School of Design and the Built Environment

The Morbid Health Implications of Living in the Interior Built Environment

Samantha Jade Webb

0000-0002-3110-2689

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DECLARATION

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgment has been made.

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2 December 2021

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Samantha Webb Date

ABSTRACT

Earlier communities and civilisations inhabited structures built from raw materials. Today, there is an alarming quantity of chemicals being incorporated into commonly used building materials (Pacheco-Torgal & Jalali, 2011, pp. 281–287). These materials significantly increase the toxicity of indoor air – that is the indoor places where we humans spend 90% of our lives (Kumar et al., 2016). In addition, the toxicity inherent across most stages of the processing of toxic building materials negatively impacts humans and the broader living environment throughout the stages of production, construction, occupation, demolition and waste disposal. These commonly incorporated toxic substances include volatile organic compounds, plasticisers, flame retardants and pesticides. In addition, the rising occurrence of mould also has serious health implications. Therefore, this research utilised the case study methodology, drawing significantly on a literature review, to investigate the occurrence and influence of 6 common toxic substances widely occurring within building materials in the building industry in Australia and beyond. The aim of this research was to make explicit the prevalence and effect of these toxic substances so as to positively raise awareness, intending to influence a reduction of poor design and construction practices including specification of hazardous materials that negatively influence human health and the environment.

KEYWORDS

Interior built environment

Interior architecture

Interior design

Construction

Indoor air quality

Toxins

Toxicants

Health

Mould

Phthalates

Formaldehyde

Polyvinyl chloride

Polychlorinated biphenyls

Brominated diphenyl ethers

Textile pesticides

Organochlorines

Organophosphates

Pyrethroid

Sustainability

Life cycle assessment

Vinyl

Timber

Paints

Composite wood products

Polyurethane foam

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My immense gratitude also goes out to my friends and colleagues, who also endlessly supported me through difficult times and encouraged me to complete my studies. And importantly, I wish to express my infinite appreciation to my family who have always encouraged me to be the best person I can be; to stand up for what I believe in; and, who always have unconditional support, encouragement and love. You are my inspiration.

And, to you. An openness to question the ‘norm’ can change mindsets. A single small step towards change can lead to big steps by the many. As Albert Einstein said,

> “The important thing is not to stop questioning. Curiosity has its own reason for existence. One cannot help but be in awe when he contemplates the mysteries of eternity, of life, of the marvellous structure of reality. It is enough if one tries merely to comprehend a little of this mystery each day” (1955).

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ABBREVIATIONS

ACLA	anticardiolipins autoantibodies
AgNPs	silver nanoparticles
ACGIH	American Conference of Governmental Industrial Hygienists
ARfD	acute reference dose
BBP	butyl benzyl phthalate
BDE	brominated diphenyl ethers
BFR	brominated flame retardant
BTS	bimetallic treatment system
CIRS	chronic inflammatory response syndrome
DDT	dichlorodiphenyltrichloroethane
decaBDE	decabromodiphenyl ether
DEHA	di (2-ethylhexyl) adipate
DEHP	di-(2-ethylhexyl) phthalate
DEP	diethyl ester of phthalic acid
DINCH	di(isononyl) cyclohexane-1,2-dicarboxylate
ECG	electrocardiogram
ECH	epichlorohydrin
ECP	eosinophil cationic protein
EPA	Environmental Protection Agency
EU	European Union
EWG	Environmental Working Group
GMO	genetically modified organism
GOTS	Global Organic Textile Standard
GP	general practitioner
HBV	hepatitis B viral
HCB	hexachlorobenzenes

HEPA	high efficiency particulate air
HMW	high molecular weight
HVAC	heating, ventilation, and air conditioning
IAQ	indoor air quality
IARC	International Agency for Research on Cancer
IgE	immunoglobulin E
IRIS	Integrated Risk Information System
LCA	life cycle assessment
LMW	low molecular weight
MCS	multiple chemical sensitivity
MDF	medium density fibreboard
MMP9	matrix metallopeptidase
MSH	melanocyte stimulating hormone
NaOH	sodium hydroxide
NCC	National Construction Code
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NMTS	non-metal treatment system
NRC	National Research Council
NTP	National Toxicology Program
octaBDE	octabromodiphenyl ether
OPP	organophosphate pesticide
PAE	polyamidoamine-epichlorohydrin
PATY	Pollution and the Young project
PBDF	polybrominated dibenzofuran
PBDE	polybrominated diphenyl ethers
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-p-dioxins

PCDF	polychlorinated dibenzofuran
pentaBDE	pentabromodiphenyl ether
PPBD	polybrominated dibenzo-p-dioxins
POP	persistent organic pollutant
PVC	polyvinyl chloride
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RfCs	reference concentrations
rT3	reverse T3
RH	relative humidity
SBS	sick building syndrome
SVOC	semi-volatile organic compound
T3	triiodothyronine
T4	thyroxine
TGF Beta-1	Transforming Growth Factor Beta-1
TiO2	titanium dioxide
TMSA	Toxic Mould Support Australia
UF	urea-formaldehyde
UK	United Kingdom
US	United States
US EPA	United States Environmental Protection Agency
USSR	Union of Soviet Socialist Republics
VCM	vinyl chloride monomer
VEGF	vascular endothelial growth factor
VIP	vasoactive intestinal polypeptide
VOC	volatile organic compounds
WHO	World Health Organization

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DISCLAIMER

This ‘Introduction’ chapter has been written in the subjective perspective of the researcher. Opinions of practitioners and experts included throughout the introduction remain the personal and professional option of those involved, and have been paraphrased and kept anonymous for confidentiality purposes – unless otherwise noted. All images included, which are not noted as from the author’s own collection, have been provided by the experts involved during the experience outlined in the Introduction with their written consent. Direct reference to those experts also remain confidential.

1. INTRODUCTION

Being raised in Australia by a hard-working family, it was always my ambition to enter the property market, purchase my own home and one day look to utilise it as an investment. In 2014, I endeavoured to turn that goal into a reality.

I came across an alluring opportunity to buy an apartment off the plan that appeared to suit me and my budget. It was a north-east facing apartment, maximising morning sunlight without the harsh Australian summer sun of the afternoons. It occupied the corner of the building on an elevated ground floor, allowing some privacy and security from the street, a larger balcony than some of the upper floor plans and crossflow ventilation with windows across two sides of the apartment rather than a singular façade like many other multi-residential complexes nowadays. The communal facilities looked promising, as did the proximity to nearby community services, suburbs and my workplace.

Just over a year had passed between exchanging contracts and settlement, which worked well in terms of allowing me to increase my savings prior to the exchange. Since it was my first home that was not a rental, I was excited about starting this new chapter.

THE START OF A NEW CHAPTER – NOVEMBER 2015

When the day arrived to finally move in, I left early to take a carload of my belongings to the apartment and do a quick clean throughout prior to furniture being moved in. I was surprised to see that the apartment was rather filthy and had an unusual stale stench to it; I felt that the final clean before handover must have just been missed for my apartment. There was a miscoloured, sticky puddle in one of the bathrooms next to the toilet, dirt and grime throughout the carpet and tiling, and a pile of cigarette butts on the balcony. Nonetheless, I pulled out the vacuum and mop and started to clean.

It was a conscious decision during the construction process to substitute the specified carpet selection from a solution-dyed nylon option to a much more natural alternative. At that point in time, I was coincidently already investigating the health implications of interior spaces and associated materials. The carpet I nominated to go into the apartment was a 100% New Zealand wool carpet. It was a sustainable, biodegradable and recyclable product, and naturally anti-static, stain resistant and fire resistant, while also being low in volatile organic compounds (VOCs) to a Green Star rating level (Bremworth Pty Ltd, 2021). Unfortunately, this good intention turned out to be short lived.

The master bedroom smelt the worst within the apartment, and part of the carpet appeared to be stained. The chocolate-coloured yarn had discoloured in a rather large section, directly outside the ensuite. Initially, I vacuumed it, thinking it was just an aesthetic issue that I could enquire about later. But it was wet. Soaked (refer to Image 1.1). In addition, grey and black staining was apparent along the adjacent skirting board and the architraves to the door jamb of the ensuite (refer to Image 1.2*).* I attempted to lodge this issue immediately as a construction defect; unbeknownst to me, it would not formally lodge until the following day owing to a lack of reception at the time.

Thinking this was a minor issue and a reasonably quick fix, the furniture was still set up as intended in the master bedroom. In the evening I went to bed, but I could not sleep. I was wheezing, and I started to cough. Assuming this was associated with the wet carpet, I took my bedding out of the bedroom and attempted to sleep on the sofa.

Image 1.1: Damp carpet outside of main bedroom
Note. From the author's personal collection 2015.

Image 1.2: Water damage to woodwork
Note. From the author's personal collection 2015.

The following morning, initial investigations began, and the wet portions of carpet and underlay were cut out and removed from the apartment, exposing the concrete slab below. Some raw pits in the concrete slab indicated that moisture was occurring there, with a damp and darker appearance (refer to Image 1.3). The mould remained on the woodwork for some time afterwards, with some of the timber appearing swollen and discoloured also. The visible mould that had damaged the woodwork outside the ensuite was sanded back just over a week after moving in, although I was concerned that this could lead to mould spores becoming airborne.

Within about a fortnight of moving in, the carpet had been replaced and the leak was believed to be fixed. And yet, I was not feeling well, and the new carpet was becoming damp again in the same locations (refer to Image 1.4). I raised the carpet edge outside the ensuite off the concrete slab and propped it up in an attempt to dry it out and prevent further damage to the new carpet. However, the new timber carpet edges used to pin the carpet down were also showing evidence of mould – black, green and grey-coloured spots (refer to Image 1.5). The underside of the new carpet, the jute backing, had discoloured where moisture appeared to have affected it already, with stain lines and further grey-like patches (refer to Image 1.6).

Image 1.3: Water damage to woodwork and damp signs to slab

Note. From the author's personal collection 2015.

Image 1.4: Dampness returning through new carpet

Note. From the author's personal collection 2015.

Image 1.5: Further signs of water damage and mould, after the woodwork had been sanded back

Note. From the author's personal collection 2015.

Image 1.6: Damage to the underside of the carpet

Note. From the author's personal collection 2015.

THE DETERIORATION OF MY HOME – DECEMBER 2015 AND JANUARY 2016

Within a month of moving into the apartment, my hands had swollen and were red and itchy, as was my left foot. I was suffering from frequent headaches, some that did not dissipate for days on end. The itchy rashes on my skin had developed into blisters that became incredibly painful. I started to struggle with everyday minor tasks like opening my front door or even putting my hands in my handbag for the keys because it was excruciating to apply pressure against my skin, let alone touch anything. I recall a professional introduction of a client within my office, and we shook hands. To what felt like the strongest handshake you could possibly receive, I momentarily froze in pain and attempted to smile it off. Wearing shoes and trying to walk as normally as possible was challenging. I even started to stall my manual car whenever the clutch was needed as a result of the pain in my left foot, and changing the gear stick with my left hand was not much better. My hands and feet were almost covered in various dressings and even bandages in an attempt to keep the raw wounds clean and reduce my scratching. Heat seemed to exacerbate the problem, whether it was the heat and humidity of summer or simply from wearing shoes and bandages. The heat would then lead to more swelling and, subsequently, more pain in my joints and raw skin. Finding any kind of relief was near impossible – nothing I seemed to try would have any positive result. A number of visits to

Image 1.7: Steel track exposed as rusted when skirting board was removed

Note. From the author's personal collection 2016.

a general practitioner (GP) practice resulted in courses of antibiotics to treat ongoing infections, which did help at times, but only temporarily until the courses were finished, at which point the symptoms would resume and continue to escalate.

The source of the leak at the time was suggested to be from a charged hydraulic pipe for the toilet, allowing the pipe to leak without the toilet being used, with the water tracking around the screed and somehow around the waterproof membrane to make its way into the main bedroom. Part of the mouldy, water-damaged skirting board adjacent to the ensuite jamb was removed (refer to Image 1.7). A draught persisted through the newly opened wall cavity, which could be felt within the main bedroom. With the exposure of the wall cavity interior, I thought I could take some photographs up the cavity and determine whether there were any further indications of the cause of the leak. To my disbelief, mould appeared to be affecting parts of both the timber stud to the jamb as well as the adjacent inner face of the plasterboard lining (refer to Image 1.8). The bottom of these timber studs where exposed and appeared to have rotted somewhat; moreover, I found rusted steel on the galvanised base track of the wall. The rusting, rotting and mould begged the question of how long the issue had actually been occurring prior to me moving in.

Image 1.8: Visible mould within wall cavity

Note. From the author's personal collection 2016.

Following a further inspection of the issues and almost six weeks after the wall cavity had been left open, the bottom portion of the timber packer and metal stud, the galvanised bottom track and part of the plasterboard were cut out and replaced. A series of small rectangular holes were also cut into the wall that separated the laundry from the ensuite, perpendicularly adjoining the water-affected ensuite to the bedroom wall, to inspect whether there were any signs of the cause or even a path for the water into the bedroom. Nothing further was discovered; the walls were patched up and prepared for painting. The carpet, while still not professionally re-laid, was showing further signs of moisture from the ensuite. The jute backing had hardened along the ensuite wall, with curved lines indicating prior water egress, and white to grey patches were also evident in that vicinity. Despite this, the carpet was re-laid properly within the two weeks that followed, and subsequently the new plasterboard and woodwork was repainted. Following a little relief from the physical health symptoms that coincided with the patching up of the wall cavities, I the experienced a downward turn then continued to progress, with an increase in the diversity of my health as well as the severity.

THE DETERIORATION OF MY HEALTH THROUGH 2016

As the months passed, my health continued to deteriorate. I sensed that there were some significant issues still associated with the 'leak,' along with the stench that I could never seem to remove. This was associated with ongoing health issues that I struggled to have any control over no matter what I tried. Inflammation skyrocketed. Interestingly, I had been asymptomatic regarding asthma and hay fever since prior to moving to the area in 2008, and I hadn't undertaken anything unusual to my knowledge that could bring on such acute response that would later become chronic. The following extracts are technical medical updates including expert input.

> *Two months after the initial water damage exposure, immunoglobulin E (IgE) levels were elevated – grass pollen was < 100, dust mites were 9.31, tree pollen mix was 1.94, and interestingly, mould mix was 0.32. These were lower at levels of 33.1, 7.63. 0.89 and 0.17, respectively, about five months prior to moving into the apartment. Ulcers, with some being infected, were noted with red abraded dry skin to both hands and one foot and requiring in steroid and antibiotic treatment.*

The irritations continued, so mould testing was sought. Owing to financial constraints, only one test was undertaken at this time, and this was a swab of the underside of the carpet – importantly, it was the same carpet that had been replaced back in January. Levels of mould detected were low enough to be classified as normal ecology; however, the mould species uncovered were considered allergenic, can produce mycotoxins and can compromise one's immune system. I had a level of

naivety in thinking water damage the issues were exclusively associated with the previous leak, while still being at a loss to understand my health issues. While I believed there was much more to the water damage than was physically visible within the apartment, I started to question whether the issues were evident elsewhere within the apartment, or perhaps even airborne.

Following the ambiguity associated with that test report, in early June 2016 a microbial treatment was applied to the leak zone in the main bedroom. I understood that that treatment was Biosan, of which Applied Products only identify 10% of the ingredients. Biosan is identified as being "Xn harmful". The warning indicates it can be "irritating to the eyes, respiratory system and skin", that it "may cause sensitisation by inhalation and skin contact" and to "avoid release to the environment" (Indoor Environmental Health Consultant, 2017). The on-site advice at the time was that the bedroom would be safe to use within 15 minutes following the application, but my headaches and wheezing increased almost immediately, and once again I found myself seeking medical advice from a GP. It was at this point that my symptoms considerably worsened, well beyond what they had been previously.

By mid-June, a clinical biologist assessed symptoms including asthma, eczema, psoriatic arthritis, headaches, fatigue, active Epstein Bar virus, pain to the chest and back, and swollen limbs. While not a commonly acknowledged method, electro-dermal testing was utilised, which identified that my liver was swollen, and although managing, it was burdened in function. The gastrointestinal tract and lungs demonstrated high lymph activity. My spleen was competent but swollen. Immune scarring that was related to fungi and moulds including *Aspergillus*, *Cladosporium* and *Microsporum* was suggested, along with less severe immune responses to *Cephalosporium* and fungal yeast. The concluding diagnosis was that I was suffering from an overburden of industrial chemicals, mould and fungi, and I displayed further deterioration over two months of treatment (Clinical Biologist, Immunology, 2016).

In late June 2016, a GP indicated a reappearance of palmoplantar keratosis, in association with an exacerbation of symptoms following chemical exposure (General Practitioner #1, 2016). Without much relief, I tried another GP a month later. This GP appreciated the chronic and diverse symptoms that included pain, stiffness and swelling to my hands bilaterally, pain to the lower and upper spine, and swelling and pain in the left foot with lesions. I recall some toes were so swollen they physically could not bend (General Practitioner #2, 2016).

A series of subsequent blood results detected nuclear antibodies of a nucleolar pattern at Titre 320, advising that "nucleolar staining patterns are more common with scleroderma or overlap connective tissue disorders" (Pathology results, 2016). X-rays of the swollen joints were "unremarkable", with reasons for the swelling remaining inconclusive (Radiology results, Radiologist #1, 2016). A whole-body scan reported, "asymmetrical mild increased blood pool and delayed activity in the right nasal cavity / maxillary sinus region" that "likely relates to sinusitis" (Radiology results, Radiologist #2, 2016). An electrocardiogram (ECG) indicated a "partial right bundle branch block" but again was inconclusive and potentially considered "normal" (Pathology results, 2016). A urine test reported high polymorphs and high epithelial cells, both between the 10–100 range but should have been less than 10, indicating a urinary tract infection, kidney or liver disease (Pathology results, 2016). An associated medical report added only "very mild arthritis in her right manubrium-clavicular region" around the sternum and collarbone area, but added it was "unusual in view of her signs and symptoms". The GP advised it was "possible that her symptoms are attributable to her current living condition and therefore would benefit from an investigation into her current situation urgently to address her significant symptoms" (General Practitioner #2, 2016).

Despite never having experienced non-specific gut symptoms and having made a personal choice at least five months prior to moving into the apartment to not consume gluten, I was referred to a gastroenterologist in September 2016 in an attempt to improve understanding of symptoms indicating possible inflammatory arthritis. The gastroenterologist discovered both chronic gastritis and coeliac disease, even though I had not knowingly consumed gluten for more than a year prior to that time (Pathology results, 2016).

With no effective long-term treatment or understanding of the cause of the widespread symptoms, by November 2016, I was referred to a rheumatologist. The rheumatologist reported the same symptoms – bullous eruptions, swelling, stiffness, "synovitis of the proximal interphalangeal joints 2–5 of both hands", but also then reported on similar symptoms to my right ankle and the "right metatarsophalangeal joints 2–4" (Rheumatologist, 2016). Proposed treatment was a form of anti-inflammatory drug that would require monthly blood tests to assess the ongoing functioning of my liver. In addition, I did not feel that the pain that this would address the vast array of symptoms nor did I think the cause(s) were fully understood, I was uncomfortable in consuming this drug in association with its potential side effects.

After a long year of ongoing and increasing severe symptoms and many medical tests, I was at a loss as to how to heal and what had caused the issues in the first place. During this whole year, I remained living in the apartment, trying most treatments and medications prescribed, to no avail. I struggled to drive my car and changing gear and pushing in the clutch had become even more excruciating. Walking became increasingly painful with the swelling and lesions of ongoing infections, which occurred in my hands as well. I struggled to even walk my dog, which had once been a daily goal when buying the apartment. Water burned the raw lesions like lemon juice to a small wound. Many showers were taken by sitting on the shower floor, with a plastic grocery bag taped around my foot or waterproof dressings covering my hands. Cleansing the wounds with a damp cloth and redressing them occupied hours, every day. My skin became inflamed with acne as well, with dark patches around my eyes that I desperately attempted to conceal under make-up. My eyes were constantly burning and bloodshot, and my limbs were generally incredibly sore and weak. Sharp pains also regularly inhabited my limbs and abdomen, and nausea was all too frequent. I was seeing specialist after specialist, each offering their own advice and prescriptions. It became increasingly clear that symptoms could only be addressed within each individual medical field, and so I was questioning the possible outcomes if I were to take each individual prescription – could my body actually handle it? I tried to hide how frail and unwell I really was from everyone I cared about and attempted to carry on each day as if everything were fine.

I was greatly concerned by the water damage that I felt remained unresolved, and still wondered how I could be in so much pain and so unwell since moving into the apartment. While I was researching mould and environmental illnesses, I came across a specialised practitioner with a focus on environmental illness, and I was able to schedule an appointment in December 2016. Although it remains a somewhat controversial illness, I was diagnosed with possible chronic inflammatory response syndrome (CIRS), with an expected recovery period of one to three years for me. This timeframe, however, would increase until the point at which I moved out of the apartment, removing myself from what was believed to be the cause. I packed a bag and went to stay at my parents' property, thinking I could have the apartment fixed within the next couple of weeks.

CIRS is defined and described as follows:

> an acute and chronic, systemic inflammatory response syndrome acquired following exposure to the interior environment of a water-damaged building with resident toxigenic organisms, including but not limited to fungi, bacteria, actinomycetes and mycobacteria as well as inflammagens such as endotoxins, beta glucans, hemolsins, proteinases, mannans and possibly spirocyclic drimanes, as well as volatile organic compound … Extensive data show that approximately 25% of the population have "mould susceptible" genes. For people within this 25%, their immune system does not effectively recognize and process biotoxins. If these individuals are exposed to a significant biotoxin load, such as a mouldy home, they are at high

> risk of developing CIRS. Unfortunately, even after the person no longer has ongoing exposure, the inflammation remains "switched on" and the symptoms remain. This inflammation further triggers hormonal, neurological and other imbalances. This chronic inflammatory response causes a broad range of systemic symptoms (Anonymous, personal communication, 2017).

THE FURTHER DETERIORATION OF MY HEALTH – 2017-2018

After spending around a year searching for answers and hoping someone could connect the pieces and help me recover, I was still somewhere in between hopeful and hopeless. I felt hopeful that I had found someone who could help me heal, with a strong understanding of the likely cause. But I also felt somewhat hopeless – how could I have become so unwell, more so than I had even realised myself in my attempts to get up each morning and to carry on with the day as normally as I possibly could. The possible CIRS diagnosis was followed in March of 2017 with further assessments of adrenal fatigue; kidney weakness; bacterial biofilms in the lungs, sinuses and bladder; sluggish liver function; sluggish gallbladder function; neurological system compromise; and small and large intestine parasite infections. A respiratory specialist also considered a form of systemic inflammatory response to possible allergens within my home (Respirologist, 2017). Allergy skin tests further acknowledged reactions to various allergens that were identified previously, but also displayed a reaction to *Alternaria alternata* – a species of mould (Pathology results, 2017).

By mid-June 2017, I was still not showing many signs of improvement. Further assessments indicated low thyroid function, mycotoxins in the system, continued adrenal fatigue, continued sluggish gallbladder function, lack of deep sleep, and bone and joint metabolism dysfunction. Some specific blood tests for water damage toxicity were undertaken (Shoemaker, 2019).

> ***Human Transforming Growth Factor Beta 1:*** *Result 2640 pg/mL.*
>
> ***Normal Range is < 2380 pg/ml****. High result indicates: "Neurologic, autoimmune and many other systemic problems also are found with high TGF Beta-1". The protein "plays a role in development before birth, the formation of blood vessels, the regulation of muscle tissue and body fat development, wound healing, and immune system function (especially regulatory T-cells)". "TGF Beta-1 can impair T-regulatory cell function, which in turn contributes to the activation of autoimmunity, yet TGF Beta-1 also plays a role in suppressing autoimmunity".*
>
> ***Vascular Endothelial Growth Factor (VEGF):*** *Result is < 31 pg/mL*

***Normal range is 31–86 pg/mL**. Low result indicates "Deficiency of VEGF … is a serious problem in biotoxin illness patients that must be corrected. If you don't have blood flow, cells begin starve and don't work properly".*

***C4a:** Result 15,988 ng/mL*

***Normal range is 0–2830 mL**. "C4a has [the] greatest significance looking at innate immune responses in those with exposure to Water Damaged Buildings. The proteins work with your immune system and play a role in the development of inflammation. Each complement activates inflammatory responses, with spill over of effect from the innate immune response to acquired immune response and hematologic parameters".*

***Alpha Melanocyte Stimulating Hormone (MSH):** Result 15.9 pg/mL*

***Normal range is 35–81 pg/mL**. MSH "has multiple anti-inflammatory and neurohormonal regulatory functions, exerting regulatory control on peripheral cytokine release, as well as on both anterior and posterior pituitary function. In mould illness, MSH will be too low in over 95% of patients. This means increased susceptibility to mould illness, ongoing fatigue, pain, hormone abnormalities, mood swings, and much more. MSH is a hormone, called a regulatory neuropeptide, and it controls many other hormones, inflammation pathways, and basic defences against invading microbes. Without MSH, bad things happen; chronic sleep disorders with non-restful sleep develop, and endorphin production is reduced, so chronic pain follows".*

The dermatological issues of the hands and left foot also continued, with the left foot being tremendously impaired and raw. A dermatologist believed the symptoms were more indicative of an infective course than inflammatory because of the unilateral manner of the signs. A biopsy of the foot returned inconclusive information, with the associated stitched wound at the biopsy site also becoming infected in the following days.

