA 2010)				
Children	Decrements in FSIQ	Decrease of cognitive ability by 1 IQ point	$BMDL_{01}$ = 12	0.5
Adults	Increase in SBP	Increase in SBP of 1.2 mmHg	$BMDL_{01}$ = 36	1.5
Adults	CKD, defined as GFR less than 60 mL/1.73 m^2 body surface/minute	A 10% increase in prevalence of CKD	$BMDL_{10}$ = 15	0.63
Joint FAO/WHO Expert Committee on Food Additives (JECFA 2011)				

Population group	Endpoint	Effect level	PbB level (μg/L)	Estimated oral exposure dose (μg/kg body weight/day)
Children	Decrements in FSIQ	IQ decrease of: 0.5 1.0 1.5 2.0 2.5 3.0 0.5 1.0 1.5 2.0 2.5 3.0		Combined Hill and bilinear models 0.8 (0.1-9.7)[1] 1.5 (0.2-10.4) 2.0 (0.3-11.2) 2.4 (0.4-12.0) 2.8 (0.4-13.1) 3.1 (0.5-14.8) Bilinear model only 0.3 (0.1-6.2) 0.6 (0.2-7.2) 0.9 (0.3-8.5) 1.3 (0.4-9.7) 1.6 (0.5-10.9) 1.9 (0.6-11.8)
Adults	Increase in SBP	Increase in SBP of 1 mmHg	Median increase of 0.28 mmHG per 10 μg/L	1.3 (0.6-28)[1,2]

PbB = blood lead concentration; FSIQ = Full Scale Intelligence Quotient; SBP = systolic blood pressure; $BMDL_{01/10}$ = lower 95th percentile confidence limit for a benchmark dose producing a 1 or 10% change in effect compared to baseline; mmHg = millimetres of mercury; CKD = chronic kidney disease; GFR = glomerular filtration rate

[1] Figures in brackets are the 95th percentile confidence interval

[2] The relationship between increases in SBP and oral exposure to lead was considered to be linear and other magnitude increases in SBP would be expected from other magnitude increases in oral exposure. For example, oral exposure of 2.6 g/kg body weight/day would be expected to result in an average increase in SBP of 2 mmHg

3.2 The Relationship Between Blood Lead and Lead Exposure

Gastrointestinal absorption of ingested lead is influenced by physiological factors, such as age, fasting status, nutritional calcium and iron status, and pregnancy, and physicochemical characteristics of particles (size, solubility and lead species). Details of the mechanism of absorption remain to be determined (EFSA 2010). The US Environmental Protection Agency have developed an Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children (under the age of seven) (US Enviromental Protection Agency 2002). The model can be used to predict the risk of elevated PbB levels in children that are exposed to environmental lead from many sources. The model assumes that 50% of the lead intake from drinking water and food is absorbed and that 30% of the lead intake from soil and dust is absorbed. The IEUBK has since been expanded to include lead exposures across a wider age

range and has been incorporated into the All Ages Lead Model (AALM).[1] It should be noted that the AALM only considers exposure to lead by inhalation and ingestion, not by dermal absorption.

Carlisle and Wade (1992) developed an exposure model for lead, considering multiple possible routes of exposure, including dermal exposure. For soil adherence to the skin, a dermal absorption of 0.06% (Moore et al 1980) and an oral absorption of 11% (ATSDR 1990) were used to derive a 'dermal constant' that allows dermal exposure to lead to be converted to equivalent blood lead concentrations. The dermal constant derived was 0.0001 (μg lead/dL blood)/(μg dermal lead/day) (0.001 (μg lead/L blood)/(μg dermal lead/day)). This model has been used in a recent risk assessment for lead, to relate lead exposure to changes in PbB (EFSA 2010). It also appears to be the only available model relating dermal exposure to lead to PbB.

[1] http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=139314 Accessed 17 March 2015

4 EXPOSURE ASSESSMENT

While adults will come into contact with children's face paints during application and clean-up of the paints, it was considered that, at worst, this would result in short duration dermal exposure and would be unlikely to contribute markedly to adult lead body burden.