Pyrroluria exhibited in test results leading up to September 2017 – a condition in which an oversupply of pyrroles or hydroxyhaeopyrrolin-2-one (HPL) binds multiple cofactors and subsequently renders the cofactors unavailable to the body. Zinc and vitamin B6 are commonly those nutrients rendered unavailable, leading to associated deficiencies (FX Medicine, 2021).

Further tests were undertaken in September 2017 to investigate progress and ongoing symptoms.

> *Eosinophil cationic protein levels returned high at almost twice the normal ug/L units. "Elevated eosinophil catatonic protein levels result from activation of eosinophils in allergic or eosinophil syndromes" (Pathology results, 2017). The nucleolar titre detected previously had dropped to 160, but still suggested that scleroderma or connective tissue disorders were associated with those staining patterns.*

By October 2017, I had also been diagnosed with Hashimotos Thyroiditis.

> *Antithyroglobulin (Anti-Tg) returned higher than the maximum range of 116 IU/mL for normal levels, and anti-thyroid peroxidase (Anti-TPO) were almost four times the maximum range of 35 IU/mL for normal levels. This indicated "raised levels of thyroid antibodies may occur in prodromal hypothyroidism before TSH elevations occur and may also be seen in thyrotoxicosis, thyroiditis, non-toxic goitre and thyroid cancer. The levels of thyroid antibodies seen in Graves' Disease and Hashimoto's can be normal, but are usually elevated" (Pathology results, 2017).*
>
> *High eosinophil cationic protein (ECP) was still evident late 2017, suggesting possible Mast Cell Activation Syndrome (MCAS). MCAS is a "condition that affects multiple systems, generally in an inflammatory manner… [with] overlapping characteristics with recurrent idiopathic anaphylaxis… [and] "distinguishing symptoms, specifically hives and angioedema". Bowel tests to assess the condition of my intestines depicted overgrowths in* E. coli, Enterococcus sp., Bifidobacterium sp., Clostridium sp.*, limited* Bacteroides spp., *along with undergrowth's of* Lactobacillus sp. *and* Eubacterium sp. *(Pathology results, 2017).*

By March 2018, mould and toxins were believed to be within my tissue, and additional binders were recommended to assist with what felt like a further increase in symptoms. Increases in allergic reactions were evident in the ECP blood tests again, assumed to be associated to the highly reactive allergy tests conducted previously. Further increases were displayed in the Anti-TPO and Anti-Tg thyroid blood tests associated with Hashimotos thyroiditis. A low histamine diet and some additional supporting supplements aimed to decrease the histamine reactions. While many symptoms were reducing or subsiding, many still had not.

Almost two months after the additional allergy skin prick tests, I was still struggling with the removal and detoxification of the biotoxins. Further test results returned positive results of small

intestinal bacterial overgrowth (SIBO), a condition whereby "the presence of excessive numbers of bacteria in the small bowel [cause] gastrointestinal symptoms" (Pimentel et al., 2020, pp. 165-178).

> *Thyroid levels of Anti-Tg and Anti-TPO remained high in mid-2018 but had marginally decreased since the previous results in April of that year. Copper levels within the body were elevated, but calcium, magnesium, sodium, iron, boron and cobalt were all at the low end of the scale.*

THE FURTHER DETERIORATION OF MY HOME – 2016-2018

In late 2016, a white residual substance was discovered under some areas of carpet within the apartment, including within the main bedroom, which was suspected to be efflorescence. Visible mould was evident to some of the woodwork surrounding the ensuite, the ensuite, with corroded metal within the wall cavities. The engineer advised that mould spores on concrete surfaces can become problematic because of the porous nature of concrete, leading to near impossible methods to remediate the mould in its entirety. This is because integral structural elements like integrated steel could be further corroded with the application of harsh chemicals, and that scrubbing mould can release spores into the air. A rectification plan was advised, including invasive methods to identify the cause of the leak (Structural Engineer, 2016).

In early 2017, a few weeks after leaving the apartment, the main bedroom carpet was determined to have "at risk" moisture levels, while the concrete in that vicinity had "at risk" and "wet" readings. Readings at that level support microbial activity, with that activity potentially contributing to structural damage. The upholstered bed frame that was located within the main bedroom for the first few months of living in the apartment, indicated a presence of mould spores that had likely settled there. All levels of adenosine triphophate detected within the air condition (HVAC) system, bed base and ensuite walls were found "unacceptable" and indicated biological activity, suggesting further investigation was needed. Signs of water damage presented as rust and efflorescence within the basement below the apartment (Indoor Environmental Health Consultant, 2017).

Later in 2017, when I was still trying to determine the cause of the leak and extent of subsequent damage, further tests showed signs of water falling into the basement below when my showers were used. Water was falling through a misplaced penetration within the ground floor slab, which was covered and duct taped (refer to Image 1.8). When this was removed, cameras were inserted up into the penetration, which coincidently was located within the hob cavity for basin and toilet within the main bathroom. This cavity that was also shared with the ensuite and laundry dividing walls.

Image 1.9: Exposed, unused slab penetration where water was evidently falling through into basement

Note. From the author's personal collection 2018.

Image 1.10: Water found, through slab penetration, pooling in bathroom hob cavities

Note. From the author's personal collection 2018.

Videos depicted water pooling in these cavities, and tracking through to penetration (refer to Image 1.9). For how long had this been occurring? Had the waterproofing failed for both bathrooms? What was the extent of the damage? It appeared that further galvanised steel base tracks had also started to rust, indicating long-term water ingress and associated damage to the walls surrounding both showers, as well as the dividing wall between the bathrooms that also backed the laundry.

By the end of 2017, elevated VOC levels were determined within the ceiling and wall cavities, caused by water-damaged materials or mould. At the time of testing, viable surface and airborne concentrations were not elevated, but the range of genera between the wall and ceiling cavities, the outdoor reference sample and the indoor spaces suggested there was mould growth within the apartment (Occupational Hygienist, 2017). However, a supplementary report tested surface mould to the underside of the carpet both outside the secondary bathroom and front door, and both were considered at contaminated levels, (refer to Image 1.11 and Image 12; Occupational Hygienist, 2018).

Image 1.11: Underlay to carpet at apartment front door, behind the apartment's bathrooms
Note. From consultant, anonymous 2018.

Image 1.12: Under the carpet outside the secondary bathroom
Note. From consultant, anonymous 2018.

The investigation into the apartment and its potential relationship to the significant deterioration of my health continued in 2018. While the aluminium waterstop angle at the doorway between the ensuite and main bedroom was not sealed and fell short of the total door opening, it was discovered that the timber door jamb was not safeguarded with a waterproof membrane below the finished floor level in the ensuite. Efflorescence was visible within the basement, below the apartment and in the proximity of the unused slab penetration. Corrosion to the steel track behind the shower of the ensuite was also evident. Tests were also undertaken of the secondary bathroom, something that had not been thoroughly assessed previously owing to lack of visible issues. Similar crystallised salt was located within the floor waste's polyvinyl chloride (PVC) pipes, indicating water passing through the tile screed, like in the ensuite. Moisture levels of the surrounding walls and door jamb to the secondary bathroom were elevated. Mould growth was also discovered under the carpet outside this bathroom, very similar to that under the main bedroom carpet, with mould also on the plywood strips used to secure the carpet edge. Water was evidently making its way into the basement from both showers, as well as wet carpet to the common area corridor that backed both bathrooms. It was concluded that the waterproof membrane had failed in both shower recesses, the waterstop angles at both bathrooms doorways did not provide a continuous waterproof barrier, the

thickness of the dry film of the membrane was not sufficient and make-up air was deficient in allowing mechanical ventilation to the secondary bathroom (Building and Waterproofing Consultant, 2018*).

TWO YEARS AFTER LEAVING THE APARTMENT - RECOVERY

Whilst I had been out of the apartment for almost two years by *late 2018*, I still had significant inflammation that was not subsiding as much as I had hoped. In September, I sought help from another Immunologist who requested various skin prick allergy tests again – wheals were now demonstrated extensively as reactions to insects including *D. pteronyssinus*, *D. farina* and cockroach mix, both cat and dog dander, grasses including 5 grass mix, Bermuda couch, bahia paspalum, rye and platain, trees including birch mix, pine, olive and privet, and, not surprisingly, Alternaria alternata and aspergillus fumigatus as mould species. The swelling in the left ankle and hands again returned an unremarkable verdict in associated X-rays. An allergic rhinitis treatment plan was recommended, however, in an attempt to address the severe allergies, but many of the symptoms remained untouched.

During 2019, I was still struggling with exhaustion, blistering skin and aches and pains. But looking back at what I had experienced during the previous few years, I felt more hopeful. I was planning to re-enter the property market and searched for a small house that was at least 15 years old. Following the issues in the apartment that I experienced, I undertook some research on the ages of properties that could potentially have lower risks of issues such as VOCs and lead, and which had a sufficient level of building quality. I understood that the certification process was no longer independent as it was around 15 years ago. I requested the building reports from the relevant real estate agents to every property that appealed to me. If any revealed the slightest sign of water damage, I would not even inspect it. After seven months of reading reports, I had only managed to inspect three properties – one I dismissed quite quickly because of its condition that was somewhat obscured in the marketing material, another was built in the mid-1980's and the third was built in the mid-1990's. The latter two both needed some minor work, but appeared to have strong bones with little to no signs of water damage.

The final house I visited, from the 1990s, I was able to undertake a second inspection of. I took my family and they helped me inspect the roof cavity for any possible issues that were potentially concealed. Generally, it appeared to be okay, and I prepared the requirements to attend the auction. I can honestly say I had never been so nervous over anything previously, but with a bit of nudging and encouragement from family during the bidding, I was the final bidder.

Given the age of the property I engaged a family friend, a reputable builder, who had previously assisted me during the apartment issues, to renovate the bathroom – with my main priority being re-waterproofing of the wet areas for peace of mind. The renovation commenced in September 2019 but was not quite complete by the time I planned to move in for the new year. There was a fair bit of cleaning required, which was expected, but not ideal given some works were still in progress. My health fluctuated for the following months, something I understood was likely due to the new building materials off-gassing, VOCs and construction dust. Leading up to the settlement of the property, I searched for new furniture that would hopefully not be a high contributor to chemicals and VOCs in my new home because of my body's acquired sensitivities. I was in the process of this research, so I sought to minimise plastics and their associated phthalates, chemical treatments including fire retardants that could be applied to textiles, synthetic fibres, which too could off-gas, and composite timbers and coatings that could be high in formaldehyde. I will admit, I did purchase a small number of book case items that are made from a composite timber for my new home, but owing to financial restraints, I prioritised fair-trade and solid timbers for spaces where most time is spent, like the living room and bedroom, and natural textiles over synthetic fabrics where I could. I felt I was finally moving forward again, starting a new chapter and, more importantly, I was recovering.

However, following a number of heavy downpours and storms in 2020, in the months after I had moved in, I noticed that the downpipes and gutters were not coping with the capacity of rainfall and were even overflowing at times. This coincided with another health flare up – more exhaustion, more rashes and blistering, more widespread inflammation. While the flare ups could occur quite quickly, they unfortunately took much longer to subside. More months of struggling followed, but again, I felt some solace in the fact that I was still nowhere near as unwell as I had been during the previous years. Moreover, the long journey to recovery meant that the cause was better understood and, therefore, somewhat easier to tackle. I had the downpipes replaced for larger capacity alternatives and gutter guards fitted to all gutters to prevent any further blockages and allow rainfall to flow more freely. The eaves were assessed and did not appear to have sustained any permanent water damage, the mould was removed, and the surfaces were treated. Again, symptoms finally started to subside, albeit slowly.

I was able to once again get out and about daily with my pup; I was socialising again and focusing on my wellbeing both physically and nutritionally. However, I began to experience some swelling and bruise-like patches on one toe, and I assumed this was caused by an injury I had acquired stubbing my toe. But it was becoming more painful and was not healing. I visited the local GP and was prescribed a penicillin-based antibiotic under the belief it was infected. The antibiotics seemed

to have no effect on the toe, during or after the 10-day course. The exhaustion, headaches, nausea and blistering rashes returned. While not severe initially, they were once again keeping me up at night, every night, for about two months. The rashes had spread across and around my left foot, my hands were itchy, and joints were swelling up again. I had not changed my diet or been exposed (to my knowledge) to any toxins that could have led to the new onset. I spoke to my specialist doctor who had assisted over the past few years with the illness associated with the water damage, and some supplements were increased, including treatment focused on restoring a healthy gut flora. Within a couple of weeks, the symptoms were again residing, but more so than they had before, and I was ecstatic.

While I still have a number of health 'conditions', I feel better than I have in a long time. Nowadays, I tend to react quite easily to certain foods – coincidently to those listed in the top 5 foods most likely to contain mould. Not to mention, exposure to mould itself, strong perfumes and fragrances, and certain toxins can occur daily. I still take supplements to maintain a healthy system, and I have also learned the hard way that I can no longer tolerate mould-based medications. I avoid any places I know that could contain water damage, and I try to minimise risk in my own home of any mould growth. I am an amateur but keen vegetable gardener now, aiming to grow my own chemical-free produce, and I try to complete an hour of physical exercise and mindfulness daily. I can honestly say that the last few years have been the hardest I have ever gone through, but I have been fortunate to emerge on the other side. I have a strong support base with my family and friends and a large group of incredible people who had faith in me and in my situation and stood by me. I only hope that I can use my experience to help others in the future by informing and encouraging people to be aware of the impacts that the built environment can have on our health.

My experience has added to the intense interest I have in the construction industry, to uncover possible causes of health detriments from the built environment and determine what role or responsibility architects and designers have in ensuring we are not compromising people's health for the purposes of budget or aesthetics. It has always been my passion to provide healthy interior environments.

Put into perspective, more than 100,000 chemical compounds have been invented since 1939, and 95% of the chemicals utilised within construction products do not have sufficient information available for health implications (F. a. Pacheco-Torgal, 2012). So, what are the true risks, where does the responsibility lie, and what is the true cost of poor design, material selection and construction processes for society?

2. LITERATURE REVIEW

2.1 BACKGROUND

Society nowadays spend at least 90% of their time within the interior built environment, where indoor air quality is compromised compared to external environments and despite indoor air pollution being classified as the ninth highest "Global Burden of Disease risk" (Kumar et al., 2016). Scientific evidence is increasing that connects contemporary diseases to contemporary exposures, especially exposure to chemicals and pollutants (Sundell, 2010, pp. 185–186). Experts from the World Health Organisation (WHO) have determined that indoor air can be 4 to 6 times more polluted than outdoors, and 6 to 8 times more toxic, with indoor air contamination leading to minor ailments and distress through to more significant illness such as asthma, allergic reactions and long-term problems like cancer (Zhigulina & Chumachenko, 2017, pp. 1–2). However, in the past couple of years, societies around the world are finding themselves confined to their own homes at an even higher rate, as a result of trying to minimise the effects of the SARS-CoV-2 COVID-19 pandemic (Burtscher et al., 2020, pp. 1544–1545).

Interior spaces are becoming progressively more impervious, especially for cross-ventilation or fresh air and often for the purpose of energy efficiency, consequentially leading to a hazardous accumulation of indoor allergens and toxic substances (Staff, 1993, p. 1). These can include moulds, dust mites, VOCs and the off-gassing of interior building products, finishes and furnishings. Relationships between endocrine disruptors and semivolatile organic compounds (SVOCs) commonly present within the interior environment are also becoming concerning and increasingly studied, which can include biocides, flame retardants and plastercisers (Benning et al., 2013, p. 2696). During a 45-day lockdown in Madrid, Spain, outdoor air quality improved drastically. However, indoor air quality became compromised with an increase of 12% in daily mean PM2.5 concentrations and Total Volatile Organic Compounds levels increased by 37% to 559% (Domínguez-amarillo et al., 2020). Dedicating so much time indoors surrounded with potential toxic concoctions, society are also degrading their microbiome and immune system without critical exposure to the natural environment. Recent studies over the past decade have concentrated on the notion that disease and immune deficiencies can be influenced by a lack of biodiversity (Haahtela, 2019, pp. 1445–1456).

Toxic substances within such building materials or products can include flame retardants, plasticisers and even pesticides (Cao et al., 2012, p. 24). The Environmental Working Group (EWG) in the United States (US) has undertaken a number of research papers over the last decade,

with one in particular, “The Pollution in People: Cancer-causing Chemicals in Americans,” corroborating that almost 1,500 chemicals or groups of chemicals are likely to be carcinogens, and that there has been a rise in kidney, liver (organs that are vital in detoxification processes) and youth cancers, while children are often more vulnerable (DellaValle, 2016). Evidence is slowly being compiled to markedly link particular illnesses to exposure of some chemicals or toxic substances. Lidia Morawska and Tunga Salthammer explained that pollutant particles can directly affect an individual through both inhalation and ingestion (2003).

A team in South Korea outlined that unhealthy interior environments can not only affect one’s health but also one’s productivity and wellbeing. The team confirmed that toxic chemicals are prevalent within furniture, floorings, paints, adhesives, cabinetry and wall furnishings, and that these compounds can include formaldehyde, acetone, benzene, toluene, styrene, acetaldehyde and xylene, to name a few (H. Kim et al., 2017).

An extensive combination of constituents that include insects, mites and dander, and fibres that can be synthetic, natural, from carpet, upholstery, furniture, drapery and bedding, all can form dust particles (Saha, 2016, p. 25). A study (Hwang et al., 2008) concluded that exposure to indoor contaminates and dust could potentially be more harmful to society, especially to children, based on their time spent indoors and proximity to potential exposure surfaces, than their dietary intake. It is understood that an amalgamation of psychological and physiological factors can influence the extent of the impact that interior pollutants have on younger children, including their size, developmental stage, ability to remove toxins from their system, proximity to the floor and so on. The authors collected interior dust samples from vacuum bags used at one community hall and 10 residential homes in California and uncovered five primary harmful substances in the analysis of said samples – phthalates, chlorinated pesticides including polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs) and pyrethroids (pp. 26–33). Therefore, the outcome of the study recommended advanced research into indoor toxins, encouraging public knowledge and raising awareness of the potential health implications of such toxicity. This thesis strives to offer further research into indoor pollutants and the effect they have on its occupants.

However, a conflicting response questioned the validity of Hwang et al.’s methods of analysis of the five categories of chemicals discovered in the vacuum cleaner bags from the original study. The critique suggested that there was a lack of confirmation of the vacuums and associated bags or for how long they were in use, and additionally questioned the toxin exposure levels and subsequent health implications (Hover et al., 2009, p. 5194). Nevertheless, considering vacuums are used with

the intention of maintaining clean and healthy environments, it is concerning that these chemicals are evident within the interior built environment incipiently.

A subsequent response by Hwang et al. (2009) to Hover et al., published by *Science of the Total Environment*, concluded that while there were some editorial issues in their work, "the levels of toxic chemicals in indoor dust were high enough to be possibly linked to some adverse effects" (p. 5197), and some of the chemicals exposed in their tests are in fact categorised by the United States Environmental Protection Agency (US EPA) as "probable carcinogens" (p. 5197).

It should be noted that, by toxicology definitions, a toxin and a toxicant differ. A toxicant is any chemical substance that has the ability to harm a living organism, but can be synthetic or naturally produced. Contrastingly, a toxin is of natural origin but is typically a metabolic product that has developed as a defence mechanism against pathogens or predators (Cope et al., 2004, p. 65).

This research investigated the pervasiveness and health implications of six common harmful substances. In conjunction with my personal interest in the topic and the prevalence in at least 50% of buildings in Australia today (Brambilla & Sangiorgio, 2020), *mould* will be the first toxin examined. Secondly, due to *formaldehyde* being one of the highest concentrations of all VOCs found in interior environments (Fang et al., 2019, pp. 309–318), formaldehyde will be scrutinised. And in addition, this research will also delve into the four prominent pollutants that were discovered as highly prevalent by Kim et al's team in indoor dust samples within their research noted above; that is, *phthalates*, *polychlorinated biphenyls*, *polybrominated diphenyl ethers* and *pyrethroids* (or more broadly in this case, textile pesticides). All six of these toxic substances have raised concerns in recent years, are highly prevalent within the interior built environment and can cause multiple symptoms or lead to multiple types of illnesses. Founder of Healthy House Institute, Jack Bower, wrote in the Healthy Housing Reference Manual, "walking into a modern building can sometimes be compared to placing your head inside a plastic bag that is filled with toxin fumes" (Centers for Disease Control and Prevention and U.S. Department of Housing and Urban Development, 2006).

2.2 MOULD

Mould is biological toxin that can affect the built environmental significantly, including workplaces, schools and the home. Humid and warm circumstances can trigger the growth of mould, with spores often evident within the atmosphere. The spores are considered inactive, although a return to their favoured conditions can lead to further growth (Geller, 2006). While references to mouldy constructions and toxicity trepidations have occurred as early as Leviticus,

since the 1900s the issue has become more concerning because buildings and their materiality have changed, along with factors that include a decrease in ventilation rates for energy efficiency and an increase in air conditioning and its subsequent indoor humidity. Building materials developed from plaster to paper-lined gypsum sheets and there was an increase in timber and wood composite products, which were becoming more prone to temperature changes and were supportive of condensation, especially around the building envelope (Miller & McMullin, 2014, pp. 9953–9966).

Mould is associated with elevated moisture levels and can occur in any building with such circumstances. However, it has also been found in building materials prior to being installed, often from the manufacturing process or during transportation. A common consequence is the decomposition of wood products. Mould has also been known to grow in interior areas where there is a moisture build-up with little ventilation, for example, behind vinyl or PVC products like wall coverings. Paints and lacquers nowadays often incorporate moisture resistant and antibacterial additives to reduce such complications. Porous materials carry a high risk when associated with moisture, since mould can propagate into them, cause deterioration and severely minimise opportunities for remediation – with replacement then being the ultimate solution. The development of mould is dependent on the temperature, humidity and surrounding air (Adan, 2011).

While mould is considered natural, it can be harmful to one's health, especially when evident indoors. Mould is known to generate irritants, allergens and even mycotoxins, leading to reactions that include hay fever–like indicators, dermatitis and respiratory or asthmatic issues (Harris & Moore, 2009, pp. 307–318). Additionally, two health reviews undertaken by the Institute of Medicine and WHO in 2004 and 2009, respectively, further advised of the health implications of mould and mentioned other possible health issues, including bronchitis, allergic rhinitis and even hypersensitivity pneumonitis (Cox-Ganser, 2015).

A study undertaken by the Pollution and the Young project (PATY) of more than 58,000 children between 1988 and 1999, from 10 countries from around North America, Russia and Western and Eastern Europe, assessed the plausibility of indoor air quality affecting the respiratory functions of youth. It was recorded that mould was evident in over 39% of the North American children, with lower levels of almost 14% evident in the Russian children, and subsequent connections found between the children exposed to the moulds (Antova et al., 2008, pp. 708–714).

In addition to mould, water-damaged buildings often contain a vast concoction of other toxins. These can include other fungi, bacteria, mycotoxins, antigens, VOCs and endotoxins (Shoemaker & House, 2006). Mould and water-damage has also been associated with diagnoses of 'Sick Building

Syndrome' (Zhang et al., 2012) and Multiple Chemical Sensitivity (Hyvönen et al., 2020) of which both will be further discussed in 'Chapter Four – Toxin 1: Mould.'

2.3 FORMALDEHYDE

Formaldehyde is a chemical believed to have been initially defined as early as 1855, but was concocted in 1867 by August Wilhelm, a German pharmacist, by synthesising methanol through dehydration. Resins utilising the chemical were produced from the 1900s as binders and adhesives for wood products, especially chipboard, although it was not until around 1980 that the potential carcinogenicity of the substance was questioned (Salthammer et al., 2010b, pp. 2535–2357).

It is now understood that the petroleum-derived chemical contains carcinogenic elements that have caused it to become banned in particular skincare and make-up products in certain countries (Statham, 2011), even though it still exists in some cleaners and even wood-comprising building products, with temperature being one of the factors that influences the rate of off-gassing (Huang et al., 2015). The Australian Air Toxics Program classifies the chemical as a "priority air toxic" due to its impact to air pollution and pervasiveness as a common additive in commercial processes (Lazenby et al., 2012).

This toxic substance is often still incorporated into timber flooring products (both plywood-comprising engineered types and solid timber), plywood sheets, some wallcoverings, melamine and even gypsum plasterboard to name a few – which has contributed to it being one of the highest categorised products impacting indoor air quality (IAQ) as a volatile organic compound (Böhm et al., 2012). The identification of the toxin within carpets, and some foam insulation also, was identified by the US EPA in 2006. It is also evident that respiratory and sinus conditions can be instigated from products containing formaldehyde, including upholsteries, window furnishings, wallpaper and rugs (Saha, 2016, p. 8).

A recent study from 2017 (Huang et al.) assessed the indoor formaldehyde levels associated with renovations between 2002 and 2015 for 39 Chinese cities. It was noted that humidity, air deviations and temperatures affected homes between the southern and the northern cities, with the northern areas carrying higher levels. Average readings of 125µg/m3 were depicted internally following renovations during this same period; however, the World Health Organization (WHO) have nominated a maximum limit of 100µg/m^3. These results are believed to be due to the increased emission proportions of contemporary building materials (2017, pp. 194–205). The WHO outlined that a secondary establishment of formaldehyde can occur within the atmosphere, including

internally in environments, through VOC oxidation processes, although the full effects of this are still being researched (Europe, 2010, pp. 103–142).

Another study (Rovira et al., 2016) discovered that, despite the substance not bioaccumulating within the environment, synthetic versions are constantly being released from a vast list of products or materials, which leads to toxic long-term exposure for much of society. The study indicated significantly higher levels of formaldehyde occurring internally when compared with external areas, probably stemming from the building materials and furnishings of the interior, with only 2% of the exposure levels originating from exterior behaviours. The main pathway for exposure is inhalation, although absorbtion through the integumentary system is also detrimental (pp. 357–363).

2.4 PHTHALATES

Phthalates are synthetic chemical compounds formed from phthalic acid esters and are predominantly used as plasticisers for flexibility and malleability properties (Wynters & Goldberg, 2012). Primarily owing to the flexibility and malleability properties phthalates produce, even though they are a toxic element, they are commonly used as plasticisers and can exist within faux leather, vinyl-associated products including upholsteries and resilient flooring, wall coverings and electrical conduit insulation (Xu et al., 2010). The compound has been in production since the 1920's (Zarean et al., 2016). It surpassed the former camphor and castor oil alternative as a plasticiser following the development of triphenyl phosphate, which was a turning point towards ester-based plasticisers and stimulation of the polyvinyl chloride (PVC) industry began (Bocqué et al., 2016, pp. 11–33).

In addition to being evident in interior dust, phthalates from off-gassing of surrounding materials as well as artificial fragrances are found in indoor air. The volatility of phthalates is caused by the toxin's ability to disperse within the material or become extraneous and airborne because it is not chemically bound to the actual material, often contributing negatively to indoor air quality and VOCs (Boor et al., 2014). Phthalates can disengage from their polymer chains, allowing diffusion into the surrounding environment and a pathway for exposure via absorption, ingestion and inhalation (Pyeon et al., 2017). While they can ordinarily maintain low toxicity levels, phthalates can also exist in paints, glues, wall coverings and cosmetics, with a variant of the compound, that being di-(2-ethylhexyl) phthalate (DEHP), also evident in cabling, wiring and toys (Saeidnia, 2014, p. 928).

Over the past few decades this toxic substance has slowly become linked to some autoimmune conditions including asthma, and acknowledged as an endocrine and immune system disruptor (Ait

Bamai et al., 2014). Phthalates can be quickly metabolised by the body and expelled through urine or faeces. However, for such chemical compounds to actually be absorbed or ingested by the body in the first place is rather alarming. High levels of exposure to DEHP, the common form used in food packaging, and PVC plastics has the potential to generate foetal growth implications, testicular complications and carcinoma. Meanwhile, the phthalate compound more commonly found in vinyl floor and wall coverings, adhesives and faux leather – Butyl benzyl phthalate (BBP) – has been linked to testicular diseases, birth defects and can affect other bodily endocrine processes, as evident in animal test studies. Former animal studies have exhibited toxicity following exposure to phthalates, indicating that serious caution should be advised in order to minimise human exposure from ubiquitous sources within the interior environment (Tran & Kannan, 2015, pp. 489–499).

2.5 POLYCHLORINATED BIPHENYLS

PCBs have low conductivity and high thermal resistant properties, making them prevalent in plastics, paints, dyed paper, transformers and capacitors to name a few. They are a group consisting of 209 congeners and 10 homologs with each having their own variations to the chlorine atoms (Yang et al., 2019, pp. 1–13).Common use of PCBs within fire retardants and plasticisers result in this toxin also existing within interior environments (Johnson & Wiley, 2011).

Despite the US banning the production of PCBs in 1977, there is still great concern of contamination environmentally and to human health due to their persistence within the surrounding environment (Hwang et al., 2008, p. 31). Importation and production into Australia have also been prohibited since the 1970's, with enduring processes in place by State and Federal Governments to phase out PCB-containing items appropriately (Environment Protection Authority Victoria, 2017, pp. 1-3).

It is believed to have detrimental impacts on the integumentary, digestive and immune bodily systems (Sherwood, 2015). PCBs, and also the chlorinated pesticide dichlorodiphenyltrichloroethane (DDT), are classified as xenobiotics and are both known to bioaccumulate. That is, the body doesn’t excrete these toxins readily and instead stores it within the body’s tissue. These toxic substances can even be transferred to the foetus in utero (Crinnion, 2009, p. 347) where the baby is undergoing prenatal development, laying down the foundations for physical and mental life, at a time when the baby has with a weaker blood-brain barrier, and so is very susceptible to possible toxicity.

During a study conducted by the EWG in 2005, two laboratories tested the blood in the umbilical cord of 10 babies all born in 2004 between August and September. They tested the blood for 413

chemicals and found 69.5% of those chemicals evident in the babies. Of the 287 of 413 toxins discovered, 62.7% are known carcinogens, 75.6% can negatively affect the nervous system and brain, and 72.5% can affect the development of the infant. All 10 of the newborn babies tested displayed the same minimum of 101 chemicals in their blood, with a total of 147 out of 209 tested PCBs (Houlihan et al., 2005). This further confirms the effect that PCBs can have on the body, and also that bioaccumulated toxins have the ability to pass through to the womb.

2.6 BROMINATED DIPHENYL ETHERS

Brominated diphenyl ethers (BDEs), and the common compounds of PBDEs, are classified as organobromine compounds, that is, bromine bonded to carbon to form an organic compound. They are extensively used within the building industry mostly since the 1970's, applied to materials and furnishings for fire retardant properties. Some applications include polyurethane foams including mattresses, textiles, plastics, furniture, polystyrene and electrical equipment (Feng et al., 2014, pp. 49-54). The spread of fire and degree of combustion is reduced when, at elevated temperatures, bromine radicals are released into the atmosphere from the PBDEs (United States Environmental Protection Agency, 2017b).

It is understood that PBDEs are carcinogens, effect thyroid processes and neurological behaviour, and impact the growth of foetuses (Chen et al., 2012, pp. 2420–2429). With a similar molecular structure to PCBs, unfortunately PBDEs also bioaccumulate within the environment, raising concerns about the effects on human health. Pentabromodiphenyl ether (pentaBDE), octabromodiphenyl ether (octaBDE) and decabromodiphenyl ether (decaBDE) are the three most common PBDEs, with the latter being the most utilised across the world. The former two types have primarily been phased out in 2004, after being manufactured by only one known producer and included to Annex A of the Stockholm Convention – this is an international treaty directed at human and environmental protection from assiduous organic toxins. DecaBDE was to be phased out by 2014, having been initiated by the United States EPA. Despite Australia not producing PBDEs and also being a part of the Stockholm Convention, decaPBDEs are still being imported into the country and contributing to the bioaccumulation within the environment and therefore providing further human exposure (Stasinska et al., 2013).

A study (Stasinska et al., 2013) conducted in 2013 of 30 residential and indoor dust domestic samples found that DecaBDE was the most prominent component at 66% of all PBDEs tested, in homes of pregnant women in Western Australian, also indicating that ranges were lower in rural areas compared to metropolitan areas. They concluded to further investigate the correlation between intensities and various areas (pp. 187–193). A further study by the same team in the following year

involving 164 pregnant women, 'Australian Maternal Exposure to Toxic Substances' also undertaken in Western Australia, displayed evidence of higher median intensities of PBDEs within women during their 38th week of pregnancy than women of the same demographic in the same period of gestation in some European areas, as evident from international quantitative studies they examined, although these were considered to be lower median intensities of PBDEs than found than in the US. They stated that there were no significant connections from accommodation, diet or maternal or foetal health factors amongst the women involved in the study. The 164 women involved were simply a minimum of 18 years old, non-smokers, were not occupationally exposed to such toxins, and maternal and birth data was provided (Stasinska et al., 2014). This raises questions as to why these various geographical areas in Australia are exhibiting higher levels of PBDE exposure than other zones internationally.

A Taiwanese study (Chen et al., 2012) outlined the impact and potential spread of PBDE within the food chain, due to the bioaccumulation of the chemical within the environment. The study was based on the sampling of sediment within twelve Taiwan rivers. They noted that the dry seasons seemed to allow a greater accumulation of the toxins, whereas these were reduced during the wet seasons under the assumption that the water flow was able to transport the chemical further through the environment. Interestingly, the river tallying the greatest PBDE readings was the Bajhang River, noted to have been near a landfill establishment and possibly contributing to the nearby water catchment areas. The study also refers to PBDEs being present in some local fish species, with further criticality raised of the effects it can have on the food chain, including as far as breast milk (pp. 2420–2427).

2.7 PYRETHROIDS & TEXTILE PESTICIDES

In contrast to phthalates, chlorinated pesticides are believed to be much more difficult to excrete from the body, and despite having been banned from use in many countries, including the United States since the 1980's, they are another toxic substance commonly found in domestic dust, often due to materials still in existence from prior to their prohibition. Whilst about half of the recognised deviations have been restricted for the past few decades there is still a good portion in existence, especially due to the bioaccumulation of the pesticides since their introduction in the 1940's (Crinnion, 2009).

Pyrethroids are nowadays generally synthetic, based on the natural alternative pyrethane, which originates from the chrysanthemum flower and is utilised as an insecticide – primarily for mosquitoes – surpassing the organochlorine pesticides and other traditional types. The study noted above by Hwang et al. concluded that detectable levels of pyrethroids and chlordanes were found in

all of their apartment samples, with more than 94% of 513 residences across Seattle, Los Angeles, Iowa and Detroit utilising insecticides either within or around their homes (Hwang et al., 2008). Extreme use of the pesticide has been known not only to leave enduring elements that affect water, soil and cultivated produce (Xu et al., 2018, pp. 2-9), but also have an evidential impact on society. Detectable levels of at least one pyrethroid and organophosphate pesticide exist in 90% of children in the United States, indicating that exposure can lead to neurotoxicity (Global Pesticide Campaigner, 2003). The pesticides have been known to also be used as domestic pesticides, pet treatments, mosquito repellents in clothing and even for lice applications.

While dosage determines the toxicity of pyrethroids, the pesticides have been found to have low effects on mice through absorption of the integumentary system, although oral or digestive absorption creates much higher levels of toxicity. From studies undertaken on rats, the alcoholic and acidic components of the chemical are understood to be quickly excreted from the body via faeces, urine or respiratory exhalation in a matter of days. However, the carbon elements have been found to bioaccumulate in the rat's body tissue and the stomach (Kaneko, 2011, pp. 2786-2791).

A study (Han et al.) from 2017 indicated that there were presently few studies on the impact of pyrethroids specific to human. However, more than 130 considered-healthy patients and more than 70 patients suffering from coronary heart disease in China had their urine assessed against 3 pyrethroid variants. Their study publicised a potential connection between heart disease risk and pyrethroid exposure, with their apprehensions focussed on long term exposure despite the prompt metabolic detoxification process undertaken by mammals. They advised of increasing concern of the volatility of such chemicals, suggesting that traces have been discovered in fresh farm produce, fish, rivers and even breast milk (2017, pp. 646–670).

An earlier study (Julien et al.) from 2008 specifically aimed at assessing both organophosphate and pyrethroid pesticides from within 42 public housing units in Boston. Critically, the team of researchers advised that almost three-quarters of homes in the early twenty-first century were estimated to utilise pesticides, with multi-unit homes demonstrating a more profound application of such pest control. Of the 11 pyrethroids and two organophosphate chemicals tested, permethrin (a pyrethroid) and chlorpyrifos (an organophosphate) exhibited the highest results from swabs of the kitchen floors of all homes tested, with a further four pesticides also evident. Remarkably, the authors uncovered relationships between the pesticides discovered from the kitchen floor swabs and the vacuum dust assessed from living rooms (Julien et al., 2008, pp. 167-174). The organophosphates – chlorpyrifos and diazinon – were prohibited for interior domestic use, in 2001 and in 2002, respectively. Though despite these toxins still being evident within these households

six to seven years later, it is very alarming. The banning of these two substances has led to increased utilisation of the pyrethroids; however, the synthetic version is more tenacious in the environment than its natural counterpart, suggesting more research is needed to assess its toxicity against humans, especially when it is clearly evident within small or contained indoor environments.

2.8 INQUISITION

IAQ is a subject of great concern, with many toxic substances evident within the interior built environment carrying properties to disperse into the atmosphere, or simply circulate within the air awaiting their perfect conditions to thrive, for example mould spores. In contrast, some harmful pollutants do not have such diffusing characteristics, but they can transfer their toxicity to occupants through absorption, like chemically treated textiles, or digestion, as per some plastic materials. Fabrics and textiles, for instance, are typically produced from extensive procedures that can involve up to eight thousand synthetic chemicals (Plell, 2018). These toxic substances can then be transferred onto final textiles in the form of clothing or upholstery, allowing for inhalation or absorption by users. (Kim et al., 2014). Despite being considered a natural fibre, cotton production utilises one of the largest global quantities of pesticides (Nussbaumer, 2004). Meanwhile, toxin ingestion by infants is believed to be common, since certain textile dyes are understood to degrade into carcinogenic pungent substances (Field et al., 2014).

A report by the EWG stated that national laws currently do not deliver the necessary mechanisms to the EPA or the Food and Drug Administration to reduce or even prohibit some toxic substances already classified as ‘known’ or ‘probable’ carcinogens (DellaValle, 2016). Individual substances require more advanced and accelerated research into the negative impacts on human health as a consequence of exposure to them, while further knowledge and publicity will assist designers, builders and the general public to create their own informed decisions in the interim.

Exposure to toxic substances in the interior environment is of increasing concern (Villanueva et al., 2013), particularly because there is still a long way to go in determining the full extent, firstly, of the presences of these substances, and secondly, of the health implications of exposure to these substances. In addition, it is important to identify healthier alternatives where they exist, and also effective remedial responses where the toxic substances have been found to be present. This thesis asks how and why such toxicity is possible in contemporary life, and how and where it occurs. Why are toxic elements being incorporated into commonly used building materials, and are there healthier alternatives available? If so, why are the alternatives not they being further developed and utilised? *What is the true impact on human health?*

3. METHODOLOGY

3.1 RESEARCH AIM

This study explored the relationships between the interior built environment and the continuing presence and occurrence of toxic substance, and the negative impact on human health. It will seek to expose what chemicals are used in common building products and what the health implications are on society (refer to Figure 3a below).

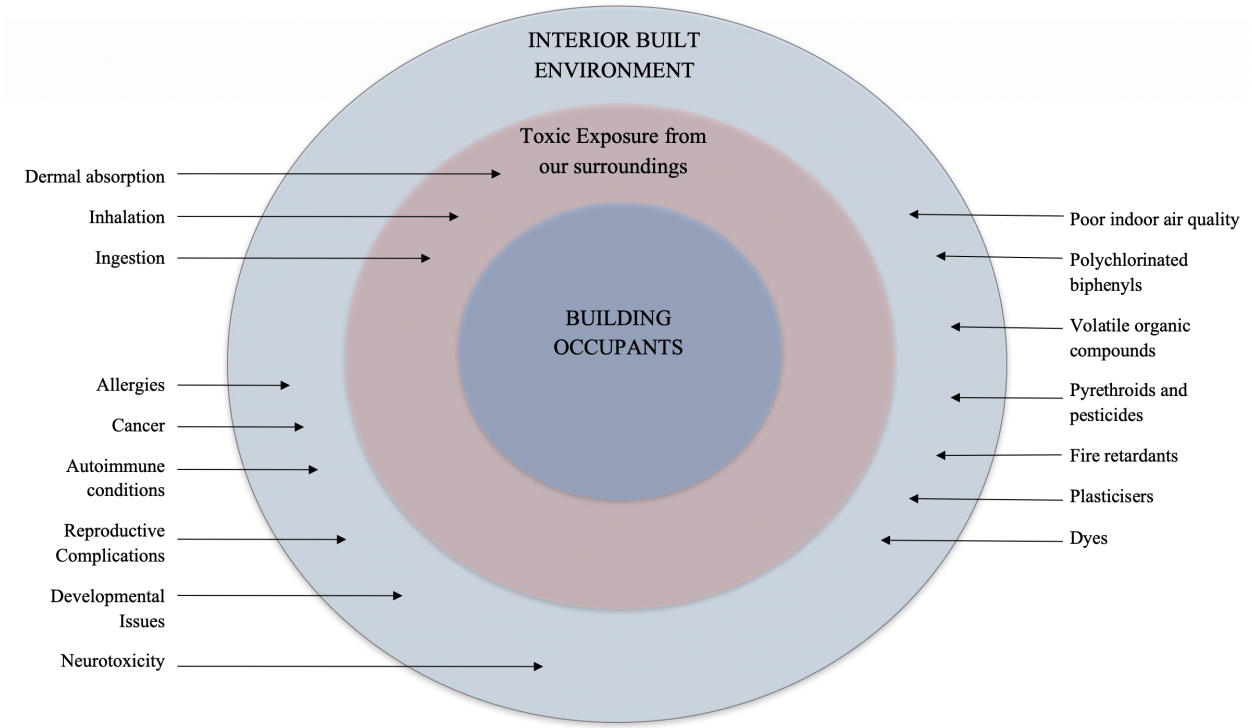

Figure 3.1: Diagrammatic illustration of topic

3.2 RESEARCH OBJECTIVES

The objectives of this dissertation concern the triangulation of three critical factors:

1. To select and analyse a series of toxic substances prevalent in interior environments. Identify **the toxic substance** – *What* is it, *how* is it made and *why* is it used?
2. To identify **the health implications linked to the toxic substance** – *How* does it affect human health and *what* are the health impacts?

3. To investigate **the life cycle of the interrogated toxic substance** within the common building materials to determine if there are any negative impacts from reuse and recycling its related products – *What* are the life cycle implications?
4. To synthesise the above objectives and offer **recommendations or actions** that could reduce the negative health consequences of these toxic substances.

3.3 METHODOLOGY

RESEARCH METHOD

Case study formats have often been applied to research where thorough explorations and qualitative methods investigate current situations, which would otherwise be difficult to establish through survey and experimental approaches, while also examining the reasoning associated to the situation (Zainal, 2007, pp. 1–6). In addition, Robert Yin also described case study research as a dual characterisation. Firstly, this method of research analyses a 'contemporary phenomenon' from a realistic perspective, expressly when the delineation between the phenomenon and the context is not evidently apparent. And secondly, it revolves around the data collection methods and the case study's design, in the sense that its variables of relevance prevail over data points, triangulation assists in the compiling and assessing of various evidential sources and can find former theoretical proposals advantageous (Yin, 2014, pp. 2–23). This methodology investigates *why* and *how* inquiries respond to current incidents or occurrences, and that the design can be customised accordingly (Meyer, 2001). Further, Yin also conveys the importance of the *why* and *how* factors when formulating case studies, with the explanatory methods requiring investigation of the past, despite the contemporary focus (Yin, 2014, pp. 1–10). The case study approach is further validated with a partial focus on natural sciences, in the sense that it has a strong focus on qualitative foundations and the underlying intentions, or rather, what may be concealed beyond the more objective evidence and the reasoning behind the decisions associated with the evidence (Gillham, 2000, pp. 1–8).

RESEARCH DESIGN

Yin discusses the following five critical elements in formulating the case study *research design* (Yin, 2014, pp. 26-69):

1. *Study Questions*

   The study revolved around the following study questions -

- *What* toxic substances (that are negatively impacting on human health) are *commonly occurring in the contemporary* interior built environment?
- Following the findings of the first question, *why* were such decisions made to implement such toxic substances into the interior built environment?
- *What*, if any, alternatives are available?

2. *Study Propositions*

   This research evaluated *why* such toxic substances are being used in the built environment, *how* they are being incorporated, and *what* are the implications on the human health?

3. *The Case – Unit of Analysis*

   The Case, or Unit of Analysis in this study, is the presence and negative impact on human health of toxic substances within the interior built environment. This explanatory research examined *what* toxic substances are incorporated within common building materials, *how* the specific building product is being used and *why*. A literature-based multiple-case study has formed a 'collective study' of individual instrumental studies to form the foundation of structure (Sarantakos, 2005). Each chapter focussed on a particular toxic substance to contribute to a depth of understanding of the case.

   Analysis of the data included the examination of the background and historical purpose and value of the element (refer to Figure 3.4a below).

4. *Linking Data to Propositions*

   In this study, an analytical strategy was implemented in a number of ways to select and interrogate data in relation to the study's propositions. Data collected from the literature examined theoretical propositions to aid in forming the 'Cases,' since they assisted to form the basis of the literature review, research questions and new propositions. Data analysis helped form patterns and produce various methods of relaying the evidence in the thesis, with the collection and collation of both qualitative and quantitative data to investigate and analyse the events associated with the Cases. Analytic generalisation aimed to consider previous research concepts, whether it supported or discarded them, or generated new theories to encourage further research.

5. *Criteria for Interpreting a Case Study's Findings*

As statistical analyses are not too prevalent in this type of case study, a focus once again was on rival explanations. A strong focus on sourcing multiple rival explanations was interpreted to either reject or include them to form stronger conclusions. The interpretation of such research will be moulded by scientific evidence already in existence (despite possible rarity) from peer reviewed research that has been published in scholarly journals. A final chapter synthesises the findings, tables the occurrence of toxic substances in the interior built environment and their related negative health impacts on human health and, finally, makes recommendations for future investigation.

Yin examined various techniques and tests to deliver a quality case study, which included reliability, external validity and construct validity. Construct validity was a prime focus throughout the data collection stages to ensure reliability, through the exploitation of multiple evidential sources, creating connections between the research objectives and compiled data, and utilising the expertise of key informants with the literature review of the thesis. Being an explanatory study, internal validity was critical in ensuring that conclusions formed – of *how* the built environment can affect human health – are genuinely a result of their presence within the surrounding environment, without neglecting a miscellaneous factor. The methodology gained external validity from the theoretical foundation, where the outcomes can be compared with equivalent studies or research through analytic generalisation. Multiple peer-reviewed research papers and studies, for consistency and repetition, from in-depth and non-bias sources, support the reliability of the research, and a database was utilised to archive the data methodically. Archival records, including peer-reviewed journal articles, provided the primary source of such information for analysis.