Three potential exposure scenarios for lead in face paints were considered for children:

- Dermal exposure, following application of face paints
- Oral exposure, following application of face paints (face to hand to mouth exposure). It should be noted that this is not an independent exposure route and will only occur in conjunction with dermal exposure, following application of face paint
- Oral exposure, following direct ingestion of face paints

The third of these scenarios refers to instances where children have unsupervised access to face paints and may ingest the paint directly. However, it seems reasonable to assume that such an occurrence would not be repeated or would only be repeated at extremely low frequency.

4.1 Dermal Exposure

The approach of Carlisle and Wade was the only study found that specifically considered dermal exposure to lead (Carlisle and Wade 1992). In this approach absorption of lead from soil adhering to the body surface was considered. The resulting contribution to PbB was defined as:

PbB = soil Pb x contact rate x dermal constant (1)

Where:

Soil Pb	=	concentration of lead in soil (μg/g)
Contact rate	=	grams of soil in contact with the skin per day (g/day). Product of the soil adherence factor (g/m^2) and the exposed skin area (m^2)
Dermal constant	=	0.0001 (μg lead/dL blood)/(μg dermal lead/day) (see section 3.2.1 for more details)

The first two terms of this equation are consistent with equations previously used in ESR Hazardous Substance assessments for the determination of dermal exposure, with 'soil Pb' equivalent to C_{derm} and 'contact rate' equivalent to the product of the remaining four terms:

$$A_{derm} = C_{derm} \times T_{derm} \times Area_{derm} \text{ x } BIO_{derm} \text{ x } N_{events} \quad (2)$$

Where:

External exposure to skin (mg/day)	A_{derm}
Concentration in the product (mg/cm^3)	C_{derm}

Thickness of the film layer on skin (default = 0.01cm)	T_{derm}
Surface area of skin exposed (cm^2)	$AREA_{derm}$
Bioavailability for dermal exposure (default = 1)	BIO_{derm}
Number of events per period (usually, events/day)	N_{events}

The contact rate in equation (1) will be equivalent to T_{derm} x $AREA_{derm}$, while equation (1) implicitly assumes one exposure event per day.

4.2 Oral Exposure

In the approach of Carlisle and Wade (1992) three components of oral (ingestion) exposure are included; dietary, drinking water and soil and dust. The general form of the exposure equations is the same as equation (1) above, including a term for the concentration of lead in the ingested media, a term to define the quantity of media ingestion per day and a term to account for media specific absorption. For children, the absorption-related constant is 0.16 (μg lead/dL blood)/(μg ingested lead/day) for food and water and 0.07 (μg lead/dL blood)/(μg ingested lead/day) for soil and dust.

Given that the presence of lead in children's face paints is believed to be due to the presence of mineral pigments, the absorption-related constant for soil and dust was considered to be most applicable to a consideration of children's face paints.

4.3 Parameters for Exposure Scenarios for Lead in Children's Face Paints

4.3.1 Concentration of lead in children's face paints

Occasional very high concentration of lead have been reported in children's face paints, although the limited information available suggests that the majority of paints contain more modest levels of lead (<10 mg/kg). In the current study two concentrations were considered; a highest reported concentration of 31,795 mg/kg (see Table 2) and the Australian regulatory limit of 25 mg/kg. It should be noted that there is currently insufficient monitoring data to say how frequently very high concentrations of lead are present in children's face paints in New Zealand.

4.3.2 Frequency of use of face paints by children

No information was found on the frequency of face paint use by children. Anecdotal information obtained through informal discussions with mothers with young children suggest that face painting is usually considered to be a 'treat' and is unlikely to occur at high frequency. These discussions suggested a frequency of no more than 5-6 times per year. For the current study a likely frequency of once every two months and a 'worst case' frequency of once per week were examined.