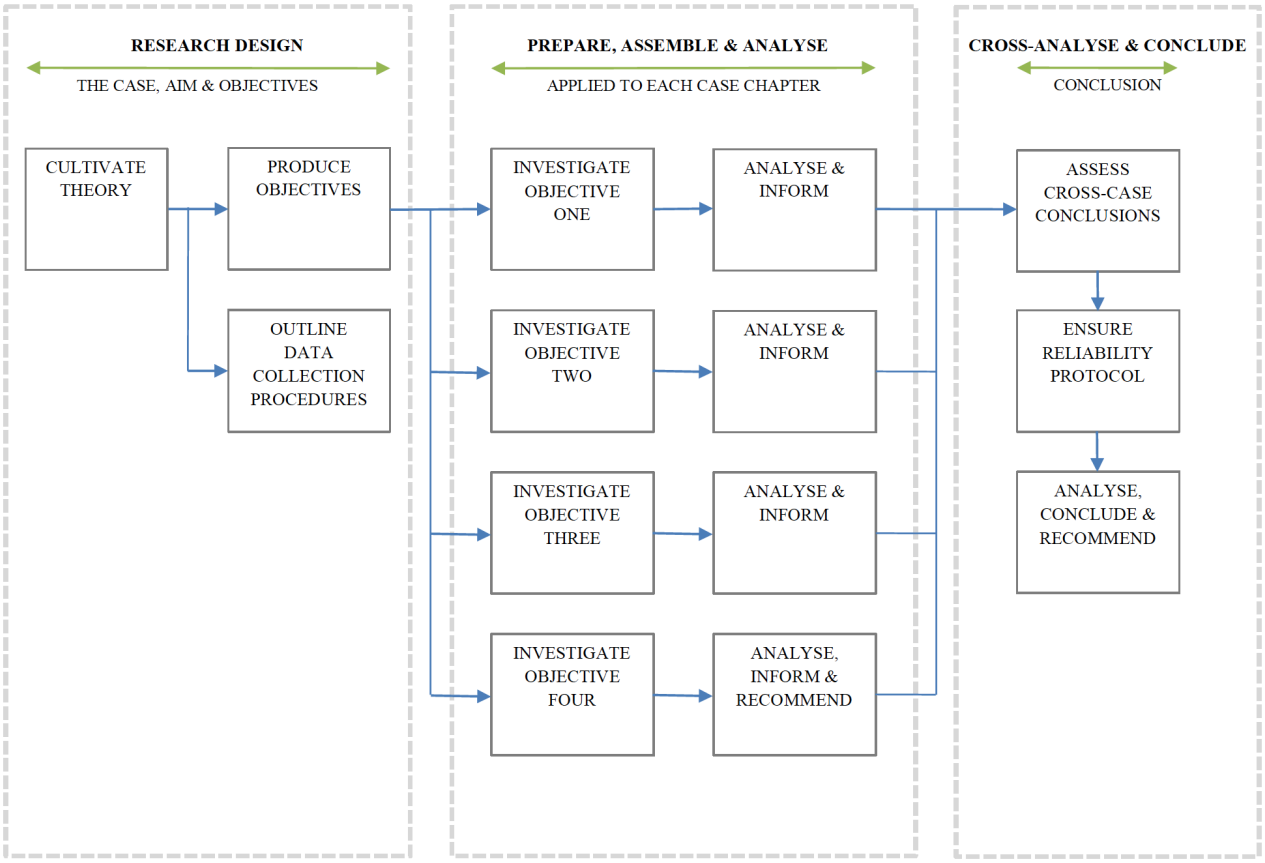

Figure 3.2: Case study approach

PREPARATION

A number of critical elements were pursued throughout the study, while also forming the initial intent and preparation of the study. These elements that must be considered are:

- Awareness – During the collection of research, an analytical assessment must occur for each piece of evidence and subsequently determine if further evidence is required on such topic (which will also enhance the reliability).
- Adaptation – It must be acknowledged that over the course of the study, shifts may occur as a result of the evidence content. While the original Case remains important, processes may need adapting in the event of unpredictable findings, encouraging precision but not inflexibility.
- Understanding – A strong understanding of the motive of the Case will assist in retaining a focus and its objectives, for a stronger study.
- Restricting Bias and Maintaining Ethical Principles – Avoiding a conclusion arising from predetermined views of the Case must be restricted to ensure reliability and credibility. The

uppermost ethical standards must be sought throughout this research from a research perspective, including animal and human protection. Owing to the nature of the topic and the historical content required, the analysis of existing material, data and evidence is assessed. Similarly, the cross-nature of various fields, including health, chemistry, construction and design, requires an in-depth investigation of preceding data to decipher further the connections between these fields. This approach alleviated the need for ethical human or animal testing of any form. However, ethics approval was sought in relation to including a personal experience, with associated evidence, within the Introduction.

- Protocol – A four-section protocol assists in the reliability of the study –
    - Part A: Case Study Overview
    - Part B: Procedures for Data Collection
    - Part C: Questions During Data Collection
    - Part D: Guide for the Case Study Report (Yin, 2014, pp. 71–99; refer to Appendix A).

COLLECTION OF EVIDENCE

Multiple Sources

While case study research can often include six common source types – archival records, documentation, observations (both direct and participant observation), interviews and artefacts – this study gathered evidence primarily from archives and existing documentation because of the nature of the cases. The cases required a historical interrogation to examine the occurrence of a series of commonly occurring toxic substances in building materials and why they first came to be used and then continued to be used, thereby providing a more contemporary focus drawing on recent studies and scientific evidence. Importantly, the research was not restricted to these two formats of evidence. Such evidential sources included journal articles, scientific studies and reviews, books, e-books, former case studies, manuscripts, press releases and reports, along with former newspaper and magazine articles to interrogate further the reasoning behind the decisions that formed these cases – specifically, why have such toxic elements been integrated into our surrounding environment. This approach provided a layer of subjectivity in lieu of other similar case study methods, like interviews and observations, owing to the historical focus. The triangulation of data collection was critical to obtain consistency and reliability from the evidence. Therefore, the data was interrogated while observing the reliability techniques noted above, to restrict possible bias. It should be noted that some historical sources, like early text books, for instance, may offer some evidential information but it has already become evident that many earlier

textbooks have now become superseded by much more recent research. However, this was strongly monitored with the use of reliability techniques, as mentioned above in this paragraph.

In terms of the toxic substances themselves and health implications, it was assumed that some chemicals associated with particular building products, for example, phthalates, formaldehyde, PBDEs or PCBs, will result in specific chemicals being holistically investigated – both as an individual or isolated element and in combination with the overall chemical content of the product. This was consequential to the literature review, which outlines both prevalent and harmful substances that are common in interior spaces today.

The search strategy sought to examine these exposed chemicals, or the cases, and were assessed and researched primarily through digital databases. The crucial sources included journal articles, previous research papers and publications, and various databases in a broader sense, including (but not restricted to) the *Journal of Interior Design*, *British Medical Journals*, *Additives for Polymers*, *Environmental Science & Technology*, *The Science of the Total Environment*, *Building & Environment* and the *Journal of Hazardous Materials*. Governing policies and bodies investigated include the *Building Code of Australia*, *Australian Standards*, Green Star, *Well Building Standard*, Safer Solutions Australia, EWG, Environment Protection Agency and the National Pollutant Inventory, to name a few. Search terms that proved vital across the toxins are noted below (refer to Table 3.1).

Table 3.1: Significant search terms utilised across the case

Toxic Substances	Built Environment	Health Implications
Phthalates, formaldehyde, mould, fungi, moisture, microorganisms, poor indoor air quality, Di-(2-ethylhexyl) Phthalate, brominated diphenyl ethers, polychlorinated biphenyls, textile pesticides, pyrethroids, carcinogen, endocrine disrupters, dust	Plasticisers, vinyl, polyvinyl chloride, water damage, Environmental Protection Agency, Australian Government regulations, Australian Building Code, Australian Standards, PVC, polyurethane, cotton, organic, manufacturing and/or production processes, paints, lacquers, coatings, timber, composite, flooring	Cancer, allergies, asthma, developmental issues, reproductive, bodily systems, neurotoxicity, endocrine system, dermal absorption, inhalation, ingestion,

Case Study Database and Chain of Evidence

A database was formed to compile all evidence and research gathered. Sources were listed in an Endnote library that is accessible online and set up for two separate devices. Each source listed contains a digital copy of the research, whether it be a journal article, report or other format. A

separate archive library also contains an additional copy of the research, listed alphabetically by author and title. Progress notes were also archived for future reference, whether scanned handwritten notes or saved electronic notes. These were archived by date of publication for ease of access.

This method of archiving will also assist with maintaining a chain of evidence, ensuring the ability to backtrack the process from how the conclusion was formed to the original question and objectives. This was possible through ongoing and consistent referencing throughout the case study report, with all sources listed at the end in the References section. Page numbers of source evidence are listed within in-text references, which is most important for textbook or hard-copy sources when a digital version is unavailable.

ANALYSIS OF EVIDENCE

A number of methods provided the basis for analysing evidence. Pattern matching was utilised to compare or contrast empirical findings in the data with the study propositions. Internal validity can be strengthened if these assumed patterns align (Yin, 2014, p. 143). As an explanatory study, explanation building was critical in defining the causal relationships between both *how* and *why* toxic substances are incorporated into the built environment and *how* they affect the wellbeing of an individual, linking back to the original theoretical propositions. A time-series analysis was important given the historical nature of *why* such decisions had been made to introduce particular toxic substances into the surrounding environment. This was especially interesting in the concluding chapter of the cross-case analysis, comparatively assessing the toxic substances of how long they have been incorporated into the built environment, how long they can have negative effects on human health and bioaccumulate within the environment (especially those that have been restricted). Similarly, logic models assisted in connecting any empirical data with the theoretical propositions in the form of a series of events and the transitions in between. As a multiple case study, evidence was also analysed as a cross-case synthesis, specifically in the concluding chapter, to relate or juxtapose the conclusions of each toxic substance.

SHARING AND REPORTING THE STUDY

Various elements were considered during the sharing and reporting phase of the study. Importantly, the research is aimed at both academic colleagues and design and construction industry colleagues – ultimately including policy makers, manufacturers or creators of such building materials, specifiers, designers and other professionals. However, this research also aims to in the general population, so society too can formulate educated choices, to ensure their surrounding environment is as healthy as

possible. In directing this research at two somewhat different communities, the thesis intends to be as clear and succinct as possible, especially regarding the cross-field analyses between the construction and medical fields.

As noted previously, the criticality within both these fields has encouraged the format of a multiple case study, whereby each chapter focuses on one toxin as a single case and address each of the objectives individually. A final chapter delves into a cross-case study to assess relationships between each toxic substance. Owing to the objectives of the study, the thesis has a theory-based structure – each chapter contributes to the overall conclusion. Each chapter leads with an introduction of the nominated toxic substance, as well as a brief history of its existence. This leads to a description of where in the built environment the toxic substance resides and the health implications of the toxic substance. Then, an outline is provided of the causal link between the two critical factors and the purpose of such substances prevailing within the interior built environment in the first place.

3.4 LIMITATIONS OF THIS STUDY

This case study methodology differs from a historiography as it can include a vast array of evidential types, including observations, interviews and documentation (Yin, 2014, p. 12). However, the research was partially limited by pre-existing data, although all opportunities to pursue alternative formats were sought. Because many connections had not already been made between the built environment and health implications, pre-existing research and evidence was limited in this cross-field area. This enforced the need to connect the evidence associated with the two disciplines, in a professional and non-biased manner.

Since no human or animal testing were undertaken, nor chemical procedures, the research presented is primarily a literature review. This thesis draws on existing literature to collect data that is analysed and amalgamated – for example, in relation to the chemical structure of toxic substances, their function or history, their use within construction, building materials and their purpose – and then forms direct links and connections between the relevant points to build evidence-based recommendations. The outcomes aim to bridge the gap between these two fields where a high level of ambiguity seemingly exists behind how harmful our surrounding environment actually is. Analytic generalisations aim to develop and conclude the theories, without including statistical generalisations that may more likely be present in an experiment format approach.

4. TOXIC SUBSTANCE ONE: MOULD

4.1 INTRODUCTION

Biblical times saw the first references to the possible health implications of mould exposure in water-damaged buildings. Remediation protocols were noted in Leviticus, in the *Old Testament*, and if remediation was not successful, buildings were demolished, and belongings disposed of (Gray et al., 2003, p. 411). Mould is a fungal contamination, and although having existed for a long time, its impact has increased since World War II, subsequent to the supply and utilisation of more cost-effective building materials, which became favourable for mould growth, and which in turn impaired building quality (Goldstein, 2010, p. 1). It is now estimated that between 18% and 50% of all buildings suffer from mould or dampness (Mendell et al., 2011, p. 748).

Many moulds that are ubiquitous within interior environments create mycotoxins, which are secondary to the mould fungi. These organisms can cause genotoxicity, neurotoxicity and cytotoxicity and are also immunosuppressive, owing to their volatility and ability to become airborne (Peitzsch et al., 2012, p. 908).

4.2 THE TOXIC SUBSTANCE

WHAT IS THE TOXIC SUBSTANCE?

Mould is a type of fungus and is, in fact, the most prevalent form of fungi on the planet. Society can be exposed to the toxin on a daily basis, often through inhalation (Woodson, 2012, p. 3). The family of mould includes mildew, yeast, mushrooms and puffballs. Mould spores circulate within the surrounding atmosphere and begin to reproduce when they come into contact with moist surfaces, which they then begin to consume that surface material and amplify to eventually destroy the material (Woodson, 2012, p. 3). Humidity, lack of ventilation and direct water contact encourage mould growth (Rabinovitch, 2012, pp. 645–646). Permeation of moisture and water from bathrooms, hydraulic conduits, windows and roofs can all lead to condensation and water damage to internal materials, although it is the biocontaminants and mycotoxins that can wreak havoc on health (Jedrychowski et al., 2011, p. 989).

WHERE IS IT FOUND?

Mould can exist both internally and externally and is strongly supported by moisture, although it can withstand harsh environments, including dryer surroundings (Woodson, 2012, p. 3). The fungal growth can affect building materials differently based on their composition, design and

construction, as well as on the surrounding relative humidity levels (Johansson et al., 2013, p. 201). Internally, relative humidity, temperature, occupant behaviour, building materials and construction quality can all contribute to providing the perfect environment for mould to prosper (Sadovský et al., 2014, pp. 348–349). A study that focused on mould in Swedish residential attics outlined that moisture originating from external sources, permeation of damp interior air through surrounding building materials and rising damp are common sources that encourage fungi (Nik et al., 2012, p. 96).

Locations or existence of mould are dependent on temperature and relative humidity, as well as on the building material composition. Two options can be investigated to determine the presence of mould. Firstly, measurements can be taken in a simulating laboratory or on the construction site. Or, secondly, programs can assess these parameters by applying mathematical solutions (Fedorik & Illikainen, 2013, pp. 19–26). However, it is the moisture content and ability to absorb moisture that distinguish which materials are more susceptible to mould growth than others (Gradeci, Labonnote, Time, & Köhler, 2017, pp. 77–88). It is difficult to establish exact susceptibility statistics of individual materials to determine a baseline of where mould might begin to generate. A Swedish study conducted over two-and-a-half years assessed nine generic building materials regularly used in residential construction. These included pine sapwood, plywood, thin hardboard, chipboard, moisture resistant plasterboard, exterior grade plasterboard, asphalt paper, cement-based board, fibreglass board and expanded polystyrene board. These were located in attics and crawl spaces in a number of different homes. Some of these materials demonstrated mould growth over the testing period; however, the results varied across the samples based on the material and its moisture content, relative humidity and temperature. Significantly, the fungi were not evident on the fibreglass, expanded polystyrene board or cement-based boards (Johansson et al., 2013, pp. 201–209). While this suggests that these three materials have a lower moisture content than the other tested materials, the study was clear that the temperature, relative humidity and length of exposure to such conditions also play a vital role.

Another critical factor that can influence indoor temperature and humidity and, in turn, mould growth, is today's lack of ventilation within interior environments. Over the past couple of decades, energy efficiency has become an idealistic consideration in new buildings, which leads to minimal natural ventilation throughout the spaces and encourages moisture accumulation. These approaches to energy efficiency were also found to affect higher moisture-containing materials, which include timber products such as plywood and other wood composites and cellulose-containing products; even anything that contains dust can become a food source for fungal growth. Fibreglass batts and plasterboard are commonly affected on their cardboard or paper side owing to the cellulose present.

Interestingly, building products that do not have a high moisture or cellulose content, such as bricks and concrete, can still be layered in dust that can act as a food source (Skrobot et al., 2017, pp. 6490–6503).

The below image demonstrates common locations in which mould may exist within a typical interior environment (refer to Image 4.1).

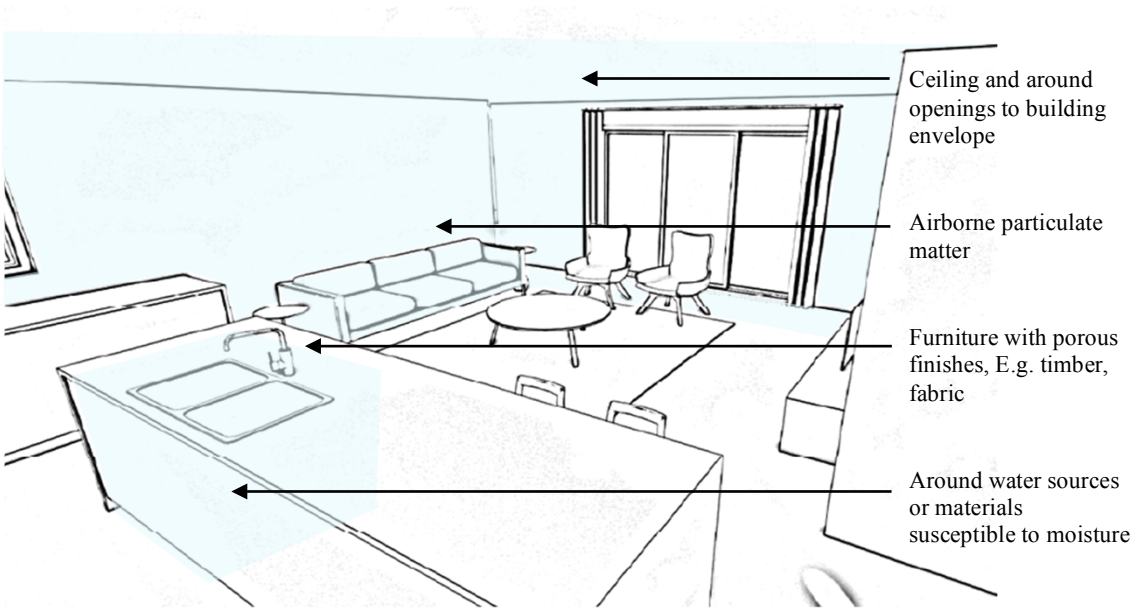

Image 4.1: Perspective of typical interior environment and prevalence of mould

4.3 HEALTH IMPLICATIONS OF THE TOXIC SUBSTANCE

HOW DOES EXPOSURE OCCUR?

The health implications of exposure to mould vary from person to person based on the duration of exposure, severity of biotoxins and mould, pre-existing health conditions and even personal gene composition (Hope, 2013). Toxicity, allergy and infection are the three pathogenic mechanisms to mould-related diseases (Terr, 2009), with ingestion, inhalation and dermal contact being the primary routes of exposure (Campana et al., 2019, p. 509). It is understood that around 24% of the population are susceptible to illness arising from biotoxin and mould exposure (Lanthier-Veilleux et al., 2016, pp. 1-2). An HLA-DR/DQ haplotype represents genetically susceptible people, which can

create an onset of inflammatory biomarkers, leading to a vast array of symptoms (Gunn et al., 2016, pp. 320–325).

Ingestion of mould has been known to produce implications for one's health, with mycotoxicosis being a specific condition arising from such exposure (Terr, 2009, pp. S217–S222). While not specifically relevant to the building industry, a 2009 study found that mould species can contaminate foods via agricultural crops, airborne mould spores and more. Further, the team outlined that allergenic reactivity can be influenced even by low quantities of aeroallergenic mould extracts (Luccioli et al., 2009, pp. 433–442). This suggests not only that mould toxicity can occur via ingestion but also that contamination of foods can occur from airborne spores that exist in the surrounding environment.

Despite some researchers crediting severe mould-caused diseases to occur only via ingestion (McKnight et al., 2011, p. 13), exposure to mould has been found to occur through inhalation also. Allergens that originate from mould spores are circulated throughout the air, both indoors and outdoors, leaving inhabitants susceptible to inhalation of the toxins, especially those who are predisposed genetically to atopic conditions (Gonianakis et al., 2006, pp. 354–362). Glucan, which is a family of glucose polymers including cellulose, is found in the cell wall of mould species. It is β-1,3-glucan that can influence immune system processes, compared with β-1,4-glucan, which is reasonably harmless as cellulose. β-1,3-glucan is a key contributor in the progression of pulmonary diseases, from both an allergic and inflammatory perspective (Rylander, 2010, pp. 9–13).

Mould can also lead to reactions in sensitised individuals from dermal exposure. Allergists can apply minute amounts of mould allergens directly to the skin and prick the skin slightly to allow the skin to break. If any of the skin pricks react, primarily through rashes and swelling of the target area, this typically indicates an allergic reaction. A study in Greece assessed 571 atopic people with these tests using 10 specific mould species. They discovered that 243 patients displayed positive reactions via their skin to at least one of the mould species. The species that exhibited positive dermal results (from the highest displayed) were *Alternaria*, *Cladosporium* f*usarium*, *Aspergillus* and *Mucor*. All but 10 of the patients with results reacted to more than one of the mould extracts(Gonianakis et al., 2006, pp. 354–362). Sadly, the New Orleans flooding associated with Hurricane Katrina in 2005 resulted in significantly water-damaged homes and premises, and dermal exposure as well as inhalation occurred to the emergency staff and first responders, recovery personnel and returning occupants through sediment, mould films and airborne mould spores (Ashley et al., 2009, p. 609).

WHAT ARE THE HEALTH EFFECTS?

Background

While litigation cases and the ubiquitousness of mould have led many to argue about the actual health implications of mould exposure, ongoing evidence suggests that exposure can lead to respiratory problems, cognitive and neurological disorders, developmental complications in newborns, insomnia and migraines (Tuuminen & Rinne, 2017, pp. 1-2). A number of laboratory tests identify the severity of mould exposure in a person. Some of the critical biomarkers that can be assessed for water damage exposure (and subsequently, mould) are as follows:

- *Vasoactive Intestinal Polypeptide* (VIP) – VIP is a neuroregulatory hormone. VIP levels are extremely low in patients suffering from mould exposure. The hormone regulates inflammatory responses, but physically presents as shortness of breath.
- *Melanocyte Stimulating Hormone* (MSH) – MSH is a regulatory neuropeptide hormone that manages the body's defences against invading microbes, as well as inflammation pathways. This hormone presents at a low level also in at least 95% of mould illness patients and the deficiency can lead to chronic pain, continual fatigue, mood swings and hormone irregularities, to name a few.
- *Transforming Growth Factor Beta-1* (TGF Beta-1) – TGF Beta-1 is a regulatory protein of the immune system. It assists in cell proliferation, motility and apoptosis. The protein can weaken the function of T-regulatory cells, which can subsequently activate autoimmunity.
- *C4a* – C4a are proteins that move throughout the body and are associated with the immune system. They have become one of the most important markers in determining mould illness. They can activate inflammatory responses, which identify as high levels within 12 hours following exposure, and levels will remain elevated until treatment successfully occurs.
- *Vascular Endothelial Growth Factor* (VEGF) – VEGF is a polypeptide that acts to stimulate the creation of new blood vessels and elevate blood flow within capillaries. Low levels of VEGF can lead to the starvation of cells and, consequently, impairment of bodily functions.
- *Anticardiolipins Autoantibodies* (IgA, IgG & IgM) – While antibodies are proteins that the body creates to attack foreign invaders, autoantibodies are antibodies that attack the body's own cells. IgA, IgG and IgM are autoantibodies usually associated with collagen vascular diseases. Levels are generally higher in mould-affected patients.
- *Matrix Metallopeptidase 9* (MMP-9) – MMP-9 is an enzyme that is programmed by the MMP-9 gene. It has been evident in the pathogenesis of chronic obstructive pulmonary disease (Shoemaker, 2019, pp. S217-S222).

Mycotoxicosis

Various fungi generate secondary metabolites called mycotoxins. The most recognisable mycotoxins are:

- *Aflatoxins*, which are created by some strains of *A. parasiticus* and *Aspergillis flavus*,
- *Trichothecenes*, which are produced by mould species that are considered deficient, which can include *Stachybotrys atra.* and *Fusarium spp*.
- *Ochratoxins*, which are produced by *Aspergillus* and *Penicillum* (Hintikka & Nikulin, 1998, pp. 66-70).

Mycotoxins can lead to mycotoxicosis, leading to chronic or acute health conditions, especially when the mycotoxins are ingested (Bryden, 2007, pp. 95-101). Mycotoxicosis is a condition named to address collectively a wide array of symptoms believed to be caused by exposure to mycotoxins, secondary metabolites caused by fungi (Rogers, 2003, pp. 528-532). Mycotoxicosis can also be caused by inhalation of mycotoxins, and also by inhalation of endotoxins or other associated toxic substances. The condition is a rare but known occupational disease in farmers (Terr, 2009, p. S217). These toxins can also specifically lead to malfunctioning of leukocyte cells and the hasty division of cells because they can obstruct the synthesis of proteins through eukaryotic 60S ribosomes (Assouline-Dayan et al., 2002, pp. 191-201).

Cancer

While cancer has been linked to mould exposure (Laakkonen et al., 2008, p. 489), people suffering from cancerous illnesses and malignancies have a greater risk of contracting infections associated with invasive mould exposure. This is because the immune system is compromised by pre-existing disease. However, immunosuppression can also affect the severity, and even the mortality, of suffering individuals who have undergone immunosuppressive treatments like chemotherapy, especially paediatric cancer patients (Georgiadou et al., 2012, pp. 125-135). An 80-year study demonstrated that the lungs, followed by sinuses and soft tissue, were the exposure sites that lead to invasive mould infections. The most common infections were caused *Aspergillus spp*., affecting 23 out of the 28 patients. Though *Zygomycetes*, *Alternaria spp.*, and *Fusarium oxysporum* were also discovered in the remaining patients. The authors concluded that while mould infections are not common, they can prove fatal to those who are already immunocompromised, namely, transplant and cancer patients (Al-Rezqi et al., 2007, p. S349). Aspergillosis, as well as candidiasis, has increased since the 1950's in patients already suffering from acute leukaemia, with the *Zygomycetes*, *Fusrium* and *Scedasprium* species increasing as the causes of such mould infections. Infections by these species are most commonly caused by the following:

- Aspergillosis – Caused through inhalation of spores through sinuses and lungs. The spores attack blood vessels and corrode cartilage, bone and facial planes, leading to infraction and thrombosis. This is most common during construction of hospitals.
- Zygomycosis – Similar to Aspergillosis, but with a higher fatality rate, blood vessels are affected, and it can lead to pneumonia, osteomyelitis and meningitis, to name a few.
- Fusariosis – Again, similar to the above, the spores can affect the blood. Around half of patients are fungemic and up to 80% can develop skin lesions(Safdar et al., 2011, pp. 3-10).

Respiratory Conditions and Allergies

Allergies to mould can generally present as allergic rhinitis, asthma and other disorders associated with the respiratory tract, especially after inhalation of spores by mould-sensitive people (Luccioli et al., 2009, pp. 433-442). In fact, more than 60 published studies have assessed the relationship between mould exposure and the exacerbation or incidence of asthma and allergic rhinitis (Le Coq et al., 2018, pp. 1-18). High levels of TGF Beta-1 have been linked to an increase in childhood asthma occurrences, with the US EPA advising that exposure to water-damaged buildings has caused around 21% of new instances of asthma (Shoemaker, 2019).

A study focusing on the outcome of mould ingestion by 34 adults who were referred to Georgetown University Medical Centre surprisingly discovered a vast array of reactions that reflected allergy-like symptoms. Of those people evaluated, 91% had allergic rhinoconjunctivitis, while 44% also had asthma, 32% had food allergies and 6% had eczema. Results were generally elevated in those adults who were already considered sensitive to mould than in those who were not (Luccioli et al., 2009, pp. 433-442).

Hepatotoxicity

Hepatocellular carcinomas are one of the most lethal forms of cancer. This condition is more prevalent in certain geographical zones, including Southeast Asia and Sub-Saharan Africa. It is understood that a combination of hepatitis B virus (HBV) infections and sustained consumption of particular mould-containing foods that produce aflatoxins are the primary contributors. While genetic disposition, immune capabilities, exposure to other toxins can of course affect the development of such carcinomas, it is recommended not only to vaccinate susceptible communities against HBV but also to minimise exposure to such toxic aflatoxins in food sources – for example, by correct food storage and substituting corn for rice) (McCullough & Lloyd, 2019, pp. 76-84).

Sensitivity

'Biotoxic exposure' occurs when a person is exposed to a biotoxin, such as mould, which can trigger a rise in inflammatory markers; but unfortunately, it is not traditionally assessed in conventional medicine. Undiagnosed or untreated inflammation will gradually affect various bodily systems as it causes damage to organs. Dr Heyman, an integrative medicine expert, explains that the brain in particular will change over time as a result of the inflammation, leading to a brain injury that can be physically represented as memory loss, fatigue, mood changes and brain fog, to name a few. The key to resolving a biotoxin illness is holistic triangulation by the following methods:

1. avoiding exposure – removing oneself from all sources of exposure, for example, remediating all mould-effected building materials
2. preventing inflammation – the body continues to create inflammatory markers until the symptoms are addressed and exposure is no longer occurring
3. repairing the body from the damage caused by exposure and inflammation (Heyman, 2018, pp. 20-21).

Multi-symptom Illnesses

Cognitive disturbances, headaches, respiratory issues, weakness and fatigue, gastrointestinal concerns, depression, physical pain and other symptoms without other specific causes have been referenced as 'sick building syndrome' (SBS) owing to the prevalence of various accumulated toxins polluting indoor air. These toxins can include pesticides, smoke, poor ventilation, carbon monoxide, uncomfortable temperatures and high humidity levels, with a common occurrence of visible microbial effects within water-damaged buildings. While this has been a controversial topic over the last two decades in particular, various biotoxin-associated tests (as outlined above in What Are the Health Effects - Background) have validated the relationship between water-damaged buildings and these types of symptoms, leading to systemic and hypothalamic processes (Shoemaker & House, 2005).

Approximately 20–25% of people have congenital gene sequences that render them susceptible to particular biotoxins, including mould. Similar to SBS, symptoms can range from anxiety, depression, insomnia, gastrointestinal issues, memory loss, headaches and endocrine disruptions, to name a few, all of which are produced by underlying, widespread inflammation at the systemic level. The inflammation can accumulate and damage bodily organs if left untreated or undiagnosed. Brain injuries are even possible, due to neurological inflammation. Unfortunately, a patient who has these susceptible genes will not recover without specialist intervention owing to the causal genetic activity. Treatment must encase 'three pillars' – omitting all forms of exposure to the biotoxins,

impeding the inflammation, and healing (Heyman, 2018). This multi-symptom, multi-system illness, subsequent to microbial growth and water-damaged buildings, has been termed ‘chronic inflammatory response syndrome’ (CIRS). This condition too remained controversial until recently, owing to a lack of consistent pathophysiological means existing and unsubstantiated laboratory diagnosis, since no biomarkers were dependably recognised. The illness has been ‘diagnosed’ as sick building syndrome (SBS), fibromyalgia, and even given a medically uncertain diagnosis (Ritchie et al., 2013, p. 396).

Multiple chemical sensitivity (MCS) has also been associated with mould, water damage and the associated toxins – the term ‘dampness and mould hypersensitivity syndrome’ is an umbrella term that is used nowadays to address CIRS, mould illness and biotoxin illnesses, for example. MCS was first reported as early as the 1950's, with primary symptoms being complex, continual and causally non-specific, and with the central nervous system being most effected. Reactions can occur consequentially to low dose exposure to smoke, synthetic odours, formaldehyde, dust, adhesives and the like. Despite MCS being acknowledged for more than half a century, few studies relate the condition directly to chronic exposure to dampness microbiota. A recent peer-reviewed study assessed and validated a large array of extrapulmonary symptoms subsequent to exposure to the microbiota of dampness(Hyvönen et al., 2020, pp. 173-174).

4.4 LIFE CYCLE IMPLICATIONS OF THE TOXIC SUBSTANCE

The life cycle of mould fungi occurs in four phases – sporulation, germination, hyphal growth and reproduction. Spores will stay dormant and settle on surrounding surfaces until moisture or nutrients become available to them. If they do not receive sufficient sustenance, they remain dormant and will not germinate. If they do, hyphae development ensues, and they develop into a mass of mycelium. The mycelium metabolises the material substrate as they acquire more nutrients and moisture. It is then, in the final phase, that the mould establishes a reproductive organism and produces more spores. While it is mostly organic and high-carbon-containing materials that are most ideal for fungi, organic matter like dust can encourage mould growth to establish on inorganic materials also (F. Pacheco-Torgal, 2012, pp. 334-350). In addition, mould can commonly develop with relative humidity levels as low as 65% and indoor temperatures ranging from 15 to 32 degrees, strengthened by minimal sunlight and ventilation condition (Singh et al., 2011, p. 37). With the combination of a damp nutrient, apt temperatures and air, mould can grow. Without these conditions, mould may remain dormant for many years, even centuries (Harkins, 2003, p. 21).

With specific focus on mould consequential to water damage in buildings, typical mould can be further classified into a life cycle of four phases, with specific regard to water-affected homes,

including from flooding – sediment, vapour, mould film and aerosolised spores. These stages were characteristic of many homes during the aftermath of Hurricane Katrina in New Orleans in 2005. Noting that water exposure affected many properties for almost a fortnight and to a depth of two to three metres, semi-volatile and volatile toxins within the sediment then transmuted into the vapour in the dwellings, which created inhalation risks to returning occupants and responders through gas exposure. The gas exposure was then further encouraged through the continual and ongoing humid, moist environment that led to mould growth on furniture, floors, walls and any other porous internal surface. Toxic spores were then released into the surrounding atmosphere from these mould films. Exposure can be accumulative and have an even more dangerous effect on occupants, and in instances such as these, the incidence of inhalation through the vapour, mould film and gas phases are toxically accrued. Significantly, further research is required to understand the impact of mould as a partitioning and transport method for semi-volatile and volatile toxins, rather than simply acknowledging mould as an individual toxin in isolation from its environment (Ashley et al., 2009, pp. 609-619).

Importantly, the life period of mould spores is dependent on both the species of mould and the conditions in which they can survive (Miller et al., 2011, p. 31). HVAC systems (or rather, heating, ventilation, and air conditioning) can play a significant role in the life cycle of mould spores, as can building materials, water incursions, rain permeation, defects with the design and/or construction, unsuitable HVAC methods and maintenance, to name a few (Moon, 2010, pp. 355-365). Similarly, it is understood that the particular mould genera drive the health implications more so than the spore concentrations and building conditions (Garrett et al., 1998, pp. 459-467).

Fungal growth can be difficult to establish in indoor air pollution owing to collective toxins amassing within the indoor atmosphere, which can include dust mites, alternative allergens, chemical pollutants and other moisture-related microbes. However, mould can develop on or within porous materials that contain as little as 12–15% water concentration and, in some instances, even less when fungus-favoured relative humidity levels are about 85%. Hyphae, which are multicellular filamentous configurations, can propagate from spores that can then germinate further into intricate systems called mycelium. The fundamental cell wall of mould spores and hyphae comprise a fibrillary skeleton that contains a matrix of glucans (primarily β-1,3-glucan), enzymes, lipids, glycoproteins and galactomannans that are all fortified by chitin fibrils; it is important to note that this cell structure differs between genera and their associated life cycle. The casing of stationary spores generally contains a layer of hydrophobic RodA protein and pigments like melanin, which are obscured when growth and germination occur. This is when β-glucans and other polysaccharide moieties can be seen. The synthesis and excretion of mycotoxins and assorted proteases can occur

from particular genera of mould, as can the types and quantities of toxins and proteases. But again, the conditions and substrate strongly affect these processes (Holme et al., 2020, pp. 662-681). These mycotoxins are secondary metabolites that have been identified as creating inflammatory and toxic implications to health. Interestingly, the incubation and growth of spores can lead to a reduction of the shielding hydrophobin layer and, therefore, an augmented exposure of immunological exterior mechanisms (Øya et al., 2018, pp. 28–39).

Unfortunately, it seems the issue does not lie solely with minimising the favourable conditions for mould growth within buildings. The minimisation of building materials that can commonly contain dormant spores is vitally important. If spores are evident, they only need to be activated to begin the vicious cycle of active growth, not to mention the health implications of the spores themselves (Johansson et al., 2012, pp. 23–32).

4.5 THE WAY FORWARD

DEVELOPING STANDARDS

It seems often the case that it takes the accumulation of hundreds, even thousands, of unfortunate incidents for links to be investigated between toxins and possible health implications. Organisations and groups, such as Toxic Mould Support Australia (TMSA), aim to offer support and raise awareness of illnesses associated with mould and water-damaged buildings. As of November 2021, TMSA's Facebook page had over 9,500 members, many who seek support and advice, while other members offer that support.

A significant milestone towards the recognition and acknowledgement of illnesses associated with mould, water damage and other biotoxins was an inquiry undertaken by the Parliament of the Commonwealth of Australia, and its subsequent report, in 2018. A list of 7 recommendations concluded this inquiry; in summary, they are as follows:

a. The Department of Health should prepare and publish a fact sheet in the short-term, and commence additional research in the medium-term on:
    i. possible health implications resulting from mould and water damage exposure
    ii. the occurrence within the built environment of mould and water damage
    iii. recommendations on the prevention and remediation of mould.
b. The Australian Government, in conjunction with states and territories, should produce standards and/or accreditation requirements for the mould analysis and rectification industry.

c. The Australian Government, in conjunction with states and territories, should provide efficient rectification advice and full disclosure to tenants of public, social and community housing, aged care facilities and also tenants in rental housing, of any former or current water damage problems prior to entering any residential leasing contract.
d. The Australian Government, in conjunction with states and territories, should pursue additional research into the effectiveness of all relevant standards and building codes, specifically regarding prevention and rectification of water damage, mould and the like.
e. The Department of Health should undertake a review into the treatment of patients who present with complex symptoms and illnesses, such as CIRS, including a focus on whether efficient treatment and support is offered to patients and also whether medical practitioners require additional support or training.
f. The Australian Government should commission the National Health and Medical Research Council to undertake research into illnesses like CIRS to identify and assess links between biotoxins and symptoms and provide diagnosis and treatment to patients.
g. The Department of Health, in conjunction with health organisations, doctors and patient groups, should produce clinical strategies for GPs for the identification, treatment and management of biotoxins-associated illnesses (Parliament of the Commonwealth of Australia, 2018).

The ongoing accumulation of similar health cases, the publicising or recognition of these illnesses, and governmental inquiries are driving further investigations into building quality, building materials and health symptoms, which are intended to bring about improvements while reducing resultant harm to people. Following accumulating concerns expressed by industry professionals that the increase in air tightness and similar energy efficiency principles are causing a rise in condensation measures, new provisions have recently been included in the National Construction Code (NCC) 2019 to assist in reducing condensation and its consequences within houses and apartments specifically. These include buildings of Class 1, 2 and 4, with additional inclusions intended in the pending 2025 update of the NCC. Interestingly, since condensation can be caused by building occupation and functionality just as much as by the construction itself, these additional provisions aim to reduce condensation management and, in turn, the health implications, rather than the suppression of the moisture itself (Housing Industry Association (HIA), 2020). These provisions include:

a. Where a pliable building membrane (sarking) is installed to an external wall in climate zones 6–8 (which includes the ACT, Victoria, Tasmania, West Sydney, and some regional zones of

Western Australia and South Australia), the membrane must be permeable to vapour and located to the outside of the insulation.

b. Exhaust fans in the kitchen, bathrooms, toilets and/or laundries must be ducted to the exterior atmosphere or into a ventilated roof space.
c. The exhaust fan requirements have been amended to have a minimum flow rate of 25 L/s for bathrooms and toilets, and 40 L/s for laundries and kitchens.
d. Where exhaust fans are ducted into the roof space, ventilation must adhere to ensuring 30% of openings are within 900 mm of the highest point of the roof space (e.g. ridge) and the rest stipulated by eave vents. The roof pitch and respective ceiling area drives the total unhindered area of ventilation openings.
e. Kitchen exhausts may still be recirculating types within Class 1 buildings (houses) as they are not required to expel into a roof space. However, Class 2 buildings (apartments) and Class 4 parts of buildings (e.g. residence over a shop) must now be ducted externally to the atmosphere (Commonwealth of Australia and States and Territories of Australia 2019, 2019).

Remediation of affected buildings is critical, but prevention and control are paramount. In instances where mould growth is an existing problem, a risk assessment is required to identify the scope and/or spread of contamination, the necessary remediation actions, the explicit containment of the affected zones, and the protection of occupants and specialists involved or affected. An airtight enclosure surrounding the contaminated zone(s) should be erected to restrict spreading of contaminants. HEPA (high efficiency particulate air) filtration should assist air movement and the negative pressurisation within the restricted space. Decontamination methods vary based on the contractors and scope but should only be undertaken by qualified specialists. Contaminated materials should be removed from the space in sealed, vacuumed, impermeable bags for disposal. Assessments of clearance and ongoing monitoring are essential (Singh et al., 2011, pp. 37–41). It is recommended that the engaged mould remediation personnel are also trained in the relevant Institute of Inspection Cleaning and Restoration Certification (IICRC) standards, including the IICRC S500 series that focuses on mould contamination and water damage. These standards are based on research, practical experience and dependable restoration and remediation ideologies; ANSI/IICRC S520 specifically has been written by industry professionals including microbiologists, scientists, hygienists, remediation and restoration specialists and training schools to name a few (IIRCR - Institute of Inspection, 2019).

However, minimising the risk of mould growth is fundamental. It is paramount that the environment does not support such microbial growth from the onset and optimum indoor air quality

(IAQ) levels are maintained. Managing IAQ is critical in minimising moisture accumulation, and subsequently, mould growth. Some key principles to manage optimise IAQ include:

a. Practices such as *Passivhaus design strategies* that focus on an explicit level of mechanical ventilation and airtightness through thermal comfort and energy consumption. Though notably, the strategy does not clearly acknowledge the health of occupants. However, the suppression of mould is encouraged through ventilation strategies, focusing on high air exchange rates (Moreno-Rangel et al., 2020, pp. 1–16). While new constructions that are thoroughly and holistically built to the Passivhaus energy standard carry low risk for mould growth, dwellings that have been retrofitted or renovated to meet Passivhaus principles can often lead to an increase in air humidity. With Passivhaus practices focusing on five core factors – thermal bridge-free construction, utilisation of efficient windows and doors, super-insulation, airtight building envelope and also heat recovery ventilation systems (Moreno-Rangel et al., 2020, pp. 1–16) – these can be more difficult to implement and retrofit than in a new building, and therefore increasing the risk of insufficient practices, and consequently, compromised IAQ (Cotterell & Dadeby, 2012, pp. 195–196). Notably, Passivhaus practices can be juxtaposed to 'green buildings.' Green buildings typically focus on sustainable practices through the life cycle of the project, though certifications vary internationally and include Leadership in Energy and Environmental Design (LEED), Building Research Establishment Environmental Assessment Methodology (BREEAM), and even Green Star within Australia to name a few. With many focussing on the environmental impact of the building, IAQ can be compromised as a result of the use of recycled materials that may have contained toxic substances in their previous form, the lack of ventilation in maintaining air tightness and even the location of the building if in proximity to high-pollution or urban areas (Steinemann et al., 2017, pp. 351–358).
b. Contrastingly, the *WELL Building Standard* is a standard that focuses on the health and wellbeing of a building's inhabitants. The central principles aim to not only design and construct buildings that are healthy for the earth, but also its occupants. With IAQ being one of the critical factors, there is a focus on reducing mould and other microbial growth internally, especially from water damage and the condensation resultant to coils within the mechanical heating and cooling systems. To retain WELL Certification, and with specific regard to this particular chapter, the mould and microbial requirements include quarterly and recorded inspections of the cooling coils, cleaning when required, the installation and ongoing use of ultraviolet lamps with 254nm wavelengths to the coils and drain pan, and no

signs of water damage or microbial growth must exist (International WELL Building Institute, 2017, p. 33)

c. *Ventilation* of VOCs and other airbourne toxins and minimising indoor humidity will actually positively impact the IAQ (McGill et al., 2015, pp. 39-60). And further to the above in relation to green buildings, with climate change also coming to the forefront of current global challenges, a lack of ventilation can increase the relative humidity and condensation within internal environments (Sundell, 2010, pp. 185–186). Subsequently, monitoring and maintaining relative humidity levels indoors is also critical in maintaining conditions that will not support mould growth (Johansson et al., 2013, pp. 201-209).

ALTERNATIVES AND RECOMMENDATIONS

Mould growth is probable indoors when the associated relative humidity reaches a certain level, with a convincing relationship evident between fungal growth and internal moisture. This can therefore pose a significant problem to many building materials and, consequentially, their occupants, who spend the majority of their time indoors. Unfortunately, reducing the relative humidity through natural ventilation methods can negatively influence thermal and energy efficiency, so it is critical to understand alternative building materials that can alleviate both issues (Y. C. Chen et al., 2018). Some alternatives include:

a. *Silver nanoparticles (AgNPs) supported by titanium dioxide (TiO2)* have shown promising antifungal properties in a study that focused on gypsum board, softwood plywood and cement board across a 28-day period with a high 95% relative humidity level. The results were conditional to a number of factors, including the silver percentage level, the metallic level of the silver within the AgNPs and the thermal reduction temperature of the AgNPs/TiO2. It was discovered that permeating these building materials with AgNPs/TiO2 did not lead to any aesthetical variations to the materials, while identifying metallic nano-silver as a supreme carrier for antifungal silver ions (Y. C. Chen et al., 2018). However, given the high concentrations of metals applied to such building materials, it is suggested that further ongoing and long-term investigations are undertaken to ensure that the dispersal of metal particles into the environment is minimised, thereby minimising risk to human health.

b. The application of *natural biocides* has been found to offer antifungal properties to building materials; however, the origin and possible health consequences of such natural treatments should still be examined for toxicity. Given that many organisms have become resistant to many biocides, natural alkaloids are being assessed by researchers for their biological characteristics and potentiality – these alkaloids are a sizeable and structurally diverse

family of secondary metabolites that present varied biological properties owing to their molecular structure. A bisindole alkaloid has recently produced a reduction and prevention in fungal growth on some wall surfaces. This particular alkaloid is 2,2-bis(6-bromo-3-indolyl)ethylamine and is derived from marine sponge and tunicate (Campana et al., 2019, pp. 509-514).

c. While bamboo has become an increasingly popular alternative to traditional wood products owing to its sustainability, bamboo naturally is susceptible to fungal growth. The compound of *Chitosan-copper complex* (CCC®), which is understood to be a "low toxic preservative", and *propiconazole* have shown promising results of preventing mould growth of some species of bamboo (Sun et al., 2012, pp. 51-56). But again, any possible health implications of these compounds were not clearly identified and would require further research.
d. *Hemp* has been used in applications including cement mortar, particleboard, epoxy-based composites and even insulation. Some bio-based building materials containing hemp can offer mould resistance, as well as other properties varying from being cost-effective to manufacture, breathable, fire resistant, moisture resistant, pest resistant, durable and lightweight (Crini et al., 2020, pp. 1451-1476). With regard to insulation and wall systems specifically, timber construction can deliver thermally weak areas (or thermal bridges), especially where large cross-sections and mechanical joints occur, despite wood generally possessing reasonable thermal features. These thermal-varied zones within wall cavities can lead to temperature disparities. Consequentially, condensation within the wall system can lead to fungal growth and poor IAQ, with organic materials being especially susceptible (Brzyski et al., 2019, pp. 1-6). So, with reference to Crini et al.'s journal article noted above in this paragraph, hemp appears to be a favourable alternative to traditional organic and mould-prone building materials, from insulation through to textiles.
e. *Moisture-reducing practices and products* are critical in minimising condensation and other forms of mould-encouraging growth. These include low-condensation windows (e.g. glazing type and location), vapour barriers, sufficient waterproof roof systems including sarking and even appropriate ventilation systems (Perry, 2005b, p. 14).
f. *Reducing paper-lined materials* can offer much lower risks of mould growth when compared with the typical organic building materials used today, such as plasterboard, insulation, ceiling tiles, carpets and the like. Inorganic alternatives, like paperless plasterboard lined with fibreglass or insulation without a paper lining, do not provide the porous and moisture-prone surfaces that fungal growth strives on (Perry, 2005a, p. 10).

5. TOXIC SUBSTANCE TWO: FORMALDEHYDE

5.1 INTRODUCTION

The use of formaldehyde in building materials and interior furnishings is one of the primary contributors to poor IAQ (Huang et al., 2013, p. 542). Moreover, because of its reasonable thermal stability and high chemical reactivity, it is also commonly used in industrial production processes (Beheshtian et al., 2013, p. 1331). It is a colourless but combustible gas that is "highly reactive at room temperature" (WHO Regional Office for Europe, 2010). It can be found in timber composite products like particle board, plywood and panelling, and woven textiles including fabric and carpet (J. Lim et al., 2014, p. 4327). VOCs, with formaldehyde being one of the more prominent contributors, are regularly found not only in adhesives and primers used for the installation of such products, but also in the products themselves – for example the adhesives used to bind the layers of material within laminate flooring (J. Lim et al., 2014, p. 4328).

5.2 THE TOXIC SUBSTANCE

WHAT IS THE TOXIC SUBSTANCE?

Formaldehyde is considered an allergen, carcinogen and one of the most dangerous VOC toxic substances; it is also highly ubiquitous within indoor building materials owing to its cost-effectiveness compared with other comparable organic compounds (Nomura & Jones, 2014). It is considered one of the most significant commercial chemicals because of the versatility in industrial processes and cost efficiency (Kowatsch, 2010, p. 27).

The most reactive aldehyde is in fact formaldehyde, and it subsists at room temperature as a colourless gas. It was initially synthesised (in an exogenous form) in 1867 by August Wilhelm von Hofmann, a German scientist who recognised it formed by moving air and methanol over a heated platinum device. This followed a Russian chemist, Aleksandr Butlerov, initially synthesising the chemical earlier in 1859 (L. a. Zhang, 2018, p. 3) when he was attempting to formulate methylene glycol (Johnson, 2018, p. 1).

There are multiple compositions of formaldehyde. Formaldehyde, in the form of urea-formaldehyde (UF), is classed as one of the most important and pervasive formaldehyde emitters in interior spaces, and is highly prevalent in composite timber materials that have been bonded with a UF resin. With UF being the predominate formaldehyde polymer in plywood, particle board and panelling products, phenol formaldehyde (PF) can be been used as an adhesive within composite timber products also, as well fibreglass batt insulation, as it has a higher water tolerance than UF.