4.3.3 Contact rate

In equation (1) the contact rate is the weight of material in contact with the skin. Using equation (2), the contact rate can be estimated from the area of skin affected

and the thickness of the face paint layer applied. For the current study, all scenarios assume that the entire area of the face will be affected during face painting.

Body surface area increases with increasing age (US Environmental Protection Agency 2008). However, the surface area of the head (and the face) decreases as a proportion of total body surface area with age, from a mean of 18.2% during the first year of life to less than 10% by age 20 years. Despite representing a decreasing proportion of body surface area with increasing age, the surface area of the head (and face) increases with increasing age.

The equations of Carlisle and Wade (1992), relating exposure to lead to increases in PbB, do not include a factor for body weight. The equations for oral exposure include separate constants for 'children' and 'adults', but these age classifications are not further defined. The equation for dermal exposure does not have separate constants for children and adults, consequently, the largest impact on PbB would be expected for individuals with the greatest body surface area.

For the current study, two situations were considered:

1. Solely dermal exposure for an older child (11-16 years). The US Environmental Protection Agency Exposure Factors Handbook gives a median body surface area for this age group of 1.57 m^2 (95^{th} percentile 2.06 m^2) (US Environmental Protection Agency 2008). The face was considered to be half of the area of head or approximately 5% of the body surface area (median 785 cm^2, 95^{th} percentile 1030 cm^2).
2. A combination of dermal and oral exposure for a young child (2-3 years) with median body surface area 0.61 m^2 (6100 cm^2, 95^{th} percentile 0.70 m^2 or 7000 cm^2), with the face being approximately 7% of the body surface area (median 427 cm^2, 95^{th} percentile 490 cm^2).

The other contributor to contact rate is the thickness of the film adhering to the skin surface. A default film thickness of 0.01 cm may be used (see equation 2). Various liquids (eg. cooking oil, bath oil, water) have been reported to have a maximum film thickness of approximately 0.01 cm (following immersion in mineral oil) (US Environmental Protection Agency 2011). Assuming full face coverage, this would result in application of 4-10 cm^3 of face paint under the scenarios discussed above. This appears plausible, as the US Environmental Protection Agency Exposure Factors Handbook gives a figure of 3.7 g per application for paste masks (mud packs) (US Environmental Protection Agency 2011). If it is assumed that the density of face paint would be near unity, then the application amount for a paste mask is similar to our proposed application amounts for face paint.

4.3.4 Distribution between dermal and oral exposure

It is considered likely that young children will ingest a proportion of the face paint applied to them, through touching incompletely dried paint and placing their fingers in their mouths. The additional impact of 5% of applied face paint being ingested was considered for young children.

4.3.5 Averaging time

The equations of Carlisle and Wade (1992) relate to daily exposure to lead, while the exposure scenarios considered in the current document have lower frequencies of exposure. However, most absorbed lead will eventually reside in a 'deep tissue compartment' (skeletal bones) with a very long elimination half-life (10^4 days) (ATSDR 2007). Consequently, the daily exposure to lead was considered to be the direct proportion of the weekly (1/7) or two-monthly (6/365) exposures.

4.4 Exposure Assessment

Table 4 summarises estimates of exposure to lead from use of face paints for 11-16 year old children, based on the various parameter options and assumptions outlined in the previous section. Table 5 summarises exposure estimates for 2-3 year old children, including consideration of ingestion of a proportion (5%) of the applied dose.

Table 4: Lead exposure (μg lead/L blood) for 11-16 year old children due to use of face paints

	Average		**95th percentile**	
SA(face) (cm^2)	795		1030	
Thickness of applied paint film (cm)	0.01		0.01	
Dermal constant ((μg lead/L blood)/(μg lead exposure/day)	0.001		0.001	
	C = 25 μg/cm^3	C = 31,795 μg/cm^3	C = 25 μg/cm^3	C = 31,795 μg/cm^3
Frequency of use = 1/week	0.028	36.1	0.037	46.8
Frequency of use = 1/2 months	0.003	4.2	0.004	5.4

SA(Face) = surface area of the face, C = concentration

Table 5: Lead exposure (μg lead/L blood) for 2-3 year old children due to use of face paints

	Average		95th percentile	
SA(face) (cm^2)	427		490	
Thickness of applied paint film (cm)	0.01		0.01	
Dermal constant ((μg lead/L blood)/(μg lead exposure/day)	0.001		0.001	
Oral constant ((μg lead/L blood)/(μg lead exposure/day)	0.7		0.7	
	C = 25 $\mu g/cm^3$	C = 31,795 $\mu g/cm^3$	C = 25 $\mu g/cm^3$	C = 31,795 $\mu g/cm^3$
Dermal exposure only				
Frequency of use = 1/week	0.015	19.4	0.018	22.3
Frequency of use = 1/2 months	0.002	2.2	0.002	2.6
Dermal exposure (95%) + oral exposure (5%)				
Frequency of use = 1/week	0.55	700	0.63	800
Frequency of use = 1/2 months	0.063	80	0.072	92

SA(Face) = surface area of the face, C = concentration

Lead is generally considered to be of very low acute toxicity, with LD_{50} values for lead salts typically greater than 2000 mg/kg bw. The lowest observed lethal doses in animals after multiple short-term oral exposure to lead salts range from 300 to 4000 mg/kg bw (EFSA 2010). If a 2-3 year old child (mean body weight 15.0 kg) were to accidentally consume a 100 mL container of face paint, containing lead at a concentration of 31,795 $\mu g/cm^3$ (μg/mL) the resultant exposure dose would be 212 mg/kg bw and would probably not be lethal. A review of Internet retail outlets for children's face paints suggests that a pack size of 100 mL is at the upper end of available pack sizes.

It should be noted that there is no evidence on which to derive a lethal acute dose for lead in humans. Blood levels as high as 4300 μg/L have been reported in non-fatal chronic poisoning cases and responded well to chelation treatment (Mikler et al 2009).

Lead absorption to a PbB of greater than 100 μg/L is a notifiable disease in New Zealand.[1] The equations of Carlisle and Wade relate to long-term exposure to lead. If it is assumed that a single ingestion event can be averaged to a chronic exposure over one year, ingestion of 100 mL of face paint containing 520 μg/mL (52,000 μg or 142 μg/day) or more of lead would be sufficient to elevate PbB above the notification level. Ingestion of as little as 1.6 mL of face paint containing 31,795 μg/mL of lead would also elevate PbB above the notification level, based on the same assumptions of averaging time.

[1] http://www.health.govt.nz/our-work/diseases-and-conditions/notifiable-diseases Accessed 29 April 2015

5 RISK CHARACTERISATION

It should be noted that there have been no case reports of adverse health effects due to the use of face paints containing lead. However, given that lead is of low acute toxicity and the chronic effects of lead exposure have multiple risk factors, it is unlikely that adverse health effects would be directly linked to use of face paints.

The risk to human health from the presence of lead in children's face paints was assessed by applying the Margin of Exposure (MOE) approach, as there was no evidence for a threshold for the critical endpoints, systolic blood pressure, chronic kidney disease and IQ scores. MOEs are calculated by dividing a dividing a defined point on the dose-response curve, such as the benchmark dose (BMD), by the estimates of exposure. For the current study, both the BMD and exposure estimates are expressed in terms of changes in PbB. Lower 95th percentile confidence limits for the benchmark dose (BMDLs) derived by the European Food Safety Authority were used for this purpose (EFSA 2010). For children, the relevant and most sensitive endpoint relates to decrements in cognitive ability, with a BMDL for a 1 IQ point decrement of 12 μg lead/L in blood. Estimates of exposure are expressed as increments in PbB due to exposure to lead from children's face paints.

The interpretation of the MOE is dependent on the magnitude and nature of the benchmark response (BMR), the dose metric on which the BMD is based and the relevance of the population in whom the BMDL was determined. In their assessment of neurotoxicity in children due to lead exposure EFSA concluded that "a margin of exposure of 10 or greater should be sufficient to ensure that there was no appreciable risk of a clinically significant effect on IQ. At lower MOEs, but greater than 1.0, the risk is likely to be low, but not such that it could be dismissed as of no potential concern" (EFSA 2010).