Fabrics, adhesives and laminate products can incorporate a formaldehyde-containing melamine resin, while methylene diphenyl diisocyanate is utilised within composite timber products, as well as other products requiring adhesion, and also as the feedstock in the manufacture of polyurethane plastics and foams (Healthy Building Network, 2021).

Formaldehyde can be manufactured in two ways – the silver catalyst process and the metal oxide process. Generally, with the silver catalyst process, air is combined with heated methanol, allowing vapour and steam to amalgamate. This concoction is then passed through a large heater to a reactor that contains either silver gauge layers or a layer of silver crystals. The fusion is then promptly cooled and passed to an absorption tower. The majority of the water, formaldehyde and methanol is compressed towards the bottom of the absorber where it is water-cooled. Much of the remaining formaldehyde and methanol is formed closer to the top of the tower with contact with clean water. Methanol is then recouped and recycled to the reactor, with the absorber bases heading for distillation. This distillation of steam is the aqueous solution of formaldehyde where formic acid is concentrated to the required specification. While this process is more cost-effective than the metal oxide process, the yield of formaldehyde is also lower (Kowatsch, 2010, p. 29).

The metal oxide process differs from the silver catalyst process by creating an amalgamated oxide, including molybdenum and iron, to create the catalytic oxidation of methanol. Recycled gas and air are combined with vaporised methanol, preheated to around 250 degrees Celsius and directed to a reactor. Tubes containing the iron oxide – molybdenum oxide catalyst allow the oxygen and methanol to react to formaldehyde. A ferric-molybdate catalyst is located on numerous fixed-bed reactors. The filtered air and vaporised methanol are sent to the reactors, where the tubes are encased in a scalding heat transfer liquid (e.g. diphenyl oxide) to vaporise the solution into a steam. This gas is just under 300 degrees Celsius and leaves the reactor before being cooled to about 130 degrees Celsius. It then goes into the absorber where a urea solution or water absorbs the formaldehyde. An ion exchange removes formic acid. A tail gas is incinerated – this is mostly oxygen and nitrogen, which can also contain formaldehyde, methanol, carbon monoxide and dimethyl ether in more minor quantities. This process does not involve a methanol tower like the silver catalyst process, and it is also easier to track contamination and does not require as many changes. UF solutions can be formed by substituting the water for urea for the quenching stage of the process (Kowatsch, 2010, pp. 29–31).

WHERE IS IT FOUND?

Formaldehyde is considered one of the most toxic and prevalent chemicals in today's industrial society, although notably it can also be produced naturally through the oxidation of hydrocarbons

and is produced within most genotypes (F. a. Pacheco-Torgal, 2012, p. 77). It is often a component of numerous building products and interior furnishings such as carpet, textiles, furniture, resins and timber composite products (Wang et al., 2012, p. 518), with over 65% of the manufacture of formaldehyde being accounted for within synthetic resins used in building materials (Li et al., 2019, p. 540). Reconstituted or composite wood panelling are renowned for their formaldehyde content. These types of products can include high density and medium-density boards, plywood and particleboard, which dominate in interior environments, for instance, in furniture structure or framing, joinery or cabinetry, and even panelling (Salem et al., 2012, p. 301). The chemical is also evident in paints, lacquers and resin-based papers or fabrics (Sheehan et al., 2018, p. 1129).

For the first three decades of the 1900s, resins consisting of formaldehyde were considered essential for adhering timber and its composites, and it was from the 1950's that particleboard and similar composite boards overran solid timber in the production of furniture and also in residential construction (Salthammer et al., 2010a, p. 2537).

Interior emissions of formaldehyde are most significant from flooring products and timber-based materials (Salem et al., 2012, p. 86). PF and UF in particular are most prevalent in wood-based products, within the adhesive additives, owing to satisfactory performance and cost efficiency (F. a. Pacheco-Torgal, 2012, p. 78). The versatility of the toxin has led to a staggering increase in demand, with formaldehyde being considered critical for the global economy. China has overtaken the US as the leading manufacturer of formaldehyde, with its production moving into the twenty-first century being a shocking 4000 times greater than it had been about 50 years prior. Although, not surprisingly, the subsequent contaminants have also increased, primarily occupationally (Wang et al., 2012, p. 518).

The below image demonstrates common locations in which formaldehyde may exist within a typical interior environment (refer to Image 5.1).

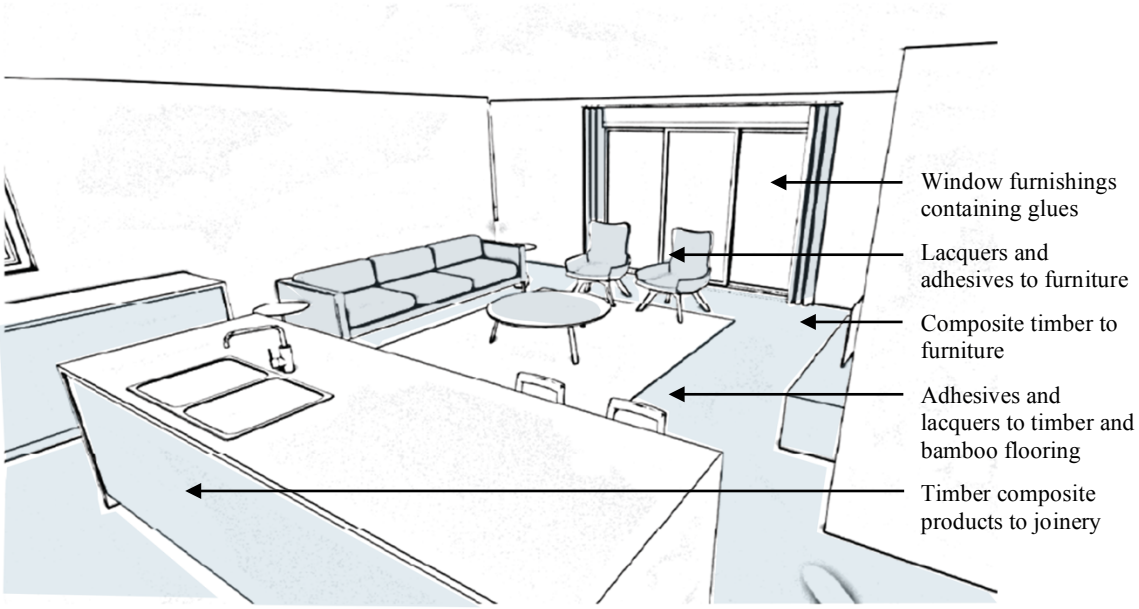

Image 5.1: Perspective of typical interior environment and prevalence of formaldehyde

5.3 HEALTH IMPLICATIONS OF THE TOXIC SUBSTANCE

HOW DOES EXPOSURE OCCUR?

While formaldehyde is pervasive in the surrounding environment in low levels naturally, it is understood that elevated exposure most commonly occurs during the manufacture of formaldehyde products, as well as from the use of formaldehyde-containing materials and through the burning of products that contain the toxin (Driscoll et al., 2015, p. 3).

Following the introduction of formaldehyde-resin building materials into housing in the mid-1900s, reports began to emerge in the mid-1960's indicating allergy-like symptoms, including respiratory complications and eye irritation, with the resin acknowledged as the cause. However, it was not until around 1980 that questions began to be raised about whether or not it was in fact a carcinogen (Salthammer et al., 2010a, p. 2537).

Studies have been conducted to assess the toxicity of formaldehyde vapour following exposure. It is understood that, for toxicity at distant bodily sites from the exposure point, the toxin must enter the bloodstream for transportation via the circulatory system. However, this is considered rare, especially from acute exposure, because when the toxin is inhaled through the nasal cavity it can generally be metabolised rapidly by the body (Franks, 2005, pp. 309–320). Absorption through

skin, primarily in the toxin's liquid form, is also an exposure source, which has commonly affected employees of the textile, resin and similar industries, as well as healthcare workers handling formaldehyde-preserved specimens (L. Zhang, 2018, pp. 20–38). Occupational exposure is believed to be the greatest via inhalation, with an increased susceptibility to hematopoietic diseases (Yuchao et al., 2013, p. 1).

VOCs originating from building materials can be absorbed through the skin and respiration, with formaldehyde being one of the contributors to VOCs (J. Lim et al., 2014, pp. 4326–4339). Skin absorption should be considered and researched more extensively to determine possible acute and chronic implications. Although, it should be noted that formaldehyde has not been considered carcinogenic orally, and it is not always bioavailable (Rovira et al., 2016, p. 361).

WHAT ARE THE HEALTH EFFECTS?

Background

Formaldehyde has been associated with a large number of symptoms and illnesses. The toxic substance has been linked to DNA damage owing to its reactions to nucleophilic materials (Bi et al., 2017, p. 36421). A review paper (Łebkowska et al., 2017) by the Warsaw University of Technology in Poland recently outlined that formaldehyde can lead to a number of illnesses, including reproductive insufficiencies, such as inflammation within the reproductive system; sterility; developmental issues; increased risk of miscarriage, menstrual diseases and birth defects; and cancers, including cancer of the brain, prostate, sinus, nasopharyngeal and even leukaemia (p. 54).

Interestingly, back in 1980, a research panel was established by the US Consumer Product Safety Commission to determine whether formaldehyde exposure could cause cancer in rat subjects. Initially, 3 of the rats developed squamous cell carcinoma in their nasal cavities, but this increased to 20% of the animals about 18 months into the 24 month study (Panel to assess formaldehyde health effects, 1980, p. 802). Significantly, Łebkowska et al. (2017) stated that 800 mg/kg is the lethal ingested dose of formaldehyde for rats, whereas the human comparitive is only 500 mg/kg, suggesting that formaldhyde would have an increased effect on humans (2017, p. 54).

In 2011, an independent report was released from the US National Research Council (NRC) of the National Academies to assess the Draft IRIS Assessment of Formaldehyde by the US EPA – the health effects of the toxin for their Integrated Risk Information System (IRIS). At that point in time, the US EPA had spent the previous 10 years or so re-evaluating the health implications of the toxic substance. The report from the NRC summarised that there was a level of ambiguity in their research methods. They stated,

> It lacks clear links to an underlying conceptual framework; and it does not contain sufficient documentation on methods and criteria for identifying evidence from epidemiologic and experimental studies, for critically evaluating individual studies, for assessing the weight of evidence, and for selecting studies for derivation of the [reference concentrations] and unit risk estimate. (National Research Council Committee to Review, 2011, p. 4).

The NRC concluded that the US EPA should review and revise their IRIS assessment on formaldehyde, focusing on the following recommendations:

1. Extensive editing is required to remove irrelevant material and discrepancies, while reporting in a more concise manner.
2. The methods must be more transparent and clearer, to greater support evidence for such conclusions, especially with regard to reference concentrations (RfCs).
3. Tables of evidence would be valuable to corroborate conclusions about all health issues raised.
4. A comprehensive evaluation is needed for critical studies to assess weaknesses and strengths using consistent methods.
5. The studies used to evaluate RfCs require clear validations for why they were selected, with risk of units also requiring identification.
6. The "weight of evidence" needs justification so that a reader can appreciate the factors that influenced their conclusions (National Research Council Committee to Review, 2011, p. 14).

However, at the time of writing this thesis, the US EPA website currently indicates that their formaldehyde Chemical Assessment Summary Non-cancer Assessment has not been updated since 1990, their Cancer Assessment has not been updated since 1989 and the IRIS Summary listed is based on referenced material from pre-1991, although primarily from the 1980's. A link to the EPA's Workshop on Formaldehyde is listed from April 2014; however, the website then indicates that the EPA is currently reassessing the IRIS of formaldehyde but the peer review draft and post final assessment have no scheduled dates advised to date (United States Environmental Protection Agency, 2017a).

An increase in investigations of the effects of formaldehyde specifically from emissions from building materials has occurred in the past few years. It is believed that the toxic substance is associated with allergic asthma and lower respiratory complications, dermal allergies, neurotoxicity, acute poisoning, pulmonary function impairments, possible carcinogenic complications and even SBS (Li et al., 2019).

Reproductive Issues

Within the last decade, there has been increasing trepidation regarding the ill effects that formaldehyde exposure can have on the reproductive system. Studies have shown that formaldehyde can lead to inherited gene mutation through paternal organs and can reduce spermatogenesis while also increasing apoptosis of spermatogenetic cells in testicular tissue (Wang et al., 2012, p. 518). Interestingly, a study of male mice in 2013 (Vosoughi et al.) aimed to assess a connection between formaldehyde exposure and 100 decibels of noise simultaneously, for 8 hours a day over 10 days. While the reported results of the study are rather vague and brief, the authors concluded that the levels of testosterone serum had significantly decreased in all subjects within the exposed subject group. Moreover, the authors found that simultaneous exposure to ongoing noise high levels *may* further escalate the "possibility" of reproductive damage within the male mice. This study sought to determine whether a relationship existed between exposure and noise, in association to humans in a typical workplace (p. 71). This study further questions how toxic substances might act when influenced by external factors, as opposed to studies focused on toxicity in isolation, of which future studies are vital.

Cancer

While acute exposure to formaldehyde has been linked to kidney failure, vomiting, irritation and even coma, chronic exposure to the substance has been associated with an increase in cancer occurrences, especially leukaemia and nasopharyngeal cancer (Hopkinson & Schofield, 2018, p. 904). In 2009, a study (Beane Freeman et al.) sought to confirm previous findings that advised exposure to formaldehyde does increase the risk to cancer. The authors concluded that there is a causal link between exposure to the substance and lymphohematopoietic malignancies including Hodgkin lymphoma and multiple myeloma. The former study that Beane Freeman et al. compared results to was from 1994 that advised relative risk of leukaemia and myeloid leukaemia increased for workers who worked in plants that either produced or utilised formaldehyde following exposure to the substance (Beane Freeman et al., 2009, pp. 151–161).

The National Cancer Institute led a study to assess the long-term effects of formaldehyde exposure and concluded that occupational exposure increased the likelihood of leukaemia in workers compared with people within limited exposure. The study assessed a variety of occupations, including manufacturing and industrial workplaces (Beane Freeman et al., 2009). Contrastingly, a team sought additional evidence following the IARC's "controversial" classification of formaldehyde being a cause of myeloid leukaemia and nasopharyngeal carcinoma. The team investigated just over 14,000 males who worked in six chemical factories across Wales and England. They discovered that, since their last investigation, over 2,000 additional deaths had

transpired, causally identified as the cancers listed below (refer Table 5.1), although they advised none of the tumours revealed a precise exposure-response connection. Despite the cases, the team outlined from their nested case-controlled assessments of 115 males with cancer of the upper respiratory tract that there were no elevated risks within the highest exposure category, and that their results provided no evidence for such cancers associations with formaldehyde. The team concluded that any elevated risks of these illnesses were "at most small," even from raised exposure (Ntani et al., 2014, pp. A3–A4).

Table 5.1: Actual deaths versus expected deaths following study of formaldehyde exposure and cancer

Disease	Expected Deaths	Actual Deaths
Oesophageal cancer	93.2	100
Stomach cancer	141.1	182
Rectum cancer	86.8	107
Liver cancer	26.9	35
Lung cancer	645.6	813

It is understood that that formaldehyde is primarily absorbed into the upper respiratory tract upon inhalation, and, owing to its swift metabolization, it does not commonly affect other bodily organs (F. a. Pacheco-Torgal, 2012, p. 82).

Respiratory Asthma and Allergies

Exposure to formaldehyde has been known to cause acute sinus and respiratory symptoms, including irritation of the throat, eyes and nose. Moreover, chronic exposure can be linked to asthma, with environmental exposure being more common in the US via items such as furniture, composite timber products and carpet than via occupational exposure such as in the case of embalmers (McGwin et al., 2010, p. 313). Despite respiratory and dermatological effects having been debated since at least the 1980's, the prevalence of formaldehyde in building products remains today (Nordman et al., 1985, p. 91).

5.4 LIFE CYCLE IMPLICATIONS OF THE TOXIC SUBSTANCE

Formaldehyde can affect society and the environment during various phases of its life cycle. Initially, occupational exposure can occur during the production of formaldehyde and UF solutions, as well as the subsequent manufacturing of associated building materials. A study that assessed 80 employees in Portugal in 2010, which comprised 30 people working in a formaldehyde-based resin

and formaldehyde plant and the remaining 50 employed in various anatomy and pathology laboratories, concluded that an increase in the frequency of micronucleus had occurred in epithelial cells and peripheral blood lymphocytes when compared with a control group with no exposure to the toxic substance (Nakano et al., 2018, pp. 1–8).

While the emissions of formaldehyde and its related bi-products present in building products are known to off-gas into the surrounding environment, it must be noted that formaldehyde is also a naturally occurring substance that can emit into the atmosphere. For example, formaldehyde emissions are a prevailing source of VOCs from vegetation, fires and also anthropogenic behaviours (Bauwens et al., 2016, pp. 10133–10134). However, importantly, the substance has commonly been shown to be at higher levels indoors (Lazenby et al., 2012, p. 966).

Life cycle assessments (LCAs) are a tool developed specifically to determine the environmental valuation of products, extending from their origin to waste (Widheden & Ringström, 2007, p. 695). An LCA (Iritani et al., 2015) focused on the process involved in furniture manufacture in Brazil, specifically of a wardrobe. The team concluded that toxicity, acidification and global warming were the highest rated categories affected, and that these were mostly contributed to by both the distribution of the product and the supply of the raw product. The wardrobe examined was constructed from composite timber products with adhesives formulated of synthetic UF resin (pp. 308–318). Similarly, a particleboard factory in Pakistan was assessed and it was found that one cubic metre of the material had 48% of its total emissions contributing to be greenhouse gas emissions. The emissions could be broken down into natural gas combustion, transport of raw material and also the UF manufacturing chain (Hussain et al., 2017, pp. 385–393). Remarkably, the study's focus was on a cradle-to-gate life cycle that ended with the distribution of the complete product – it did not assess the life cycle of the product while being utilised or once the product had concluded its purpose. Piekarski et al. (2017) conducted a similar study to that of Hussain et al. above, but of LCA of medium density fibreboard (MDF) in Brazil. The team's concluding recommendations were the reduction of various power consumption methods and transportation and also minimising or replacing the UF consumption, especially since Brazil is one of the highest yielding global manufacturers of MDF (Piekarski et al., 2017, pp. 103–111).

Studies have acknowledged the reuse of timber particles in new composite timber products, for example, recycling mill waste to create new materials (Azambuja et al., 2018, pp. 488–489). While this method aims to reduce waste, it does not address the ongoing health and environmental toxicity posed by formaldehyde. Redundant furniture has been used as raw materials to produce composite timber panels to recycle the original timber and reduce landfill. However, UF resin is one of the

catalysts added, along with ammonium sulphate and paraffin emulsion (Zamarian et al., 2018, pp. 1-8). Plywood that has been bonded with either post-industrial PF or UF can be ground down to produce particleboard too, although it is these formaldehyde resins that are listed as carcinogenic (Laskowska & Mamiński, 2018, pp. 427–435).

Studies have been undertaken to assess formaldehyde removal options from the surrounding atmosphere, given the toxicity and prevalence of formaldehyde. These include anaerobic and aerobic biological degradation, chemical oxidation and also carbon adsorption (Fulazzaky et al., 2013, p. 5100). A number of methods are also utilised to trace formaldehyde levels, although each has its own pros and cons, varying from cost efficiency to accuracy. However, it is suggested that nanomaterials could be the most useful in detecting formaldehyde from samples that include food, biological and environmental samples (Bi et al., 2017, p. 36430). It should be noted that little research seems to evaluate the risks of formaldehyde exposure as the life of a product reaches obsolescence or the product is recycled or reused.

5.5 THE WAY FORWARD

DEVELOPING STANDARDS

With formaldehyde a large contributor to poor IAQ, the South Korean Ministry of Land, Infrastructure, and Transport invoked requirements to reduce such toxic emissions through its Clean-Healthy House construction standard in December of 2010. It outlined the minimum standards that must be achieved to obtain the “Clean-Healthy House” classification, which the construction company must submit an evaluation report to the government body for approval. This has proved successful, with levels of formaldehyde and other substances all falling within the dictated limits, especially when compared with existing residences that were not part of the initiative. However, the Clean-Healthy House standard to date is only applied to multi-residential apartment complexes of more than 1,000 dwellings, and not smaller developments, commercial buildings or even standalone residences (H. Kim et al., 2017, pp. 633–639).

Initiatives such as these that do indicate positive results should be enacted by federal governments globally, but ultimately should also be applied to the multitude of indoor environments – all homes, regardless of the size, office tenancies and the like.

ALTERNATIVES AND RECOMMENDATIONS

Sources of formaldehyde in interior environments can be excessive and range from carpets, resilient flooring, furniture, textiles, composite paper products, composite timber products and even some

cleaners (L. Zhang, 2018). Therefore, it is imperative other options are sought. Some alternatives include:

a. *Lignon-epoxy resin* has been assessed for use as a formaldehyde-free alternative to adhesives or binders required in wood-based products. Lignon is believed to offer similar characteristics to formaldehyde-based resins, such as chemical stability, durability and water resistance. It is also a by-product of commercial processes and is therefore created in large quantities, providing the opportunity of up-cycling. But importantly, there are no known health implications of lignon. A team in 2018 (Li et al., 2018) further investigated the plausibility of such an alternative, with promising results. Performance tests proved comparable to formaldehyde-based properties, outlining that governmental mandates and improved manufacturing processes could result in a more efficient and cost-effective alternative (pp. 1459–1466).
b. Another adhesive alternative has been developed to minimise the use and emissions of formaldehyde. Melamine-urea-formaldehyde and UF are the primary formaldehyde-based adhesives currently utilised in wood composites products. With urgency raised during the last two decades to create an environmentally friendly, cost-effective and convenient alternative, a *cornflour and sodium hydroxide (NaOH)*–based adhesive has also displayed promising results. The highest performing results contained proportions of mimosa tannin/hexamine and cornflour/NaOH at 50:50. The adhesion of wood composite materials and associated characteristics was successful for indoor use, but also, importantly, the formaldehyde-based emissions from the cornflour alternative were minimal in comparison with the controlled UR particle board, excluding small levels of formaldehyde that were naturally derived from the wood itself (Moubarik et al., 2013, pp. 675–683).
c. *Soybeans* have been utilised in some plywood products since 2004 to minimise formaldehyde emissions. The beans are considered renewable, cost-effective and easy to obtain. Soy flour is the main component, but it has been used in conjunction with a curing agent – polyamidoamine-epichlorohydrin (PAE**)** – which is both petroleum-derived and expensive. Since the introduction of plywood products that used a soy and PAE-based adhesive, investigations have occurred to replace the PAE resin. Ammonium hydroxide and epichlorohydrin (ECH) have been produced to determine the plausibility to replace the PAE component, with ECH being primarily derived from renewable glycerol. When the curing agents were prepared at temperatures of 45–60 degrees Celsius, all the tested plywood products met the requirements for use internally. Subsequently, the addition of ECH and ammonium hydroxide simultaneously resulted in increased water resistance compared with

the addition of ECH and ammonium hydroxide in marginal portions intermittently (Jang et al., 2011, pp. 754–759).

d. With paints a contributor of formaldehyde emissions indoors, a *latex-based paint* was developed in 2015 as an alternative to traditional paints. It promised stain-resistant, formaldehyde-resistant and low-VOC properties. The product comprised latex, a coalescing agent, hydroxyethyl cellulose, a PH-conditioning agent, freeze-thaw stabiliser, a wetting agent, a dispersing agent, foam killer, titanium dioxide, bergmeal, calcium carbonate, mould inhibitor, disinfectant, rheological additive and water (Global IP News, 2015).

6. TOXIC SUBSTANCE THREE: PHTHALATES

6.1 INTRODUCTION

Phthalates are a group of synthetic chemical compounds commonly utilised to enhance features of plastics, solvents and even skincare products. The diethyl ester of phthalic acid (DEP), in particular, can be used as a carrier compound for artificial fragrances, especially in skincare (Api, 2001, p. 98), in which its identity can be obscured by simply listing it on the ingredients list as “fragrance”. It is also used to lubricate other ingredients. In plastics, including interior furnishings and building materials, phthalates are often added to increase malleability and flexibility in the product (Wynters & Goldberg, 2012, pp. 137–138). As an example, the formulation of polymers such as PVC with a plasticiser like phthalates creates a more malleable alternative to standard PVC (Huang et al., 2011, pp. 90-94). Phthalates can also be combined with other resins to then form a final plastic compound (Roy et al., 2017, p. 129). In addition, they can be added to synthetic rubbers and nitrocellulose, in proportions greater than 40%, for use in food packaging (Luch, 2012, p. 88).

6.2 THE TOXIC SUBSTANCE

Phthalates were developed as a result of technological advancements in the plastic industry. In 1846, cellulose nitrate was first produced as a plastic. This foundation then encouraged the patent of castor oil subsequently being incorporated as the primary plasticiser from 1856. However, castor oil became redundant as camphor grew as the preferred plasticiser for cellulose nitrate (International Agency for Research on Cancer, 1989, p. 337 (IARC)). Plasticisers continued to evolve, with phthalates being produced from the 1920’s, with one of the more prevalent forms, DEHP, revolutionising the plastic industry from the 1930's, (Posnack, 2014) owing to its versatility (Pigment & Resin Technology, 1972, pp. 21–22). This became available in commercial quantities in Japan in 1933, and in the US from 1939 (IARC Working Group on the Evaluation of Carcinogenic Risk to Humans, 2013, pp. 149–284). Resilient flooring was predominantly linoleum until the 1950’s; however, decreasing petrochemical costs and technological advancements quickly proved vinyl alternatives a more common option from the 1960’s in healthcare environments. While linoleum had many advantages over vinyl products, it was typically 200–300% more expensive to purchase than vinyl, hence the change in popularity (Scott, 2009, p. 15).

Phthalates are formulated from phthalic acid (Pacheco-Torgal & Jalali, 2011, p. 282) and commonly used as modifiers or additives. They are grouped into two classifications – high molecular weight (HMW) and low molecular weight (LMW) phthalates – based on their molecular composition. It is understood that HMW phthalates pose minimal concern to society in their current

functions, and it is in fact the LMW phthalates that have been categorised as ‘substances of very high concern’ under the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) classifications (Content, 2013, p. 48). The most common phthalate compounds are shown below in Table 6.1.

Given the prominence of PVC products in the interior built environment, this chapter primarily focuses on the integration of DEHP into the manufacturing process of PVC products.

PVC is formulated primarily from crude oil and chlorine (Pacheco Torgal, 2011, p. 21). The process begins with the manufacture of ethylene dichloride as the first step, which includes the cracking of ethane gas to form ethylene. Chlorine is obtained from the electrolysis of a brine solution (salt and water), and the ethylene derivative from hydrocarbon raw materials. These two elements react to form ethylene dichloride: *Ethylene + Chlorine = Ethylene Dichloride.* The second step involves the further cracking of the solution to form vinyl chloride monomer gas, which then endures polymerisation to create vinyl resin. The product of ethylene dichloride endures high temperatures in a reactor or furnace for decomposition: *Ethylene Dichloride = Hydrogen Chloride + Vinyl Chloride Monomer.* Oxychlorination then occurs when, in the presence of oxygen, hydrogen chloride is combined with more ethylene to produce auxiliary ethylene dichloride. Decomposition of the subsequent ethylene dichloride occurs, with oxychlorination reoccurring with the return of hydrogen chloride. This process can be summarised as: *Ethylene + Chlorine + Oxygen = Vinyl Chloride Monomer + Water*. In the third step, the polymerisation of the vinyl chloride monomer (VCM) creates PVC when the VCM’s double bonds are untied and the adjacent molecules combine to form long chain molecules (World of Chemicals, 2021). The integral chlorine component of vinyl provides the material with innate flame retardant characteristics, which unfortunately cannot be removed from the material without it compromising the construction and integrity of the vinyl. This is why vinyl is nowadays used for electrical wiring, to reduce the likelihood and spread of fire. Modifiers or additives can also be combined with the vinyl chloride resin to then form the vinyl compound. One such additive at this point is phthalates, in particular DEHP, formulated from phthalic acid (Pacheco-Torgal & Jalali, 2011, p. 282).

DEHP, specifically, is commercially produced by the combining of phthalic anhydride with a surplus of 2-ethylhexanol, in conjunction with sulfuric acid, para-toluenesulfonic acid or an alternate acid catalyst. It is colourless, mostly odourless, and dissolves better within harsh liquids and oils than in water (Agency for Toxic Substances and Disease Registry, 2002). DEHP, and other plasticisers, are incorporated into the PVC manufacturing process to achieve its flexibility and

Table 6.1: The most common types of phthalates

Abbrev-iation	Name	Class	Intended Purpose(s)	Most Commonly Found	Toxicity
BBP MBzP MBuP	Butyl benzyl phthalate Mono benzyl phthalate	Low (BBP)	Polyvinyl chloride plasticiser, acrylic-based polymers, polyurethane and polysulfide	Vinyl flooring, faux leather, adhesives and sealants, food coverings	BBP is a known endocrine disrupter – has led to testicular, mammary and developmental toxicity in mice experiments (Chatterjee & Karlovsky, 2010, p. 62). BBP can be metabolised in the body, after ingestion, into MBzP and MBuP (M. G. Kim et al., 2017, p. S26)
DBP DiBP MBP MiBP	Di-n-Butyl phthalate Di-isobutyl phthalate Mono-n-butyl phthalate Mono-isobutyl phthalate	Low (DBP) (DiBP)	Polyvinyl alcohol (PVA), PVC plasticiser, rubber	Nail polish, food coverings, latex adhesives, interior furnishings, paint, textiles, selected dyes and sealants	DiBP and DBP can lead to necrotic cell death at high doses (Naarala & Korpi, 2009)
DEHP MEHP	Di-(2-ethylhexyl) phthalate Mono-(2-ethylhexyl) phthalate	Low (DEHP)	PVC plasticiser	Furniture, vinyl flooring products, medical supplies, footwear, clothing and raincoats	DEHP classified as a "probable" human carcinogen by US EPA
DEP MEP	Diethyl phthalate Monoethyl phthalate	Low	Fixative and solvent, plasticiser	Skincare and cosmetics, fragrance	Developmental and reproductive implications (Saravanabhavan et al., 2014)
DiDP	Di-isodecyl phthalate	High	PVC plasticiser	Automobile leather, vinyl flooring, electrical cords	Further research required for human health
DiNP	Di-isononyl phthalate	High	PVC plasticiser	Paint, lacquers, textiles, adhesives, rubber, toys, drinking straws, synthetic rubber products as a softener	Cancer in rats; further research required for human health
DnOP	Di-n-octyl phthalate	High	PVC plasticiser	Floor finishes, linings, garden hoses, carpet backing, furnishings, toys	Reproductive complications (Saillenfait et al., 2011)

Note. (Lowell Center for Sustainable Production, 2011, pp. 4–12, unless noted otherwise).

malleability (Erythropel et al., 2014, p. 9967). In addition to PVC and DEHP, plasticisers can also contain glass fibres, pigments, stabilisers and limestone. Other copolymers to PVC include vinyl acetate, propylene, vinylidene chloride, ethyl, 2-ethyl-hexyl acrylate, n-butyl and even resins of post-chlorinated PVC homopolymers (Organisation for Economic & Development, 2014).

When phthalates are incorporated into plastics, in particular PVC, this allows the long polyvinyl molecules to glide against each other. A polar polymer is one that can be measured as negative or positive. This relates to the characteristics whereby phthalates affect the plasticisation. For example, the interaction between the positively charged vinyl chain and polar centre of the phthalate molecule, typically within a carbon atom of a carbon-chlorine bond, is the initial incorporation stage. The presence of the plasticiser, combined with significant heat, forms the relationship between the plasticiser and the polymer. Once this is cooled, the relationship remains. Importantly, the plasticiser is not chemically bonded to the plastic, allowing phthalates to be released with further heating or extraction (Pacheco-Torgal & Jalali, 2011).

Interestingly, an article written almost 4 decades ago identified such esters being evident in humans, animals and the environment, but also that it was unknown how the phthalates entered the environment, if they were even toxic, and what portion of the substances were naturally occurring in comparison to synthetic phthalates (Graham, 1973, pp. 3–4). Though since then, the prominence of phthalate toxicants within interior environments has been linked to various building materials and interior finishes or furnishings, including the widespread PVC flooring types, and polishes and lacquers applied to furniture (Bornehag & Nanberg, 2010, p. 334), which unfortunately is all too common in many homes and workplaces today.

The below image demonstrates common locations in which phthalates may exist within a typical interior environment (refer to Image 6.1).

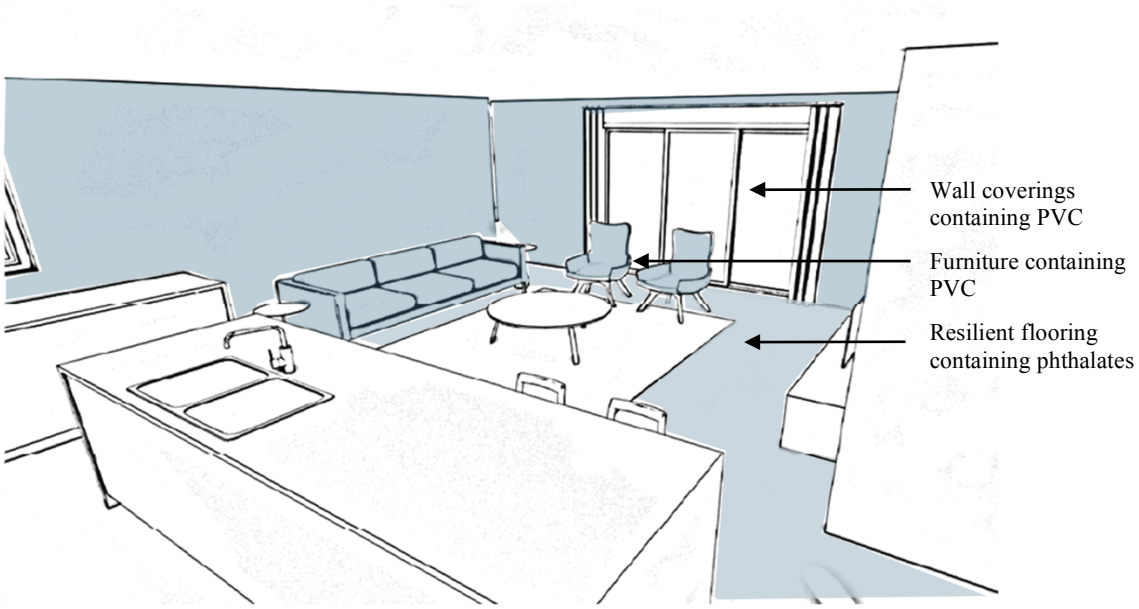

Image 6.1: Perspective of typical interior environment and prevalence of phthalates

6.3 HEALTH IMPLICATIONS OF THE TOXIC SUBSTANCE

HOW DOES EXPOSURE OCCUR?

Di(2-ethylhexyl)phthalate, or DEHP, in particular is known to exist in many different types of building products and interior furnishings or finishes. DEHP was listed by the US EPA as a "probable carcinogen" (Miao et al., 2017, pp. 312–320). However, in 2000, a statement was released by the Vinyl Institute indicating that the International Agency for Research on Cancer (IARC) had decided to overturn the previous probable carcinogenic classification, deeming DEHP as "not classifiable as a human carcinogen" (Burns, 2000, pp. 1–3), and subsequently supporting its use in vinyl medical products based on prevailing scientific evidence. Despite this, DEHP had more recently been re-classified as a probable human carcinogen by the US EPA and reasonably anticipated to be a human carcinogen by the National Toxicology Program, according to the Agency for Toxic Substances and Disease Registry (Agency for Toxic Substances and Disease Registry, 2011). Though curiously, this online source seems to have been dissolved since obtaining it during the course of this study in 2019, and a Toxicological Profile for DEHP report has since been released in December 2019 as a draft for comment. Interestingly, the 2019 report does depict the fluctuating views on the toxicity of DEHP. DEHP was classified as a Group B2 probable human carcinogen by the Integrated Risk Information System (IRIS) in 1988, carcinogenic to animals but with an unidentified association to humans by the American Conference of Governmental Industrial

Hygienists (ACGIH) in 2001 and 2016, realistically expected to be carcinogenic to humans the National Toxicology Program (NTP) in 2016, and possibly carcinogenic as a Group B2 in both 2013 and 2017 by IARC, with these agencies forming their conclusions from animal tests (Agency for Toxic Substances and Disease Registry, 2019, p. 10). No outcome of this report, following public comment, could be sourced and assumed to still be in progress at the time of writing this.

Importantly, it has been suggested that plasticisers can be found in corresponding polymers in proportions as high as 40% (Erythropel et al., 2014, p. 9967). Further, a more recent study conducted in 2018 suggested that DEHP has in fact been confidently linked to allergic diseases (Wang et al., 2018, p. 253).

Few studies have been conducted that associate exposure of phthalates to works during the manufacturing stages of the plasticisers. A study conducted many decades ago in 1973 assessed chronic exposure to occupational toxins of 147 employees. These employees operated as primers, mill and calendar operators, paint millers and mixing apparatus machinists. The study observed these employers, who were all under the age of 40 and had experienced exposure to the substance varying from six months to over 10 years. Health implications most reported included depression, or reduction in emotionality of the olfactory and vestibular receptors, and also toxic polyneuritis – numbness and pain associated mostly with the upper and lower extremities of the body. Significantly, concentrations of vinyl chloride, hydrogen chloride and carbon monoxide were confirmed *not* to exceed the maximum accepted levels (Milkov et al., 1973, pp. 175–178), begging the question – were the allowable levels of exposure to the substances in fact set too high for what was considered 'safe'? However, more recently, a study by Wang et al. (2015) was also conducted to analyse the possible health implications of 456 adults (all under the age of 45) operating within three different sized PVC production factories – 104 of the employees were office workers utilised as control workers for the study and the other 352 worked in the manufacturing departments. The workers were exposed to DEHP for up to nine hours per working day, for a median of three years. Blood tests were performed on each participant, one when just starting at the factory and the second after working there for three years. The blood tests assessed a number of plasmas, proteins and serums to determine a medical evidence base to assist in the study. Plasma cholinesterase, hepatic and renal biomarkers and residues resulted in convincing evidence that increased exposure to DEHP was in fact toxic to health, as did the difference in results between the office-based employees and the PVC production–based employees. The gastrointestinal or digestive, central nervous, respiratory and integumentary systems were the systems reported most as demonstrating the common clinical symptoms of exposure. These symptoms again included depression, muscle weakness and headaches, to name a few (pp. 37–44). Noting that over 40 years passed between these two studies

in particular, which both indicate similar findings, why is DEHP so prevalent still in the manufacture of PVC?

It is also understood that DEHP, along with other phthalate types, can affect the human genome primarily through dermal absorption, inhalation and ingestion (Lin et al., 2011, p. 217). However, because of its attention to the interior built environment, this study focused on those methods more prevalent in such scenarios – that is, the former two.

Phthalates can gradually emanate into the surrounding atmosphere throughout the life cycle of a product that contains DEHP, owing to phthalate not being chemically bound within its chemical structure (Clausen et al., 2010, p. 2760). SVOCs, in particular, can then tether to air particles and surrounding surfaces. Research-based ideologies of gas-particle partitioning continue to be investigated to determine how it affects the emission of such compounds during the particle and gas phases (Benning et al., 2013) – partitioning being the diffusion of a solute between molecules. DEHP has been studied to understand how emission of the toxicant into the surrounding atmosphere can be subject to external factors, which includes greater air flow (Clausen et al., 2010, pp. 2760–2766). Particulate matter, especially matter with a diameter of less than 2.5 micrometres, has been acknowledged by IARC as being one of the leading causes for lung cancer associated with airborne pollution (Martoni, 2018, pp. 240–251). DEHP and DBP have been found to be the predominate phthalate esters identifiable in such particulates indoors, contributing to exposure of phthalates via inhalation. Though importantly, it must be assessed how inhalation of these particular toxicants may then accumulate within the human body when other exposure routes occur (Zhang et al., 2019, pp. 1–10).

Phthalate diesters are considered highly lipophilic; that is, they have the ability to merge or dissolve into fats and lipids. Therefore, it may be assumed that the toxicants can be easily absorbed into the skin, especially with greater body temperatures (Alexander & Baxter, 2014, p. D44). It is understood that higher body temperatures can also increase the plausibility of the phthalate diesters being absorbed through the skin. One example, emerging from multiple studies, suggested this is the increased risk firefighters encounter, primarily thorough their fire-resistant protective clothing. Such clothing commonly contains phthalates, which can be dermally absorbed when body temperatures rise from the nature of the clothing itself, as well the prominence of higher temperatures associated to fires (Alexander, 2012). Importantly, the 2015 study by Wang et al., noted on the previous page, also discovered significant connections between increased levels of DEHP and human exposure to the higher temperatures of the PVC production plants (Wang et al., 2015, pp. 39–40). A published case study from 2014, also by Alexander, discussed how samples of

firefighter clothing were assessed for the presence of 26 particular chemicals. Surprisingly, at least one sample displayed the presence of 22 of the chemicals; moreover, DEHP was evident on all the samples. The amount of DEHP discovered ranged from 52 to 875 times more than the polycyclic aromatic hydrocarbons on the same samples. The study identified that the firefighter clothing samples included materials of kangaroo leather, which was noted as including low levels of other phthalates (Alexander & Baxter, 2014, pp. D43–D49); the question arises – do leathers used as interior furnishings also contain such phthalates? Further, why are phthalates used in such materials?

A prevalent example of phthalates incorporated into interior building finishes includes vinyl flooring products. DEHP has been known to exist in vinyl flooring products in proportions of 10–60% of the overall content of the product (Benning et al., 2013, pp. 2696–2703). Some vinyl floor products also contain at least one layer of foam to enhance under-foot comfort or softness. Cushion vinyl flooring, as well, incorporates the phthalate plasticiser DEHP (Bilitewski, 2013), and, as depicted in the table above (refer Table 6.1), this further suggests increased or additional exposure potential.

WHAT ARE THE HEALTH EFFECTS?

Studies are increasingly investigating the relationship between phthalate exposure and health concerns. For example, Benning et al. (2013) discussed previous studies that indicate exposure to DEHP can be linked to foetal and reproductive complications, allergies, asthma and neurological issues. Butyl benzyl phthalate (BBP), which can be found in vinyl flooring and PVC products, has direct links to endocrine disruption, affecting hormones (Chatterjee & Karlovsky, 2010, pp. 61–73). Specifically, women suffering from endometriosis have been found to contain higher levels of phthalates (Buck Louis et al., 2013, pp. 162–169).

Studies are also increasingly investigating the prevalence of asthma and allergies in association with phthalate exposure. DEHP, in particular, is the most prevalent of the phthalate types in indoor dust, as apparent from its existence in building products as well as children's toys and skincare products, with n-butyl benzyl phthalate (BBzP), diethyl phthalate (DEP), di-n-butyl phthalate and diisobutyk phthalate (DIBP) also evident (Bornehag & Nanberg, 2010, pp. 333–345). Studies compiled by Bornehag and Nanberg (2010) explained the bodily mechanisms that are affected by such substances, with direct relations to allergies and asthma. These changes in mechanisms can be an increase in concentrations of Th2-endorsed IgE and IgG1 immunoglobins, the differentiation between Th2, and the production of Th2 cytokine concentrations (p. 333). Exposure to DEHP and BBzP has also been linked to the increasing prevalence of asthma, with a greater increase evident in

Taiwanese communities' as a result of environmental catalysts and indoor settings. A particular study by Hsiu Ying et al. (2015), which was a progressive study conducted over nine years on Taiwanese societies, demonstrates that evidential and early exposure to these phthalates can influence the development of asthma and allergic sensitisation to children under eight years of age (pp. 1–14).

Phthalates are considered endocrine disruptors, having the ability to negatively impact hormones. Research has linked various phthalates to reproductive complications, developmental issues and neurotoxicity. DEHP has been investigated in relation to possible reproductive and developmental implications. Moreover, studies have confirmed that both Mono-(2-ethylhexyl) phthalate and DEHP can be transferred from a mother to her foetus and reduce the gestational period (Lin et al., 2011, pp. 245–251). It has also been noted that such toxicants may not display symptomatic complications until later in life, subsequent to toxicant exposure at any given point in time during foetal or infant development stages (Bornehag & Nanberg, 2010, pp. 333–334). Levels of Di-isodecyl phthalate/Di(2-propylheptyl) phthalate, DBP, DEHP and also BBzP have been detected in urine samples, which is concerning for children and pregnant women, A study (Shu et al., 2019) that explored the effect of phthalates from PVC flooring on pregnant women discovered new PVC flooring in tested preschools had elevated levels of DPHP, along with prominent metabolites of BBzP in homes. The study focused on Swedish homes, where PVC flooring is frequently utilised in bedrooms and bathrooms. While the European Union (EU) had prohibited the use of BBzP without permission back in 2015, the team raised concerns about the lengthy lifespan of typical PVC, being in excess of three decades, and that related products manufactured before the restrictions were implemented will continue to pose exposure dangers (pp. 43-54).

Neurological effects have been examined and linked to phthalates, in particular DEHP. Vision impairment, as a result of disruption of synaptic signals between laminar neurons and photoreceptors, have been studied subsequent to DEHP exposure (M.-Y. Chen et al., 2018, pp. 1558-1567). Rodent-based studies have also suggested that neonatal contact with DEHP can escalate motor activity, leading to neurotoxicity during development (Htet Aung et al., 2014, pp. 217-229). Mental illness is also believed to be influenced by phthalate exposure, with conditions including cognitive impairment and schizophrenia, as a result of endocrine disruption (Cho et al., 2010, p. 1027). An extensive study (Cho et al., 2010), of 667 children across nine schools in South Korea, investigated possible links between environmental phthalate exposure and the intellectual ability of school children. The study compared phthalate content in urine samples, examining the gender of the school children and verbal and full-scale IQ scores. Interestingly, the results indicated significant negative links between the children's vocabulary scores and the DBP and DEHP

metabolites in the associated urine samples. A negative connection between the vocabulary scores and MEHP-containing urine concentrations was evident for the male children. However, no significant connection was evident between the same variables and female children. This further clarifies a link between endocrine disruption, or hormones, and phthalate exposure, with "oestrogen-like effects" (pp. 1027–1032).

Cardiovascular health can also be affected by phthalate exposure. As a result of phthalates not being covalently bound to material such as PVC polymers, it is again evident that the substance can leach, especially when combined with lipophilic solutions such as blood. A team recently further investigated the link between cardiovascular health and plastics, with a focus specifically on the healthcare industry and medical supplies. The team concluded that phthalates do in fact affect the variability of heart rates and cardiovascular reactivity and also produce variations in autonomic regulation (Jaimes et al., 2017, pp. H1044–H1053).

In addition, dermal exposure is understood to occur through direct contact with the toxicant, particle deposition and even direct absorption from the surrounding atmosphere, which leaves the highest concentrations of phthalates discoverable on or around the hands, face and arms (Anderson & Meade, 2014). Notably, Wang et al. (2015) outlined that the most affected dermal exposure area of the body accounts for around 3,300 cm^2, which includes the legs. In addition, back in the 1970's, Milkov reported polyneuritis increased in the people studied, with amplified effects the longer the employees had worked in the PVC production plants under study; automic-sensory symptoms contributed to numbness and pain in the extremities or limbs (Milkov et al., 1973) – the same bodily areas reported to exploit dermal exposure.

Importantly, while studies have been undertaken to improve understanding of the health implications of exposure to both phthalate esters and xenoestrogens, limited studies have occurred that assess possible accumulated effects when such toxicants are combined (Chen et al., 2015, p. 518).

6.4 LIFE CYCLE IMPLICATIONS OF THE TOXIC SUBSTANCE

It is clear that research has been examining the health effects of exposure to phthalates the past few years especially, from simply using products that contain such substances, mostly building products, materials and furnishings. Numerous research studies have assessed the health implications of exposure to phthalates, in particular from DEHP, throughout various stages of the toxicants' life cycle processes. For example, Milkov et. al (1973) and Wang et. al (2015) discussed the toxicity of DEHP to workers throughout the manufacturing process of PVC.

However, with current sustainability pressures to recycle wherever possible and reduce landfill, the possible issue arises regarding how phthalates might affect a person's health post-use of these products. For instance, what happens if phthalate-containing materials, like vinyl flooring, are recycled and then reproduced into another building product? Are the health implications reduced? Does the chemical make-up of such new product affect the chemical bond of phthalates? Do they even still exist?

Phthalates can easily move throughout the environment, through processes such as production, transportation and disposals, owing to their unbound chemical nature with the originating material, for example, plastic or vinyl. As a consequence, the toxicants are often discoverable in water, air and soil throughout the environment, in addition to the bodily tissue, cells or plasma previously discussed. Photolysis and hydrolysis are the primary abiotic, or non-biological, processes that occur when phthalates are released into the environment, though the transportation of phthalates is affected by both chemical and physical variables within the vicinity – for example, in landfill (Whitacre, 2013, pp. 39–52). DEHP in particular has been listed by both the Chinese Environment Monitoring Centre and the US EPA as a water pollutant, and can be reduced within the environment through wastewater filtration processes as well as by aerobic biodegradation means; it is still one of the most concerning phthalate types polluting the environment globally (Gao & Wen, 2016, pp. 986–1001).