For children aged 11-16 years, MOEs range from 0.26 to 4000. The MOEs less than one are associated with use of face paint containing very high concentrations of lead (31,795 mg/L or μg/cm^3). For face paints containing lead at the Australian regulatory limit of 25 mg/L, MOEs are in the range 320-4000.

For children aged 2-3 years, MOEs range from 0.015 to 6000. The lowest MOEs are associated with the use of face paint containing very high concentrations of lead and with ingestion of a proportion of the applied face paint. For face paints containing lead at the Australian regulatory limit of 25 mg/L, MOEs are in the range 19-6000, irrespective of whether face paint is ingested or not. The wide range of MOEs, reflecting a wide range of lead concentrations, suggests there is no technological reason for children's face paints to contain high concentrations of lead and it is desirable, from a public health perspective, that such high-lead product be identified and removed from the market.

It should be noted that the dose-response relationship used to derive the BMDL used in this risk characterisation uses PbBs that will result from exposure to lead from a range of sources, while the estimated PbBs from use of children's face paints only consider a single source of exposure. Although dietary exposure to lead in New Zealand has decreased markedly during the last 30 years (Vannoort and Thomson 2011), diet will make an additional contribution to PbB, as will ingestion of soil and

dust from the environment. Some drinking water supplies may also contain lead, usually derived from metallic fittings in the reticulation system.[1]

Children's face paints are usually sold in relatively small containers (100 mL or less) and it is unlikely that children would be exposed to consistently high levels of lead from this source over an extended period of time.

The exposure model used is similarly conservative in assuming that the entire face would be painted during face painting. Although this may be true in some cases, face painting often involves coverage of much less than the entire surface area of the face.

[1] http://www.health.govt.nz/our-work/environmental-health/drinking-water/plumbosolvency Accessed 5 March 2015

6 CONCLUSIONS

It is currently unknown what concentrations of lead are typically present in children's face paints in New Zealand. Surveys in New Zealand and Australia have shown that some face paints can have high levels of lead, but insufficient monitoring has been carried out to determine the frequency of such high concentrations in children's face paints. No case reports of adverse health effects due to lead in children's face paints have been reported in New Zealand or internationally. However, given the nature of the chronic adverse health effects that lead is associated with, this is not surprising.

There is good evidence for a causal relationship between lead exposure and a range of adverse health effects. The most sensitive adverse health effect for children relates to decrements in cognitive ability, as measured by full-scale IQ score. A BMDL (lower 95th percentile confidence interval of the benchmark dose) of 12 μg lead/L blood has been derived for a 1 IQ point average decrement in cognitive ability.

Analysis of results from face paint exposure modelling, using a MOE approach and the benchmark dose for decrements in child IQ, suggests that use of face paints containing up to the Australian regulatory limit for lead of 25 mg/L is unlikely to result in major adverse health effects. MOEs for use of face paints containing 25 mg/L of lead were in the range 19-6000. However, it should be noted that, while exposure estimates predict blood lead levels well below the benchmark dose, no threshold for the impact of lead on child cognitive ability has been established.

Application of face paints containing the highest reported levels of lead (31,795 mg/L) resulted in MOEs less than one for most exposure scenarios. Use of face paints containing this level of lead over an extended period of time has the potential to result in adverse health effects.

Direct ingestion of children's face paints during exploratory behaviour is unlikely to be fatal, even at the highest observed lead concentrations (31,795 mg/L) and assuming ingestion of a complete large pack of face paint (100 mL). However, if a single ingestion event is averaged to one year of daily exposure, ingestion of 100 mL of face paint containing 520 mg/L or ingestion of 1.6 mL of face paint containing 31,795 mg/L of lead would potentially raise PbB above the New Zealand notification level (100 μg/L).

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