Similarly, a study published in 2011 (Wang et al.) assessed the possible relationship between workers exposed to DEHP within the environment and oxidative DNA damage. Over 300 people were assessed, with half being located near a plastic recycling centre (the exposure region) that had operated for two decades and the other half located 50 km away (the control region). All participants were gender and age matched. Soil and water samples of the exposure region were higher than those of the control region, with DEHP in the river water in particular being more than six times higher than in the control area. Significantly, water tested for DEHP in the well and pond were 215- and 18-fold of the control section, respectively. DBP, DnOP and DEP were also evident in the test results. The authors concluded that, while some of the urine substances discovered could have been due to other pollutants from the plastic recycling centre, oxidative DNA damage was evident (pp. S296–S297). This is concerning because a common perception abounds that recycling is a positive action for the future; however, is it actually as beneficial as it is intended to be – both for people who are involved in recycling processes and those who purchase building materials or products, in particular, with good intentions to contribute to sustainability? Their health may be affected by such products.

With respect to DEHP-containing products and the plausibility of recycling those for subsequent uses, in late 2015, the European Parliament passed a non-binding recommendation that forbade the recycling of plastic materials that contain DEHP. However, the recommendation was later overturned in April the following year by the European Commission's committee, who stated that these plastics could be recycled for the purpose of decreasing waste. It was also noted that the PVC plastic materials will mostly be used for future flooring and footwear (Stieger, 2016), leading to questions such as: Will the effects of the original phthalates be proportionally reduced at all? Will more phthalates be added to produce the new products? Will the new products be any healthier for users than the original products?

Pivnenko et al. (2016) noted that phthalate concentrations in recycled plastics had been theorised but were not yet well substantiated. Their study in 2016 assessed nominated phthalates within both virgin and recycled plastic materials. DEHP, DBP and DiBP resulted in the highest concentrations within their samples, with indicators revealing that phthalates were incorporated into the materials later in the production processes, and importantly, phthalates were not extricated from the recycled plastics and may consequently persist and bioaccumulate in the surrounding environment. The authors concluded that DEHP represented a possible gauge for phthalate contamination, advising that strict monitoring should be observed when utilising recycled plastics for new materials where phthalates were already of concern or even restricted, for example, in children's toys (pp. 44–52).

A report conducted in the Netherlands in 2016 confirmed that REACH had listed DEHP as a 'substance of very high concern' in 2008, and since then its production volume had been reduced. In 2015, the EU prohibited the use of DEHP in PVC without strict permission. The greatest concentration of DEHP in new products is restricted to 0.3% by REACH. Importantly, there are currently no commercially practical methods to remove phthalate from PVC (Janssen et al., 2016, pp. 36–38), which further contributes to concerns that building materials or products manufactured from recycled PVC continue to pose health risks.

An annual review compiled in 2010 (Halden), which analysed over 120 peer-reviewed publications on the health implications for people and animals of plastics and plasticisers, reached some remarkable and alarming conclusions: "The quantity of plastics produced worldwide in the first decade of this century is equivalent to the total world production in the century prior" (p. 189). The author further advised that both BPA and DEHP are of the greatest concern to health in the plastics industry. Most plastics are non-biodegradable and are unsustainable, and it was concluded that the previous 'reduce, reuse, recycle' statement should in fact be replaced with 'reduce, reuse, recycle, rethink, restrain,' in order to minimise plastic consumption where possible and, in turn, reduce

plastic production. Converting to a biodegradable and non-petroleum-based alternative would also likely be beneficial (Halden, 2010, pp. 179–194).

6.5 THE WAY FORWARD

DEVELOPING STANDARDS

At the turn of the twenty-first century, comparable regulations were endorsed by both the US Federal Government and the EU, owing to mounting apprehensions regarding a range of phthalates and their effects during pregnancy and for young children. Similarly, recommendations were made by the US Consumer Product Safety Commission to monitor and even ban some phthalates and their alternatives, particularly those that may affect children and women. DiDP, DiNP, di-2-propylheptyl (DPHP**),** dioctyl terephthalate (DOTP), cyclohexane-1,2-dicarboxylate (DINCH) and di(2-ethylhexyl) terephthalate (DEHTP) became alternatives for DEHP. Di-isobutyl phthalate (DiBP) became the alternative for DBP, until DiBP also became restricted (Shin et al., 2020, pp. 13157–13158).

In Australia, the *Competition and Consumer Act 2010* (Cth) prohibited the use of DEHP in children's products at percentages greater than 1%. The prohibition specifically included toys, childcare items and food containers and utensils, for children up to three years of age; but interestingly, the Act excluded footwear, clothing, sporting goods and second hand items (Commonwealth of Australia, 2011). These restricted items are assumed likely to be sucked by young children; only 40 minutes per day of exposure, especially directly in the mouth, carries a risk of toxicity to a child (Product Safety Australia).

ALTERNATIVES AND RECOMMENDATIONS

Lengthy evidence of embryonic stem cells of rodents identifies developmental detriments of foetuses, though further research into human health implications is required and must involve a multi-generational approach to assess long-term consequences of both human health and the environment (Dutta et al., 2020). It is vital that consideration is taken to minimise daily exposure levels of phthalate esters (Li et al., 2021, p. 14), but not just by building occupants. A broader and multifaceted approach is critical in reducing the use of DEHP and other phthalates. This needs to include interior designers and architects, building management, construction teams, and product developers (Dodson et al., 2017, pp. 114–127). Some alternatives to date include:

a. *DEHP-free plasticisers* can offer less-toxic alternatives to building products, bearing in mind that any alternatives should also be thoroughly investigated to ensure no long-term

toxicity similar to that of DEHP has been proven. An issue with DEHP-associated plastics is the degradation process; in turn, off-gassing, can increase with higher relative humidity. Castagnoli et al. (2019) outline that the Finnish protocol RT 14-10984 recommends that *if* plastic-like flooring materials are preferential by the client, they are installed onto the substrate when the relative humidity is less than 75%, although the alkalinity, temperature and other factors can also affect the extent of emissions. Screed between a concrete slab and the material can reduce rises in the pH that affect the flooring material. However, Castagnoli et al. strongly advocated to minimise all sources of moisture, temperature and humidity changes that can then also encourage microbial growth (for more information on this, see Chapter 4, Toxic Substance 1: Mould), which, consequentially, will also reduce the degradation of such materials. In addition, selecting low-emission materials will also assist with IAQ (Castagnoli et al., 2019, pp. 903–912). Similarly, Nalli et al. (2006) stated, "microbially mediated hydrolysis" of 2-ethylhexanol plasticisers will continue to influence air quality in many interior environments (pp. 181–185).

b. *Di(isononyl) cyclohexane-1,2-dicarboxylate (DINCH)* has no known ill effects on reproduction, nor has it caused carcinogenicity, acute toxicity or genotoxicity while being a potential substitute for DEHP, especially for use in hospitals, for example in blood bags. However, while DINCH-plasticised PVC is mechanically comparable to DEHP-PVC, it does not offer the same long-term integrity as the former DEHP alternative but carries less risk of leaching into the content within the bag (Lagerberg et al., 2015, pp. 522–531). Interestingly, a study (Fromme et al., 2016) outlined the presence of 4 DINCH metabolites in the urine of 208 children, with all children attending 1 of 27 investigated day care centres. While levels of DINCH were evident, it seems that the values were under the 'tolerable daily intake,' with exposure understood to be from inhalation of dust (pp. 33–39). Given the history of toxicity at times not being evident in the short-term, further investigation is required to ensure DINCH, or other non-DEHP plasticisers, do indeed carry little risk to society.
c. *Di (2-ethylhexyl) adipate (DEHA)* is utilised in many vinyl and plastic products as an alternative to DEHP since it has displayed less toxic consequences to DEHP but has a higher rate of leachability (Sheikh & Beg, 2019, p. 47). Xu et al. (2019) identified that studies that assessed the safety of DEHA as a substitute have primarily focused on oral exposure, that is, ingested, and aimed to assess intravenous exposure of DEHA in rat subjects. Despite some minor health changes in the subjects, the effects were considered reversible after two weeks (following a 28-day exposure period). That being said, the team concluded that systemic exposure is probable with the release of such plasticisers in items such as medical devices.

In addition, they noted that the IARC classifies the chemical as an IARC Group 3 – that is, it is not cogitated as a human carcinogen owing to *inadequate evidence* (pp. 50–55). This suggest that more research is required to assess the safety or toxicity of DEHA also. Further, DEHA can erode, much like DEHP, upon exposure to microbes and similarly can still release the 2-ethylhexanol (a VOC) when in the company of a carbon source (Nalli et al., 2006, pp. 182–183).

d. In terms of materials specifically, *bamboo* has proven a durable, sustainable and reasonable cost-effective alternative given its life span, with many options carrying 25-year warranties. However, caution should be undertaken when selecting bamboo materials since many include formaldehyde-containing adhesives. Bamboo materials that are free from formaldehyde will reduce possible VOC emissions (Wynters & Goldberg, 2012, pp. 245–246).
e. *Cork* and *linoleum* are other materials that can minimise exposure to phthalates. Linoleum, which was discussed '5.2 The Toxic Substance' as being a popular predecessor to vinyl until about the mid-1900s, when the popularity of vinyl soared in opposition, is a sustainably natural option that comprises flax-based linseed oil, wood flour, pine resin, limestone dust, cork powder, jute and mostly natural pigments. It is also anti-static, dust and dirt repellent, naturally antimicrobial and low in VOCs. However, it is not recommended for high moisture areas because of its natural porosity, nor in areas that may be subject to sensitive individuals (including children) because the linseed oil resins may be somewhat pungent (Wynters & Goldberg, 2012, p. 246).
f. *Tiles* remain a commonly utilised alternative to vinyl and are generally environmentally friendly. They are durable and easily maintainable, with infinite aesthetics available nowadays, and can be manufactured in the form of, or contain, porcelain, ceramic, stone and terrazzo, to name a few. However, only silicone sealants are recommended for interior spaces to reduce off-gassing of VOC's and other toxic substances (Wynters & Goldberg, 2012, p. 247).
g. *Polyolefin* has been used as an alternative material to PVC, primarily in the medical industry for blood bags. Though platelets and plasma from blood is adsorbed into the polyolefin. However, incorporating 2-methacryloyloxyethyl phosphorylcholine during the production of the polyolefin reduced the rate of adsorption (Yoon & Chung, 2020, pp. 319–326). Polyolefin textiles are considered an environmentally friendly substitute to traditional PVC products, including PVC-lined clothing, and does not require plasticisers for malleability (Community Research and Development Information Service).

7. TOXIC SUBSTANCE FOUR: POLYCHLORINATED BIPHENYLS

7.1 INTRODUCTION

A number of toxic substances exist that are ubiquitous within the surrounding environment but are also very difficult to degrade include PCBs, polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs; Hester & Harrison, 1996, p. 1). This thesis focuses on PCBs, owing to their prevalence in the surrounding environment, with a focus on interior spaces.

PCBs are a synthetic additive incorporated into products such as paint, electrical equipment and sealants, to name a few, mostly between 1950 and 1980 (Johnson & Wiley, 2011, p. 128). Aroclors are a commercial amalgam of PCBs that were produced in the US between 1929 and 1977. Other countries manufactured similar compounds, including Kanechlors in Japan, Fenclors in Italy, Clophens in Germany, Phenoclors in France and Soval in Russia. While the compounds significantly increased in the US in the early 1970's, restrictions on the production and utilisation of PCBs began to occur from the late 1970's. Importantly, owing to the properties of this toxicant, PCBs have persisted in the surrounding environment (Spengler et al., 2001, Section 36). Despite the occurrence of PCBs decreasing over the past few decades since their prohibition, they are still pervasive in the lithosphere, hydrosphere and atmosphere, with extensive global contamination having led to animal and human toxicity. Exposure can occur through the food supply, primarily via soil, water and air; however, they have also been found in buildings – especially those built during the second half of the twentieth century (Hansen et al., 1989).

7.2 THE TOXIC SUBSTANCE

PCBs are utilised for their minimal flammability, thermal stability, minimal vapour pressure when at ambient temperature, chemically inert manner and enduring ability to inhibit microbial degradation (Spengler et al., 2001, Section 36). They have been commercially produced since around 1929, with the first acute symptoms reported in 1936 from occupational exposure (Hansen & Robertson, 2001, pp. xi–xxx). However, the first chemical structurally similar to PCB was discovered in 1865 and was a derivative of coal tar (Risebrough & Brodine, 1970, pp. 16–26), although the first PCB is believed to have been amalgamated by German chemists in 1881, with large quantities introduced into the environment between then and 1914. The Swann Chemical Company in the US began to commercially produce PCBs from about 1929, with Monsanto Chemical Company attaining commercial production from 1935 (Silverstone et al., 2012). Monsanto, being the primary manufacturer, promoted the chemical under the trade name 'Aroclor' between 1930 and 1977 (Hansen & Robertson, 2001, p. xi). Between 1958 and 1971, more than 70

million kilograms of PCBs were retailed to be utilised specifically for sealants, adhesives, paint, caulk and ceiling tiles (D. Macintosh et al., 2012, pp. 1–10).

The decades following World War II saw a construction boom requiring concrete, plaster, paint and other building materials, which were in high demand. However, records of lubricant and paint formulas signify that Aroclors and other chlorobiphenyls and chlorinated diphenyls were utilised in such construction materials as plasticisers, similar to phthalates. While many of these toxic building materials were covered over time by more contemporary wall coverings and other finishes, demolition and renovations can expose these toxic substances once again, releasing them into the surrounding environment and atmosphere (Johnson & Wiley, 2011, pp. 128–133). Similarly, PCBs have recently become more concerning because of their use in elastic caulking for concrete, windows and doors (Broding et al., 2007, pp. 1427–1434). Their use to seal joints between these materials has increased the PCB contamination within some interior environments. A 2004 study of 24 buildings in Boston revealed that 13 of the buildings included PCBs at detectable levels, with eight of those 13 surpassing the US EPA benchmark of 50 ppm. These buildings were mostly public buildings, universities and schools. Interestingly, the US EPA requires building materials containing PCBs to be managed and disposed of when necessary; however, there are no protocols guiding how PCBs must be assessed in materials such as caulking (Herrick et al., 2004, pp. 1051–1053).

Another source of exposure to PCBs has been found to be fluorescent light ballasts. Four different education buildings – three campus buildings at the Indiana University (built in 1961, 1966 and 1980) and a fourth building at a Bloomington public school that was built in 1973 – were assessed for their possible levels of PCBs within the indoor environment. The team (Wallace et al., 1996) undertook the replacement of the PCB-contaminated ballasts in the subjected rooms in 1995, hoping the associated PCB levels would decrease over time, with their sampling occurring between 1987 and 1995. Importantly, the levels had not decreased when they tested the air three months after removing the contaminated lights; rather, they had increased. They hypothesised that perhaps the increase was due to other fluorescent light ballasts throughout the building contaminating the indoor air supply through the mechanical systems. Alternatively, evaporation of PCBs existent within interior building surfaces, including wall and ceiling finishes, could still be occurring (pp. 2730–2734).

The below image demonstrates common locations in which phthalates may exist within a typical interior environment (refer to Image 7.1).

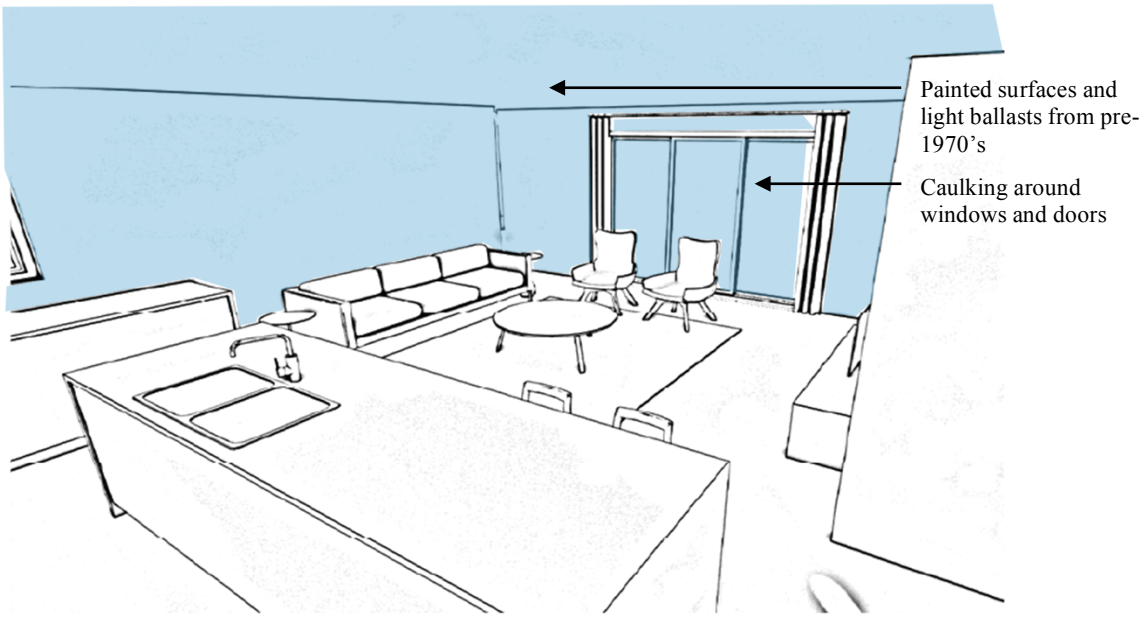

Image 7.1: Perspective of typical interior environment and prevalence of polychlorinated biphenyls

7.3 HEALTH IMPLICATIONS OF THE TOXIC SUBSTANCE

HOW DOES EXPOSURE OCCUR?

Exposure to PCBs has been found to occur in a number of ways. While some exposure can occur through diet as a result of the PCBs that have found their way into the surrounding environment, exposure can occur in percentages of up to 63% of the total PCBs for adults and 36% for young children within the interior environment owing to their slow release into the air (Bräuner et al., 2016, pp. 1–10). The toxicants can be inhaled through dust and vapour and possibly absorbed from contaminated surfaces, although evidence on dermal absorption are not widely published. That said, while the US EPA's accepted levels of PCBs that are under 50 ppm, a university building exhibited much higher levels internally, with the caulking itself reflecting levels of up to 33,000 ppm, window surround gaskets between 1.1 and 4,300 ppm, foam insulation below 310 ppm and parts of the ventilation system showing levels between 3.7 and 63 ppm (Herrick et al., 2004, p. 1051). Most of these would be considered inhalable sources.

Absorption has also been found to be a direct source of exposure to PCBs. Physical contact of such substances can infiltrate the skin through the dermis layer, with a primary contaminant of PCBs being domestic dust (Minzaghi et al., 2019, pp. 1–32).

Ingestion is a known bodily exposure route. During the years leading up to the prohibition of PCBs, the toxicants were detectable in adipose tissue and even breast milk, primarily in people living in more industrialised countries. Exposure was believed to have occurred from the consumption of freshwater fish and contaminated rice-based products, and also, employees utilising PCBs in industrial activities (Arnold et al., 1990, pp. 847–857).

WHAT ARE THE HEALTH EFFECTS?

Type II Diabetes

Type II diabetes has been associated with exposure to PCBs. A study of 774 people residing in Anniston, Alabama, resulted in 27% of the tested people being found to have diabetes, with 75% of those people already taking glycaemic control medication. Interestingly, a PCB production plant operated in the city from 1929 to 1971 that was bought from Monsanto Corporation in 1935. The PCB-containing pollution of the area affected sediment, soil and the air, with elevated PCB serum results in resident's blood still being reported within the last two decades (Silverstone et al., 2012, pp. 227–232). Similarly, exposure to PCBs has been found to increase the prevalence of diabetes in the wider population, just from elevated rates around higher PCB-polluted areas like chemical manufacturing plants (Persky et al., 2011, pp. 817–824).

Neurotoxicity

PCBs are a known neurotoxin (Shimada et al., 2010, pp. 271–286). While many studies (Goodman et al., 2010) have inconsistent conclusions linking neurodevelopment complications to the exposure of toxic substances, and subsequently not offering governing bodies sufficient evidence to regulate toxicants (pp. 727–734) like PCBs, other studies have found that neurological dysfunction can occur in people following occupational exposure (Seegal et al., 1986, pp. 197–202). PCBs have also been identified as influencing neurological conditions, such as attention deficit disorders, and exposure can be both prenatal and postnatal (Rosenquist et al., 2017, pp. 1–8).

Endocrine and Reproductive Health

A number of studies have assessed the effects on reproductive health consequential to exposure to PCBs. A study (Neblett et al., 2018) reported in the journal *Fertility and Sterility* in 2018 indicated that, while a reduction in lifetime pregnancies and births was correlated with PCB exposure, with

higher total serum counts, exposure was not connected to endometriosis, menstrual cycle abnormalities, uterine fibroids, ovarian cysts, pelvic inflammatory disease and other similar gynaecological conditions. Similarly, no connections were identified between exposure and pregnancy or development complications (p. e171). Although interestingly, a study conducted two years prior identified that triiodothyronine (T3) and reverse T3 (rT3) were influenced in the cord blood of newborn babies following exposure to PCBs (Soechitram et al., 2017, pp. 1117–1124). The toxicant is one of the most researched thyroid disrupters; it has an ability not only to affect the function of the thyroid directly, but also to disrupt the immune system primarily, which can in turn affect the thyroid indirectly. Employees exposed to PCBs in Slovakia displayed clinical symptoms of thyroid disease, following higher levels of thyroid antibodies and an increased thyroid volume (Benvenga et al., 2015, pp. 319–340).

PCBs have also been linked to the interference of the endocrine system and hormonal changes. A study in 2011 assessed the results of PCB exposure to rats via injections. The team discovered that hormonal pathways can affect the hypothalamus in the brain, leading to negative impacts on reproductive maturation and sexually dimorphic development within the neuroendocrine system. The latter, in particular, can lead to masculinisation of female species (Dickerson et al., 2011, pp. 581–594).

Despite this evidence, data gaps have been reported regarding the true impact of PCBs on one's health. Notably, there is a difference between how the toxicant has been found to affect animals when compared with people. Similarly, it was reported that commercially utilised PCB products can have a different composition from those later found within the environment (Cogliano, 2016, pp. 2212–2219).

7.4 LIFE CYCLE IMPLICATIONS OF THE TOXIC SUBSTANCE

The life cycle implications of PCBs appear to be most significant from the mid-1900s. Interestingly, concentrations of PCBs in indoor air have been recorded as the greatest in buildings from 1960, prior to the chemical ban, with respect to the study of the four buildings at Bloomington public school and Indiana University (Wallace et al., 1996), noted above. Buildings constructed prior to the ban in 1977 were identified as incorporating a number of electrical devices that contained PCB. The off-gassing of such materials, assumed to also include caulking, insulation and more, is estimated to wreak havoc for quite some time to come. Scientists have hypothesised that the slow decrease in PCB quantities in recent decades has perhaps not been as fast as hoped for following the ban owing to the possibility that some surrounding interior finishes or furnishings have absorbed airborne PCB particles, as well as ongoing emissions from PCB-containing interior products. Sadly,

many of these products cannot be replaced because of the expense associated with the widespread applications of the associated products (Wallace et al., 1996, pp. 2730–2734).

PCB exposure may still also occur, unrelated to the built environment, through the food chain. Animals are fed foods that can contain tenacious PCBs (Carpenter, 2013, pp. 1–7), enabling the PCBs to circle into our own food in a vicious cycle.

Closed applications of PCBs exist within hydraulic oils, transformers and capacitors. Germany contains around 60,000 tonnes of PCBs in this format, although PCB oils for these closed applications have been banned since 2000. However, open applications may include sealants (fire retardants and plasticisers, namely), paints and other building materials, which, significantly, have not been banned, and more than 12,000 tonnes are approximated to exist still in buildings, as of 2013. Emissions of these are estimated to be between about 7 and 12 tonnes per year into the environment. Nonetheless, it should be noted that management systems to minimise existing and future exposure are still inadequate, especially for the built environment. To minimise PCB exposure in foods, some recommendations include the following:

- creating inventories of contaminated sites and assessing plausibility of remediation
- creating inventories and management solutions of PCBs used in open applications
- developing management solutions specifically for end-of-life applications of PCBs, such as removal of coatings and paints, demolition of buildings or fitouts
- making recommendations on how to manage existing PCB contaminants in situ
- reducing and managing exposure to livestock and other animals
- assessing measures to analyse PCB amounts (Weber et al., 2018, pp. 16325–16343)

A study (Xing et al., 2011) investigated PCB levels in ambient air in residential zones of Taizhou, where a prominent transformer-recycling plant is located. Significantly elevated levels of PCBs were evident in the dust and air within the e-waste recycling plants. While dietary exposure and dust ingestion are generally considered the paramount exposure route, it is believed that the high levels of the toxic substance in the air in turn affect agriculture crops intended for human consumption. Notably, the samples were collected in 2006 and 2007, and there had been a history of recycling capacitors and transformers at the e-waste recycling plant for the two decades prior (pp. 605–611).

Interestingly, in the last few years it has been understood that Germany alone utilised 60,000 tonnes of PCBs in machinery – such as capacitors, transformers and hydraulic oils – although “closed applications” were banned in 2000 in Germany. Between 30 and 50% of these were considered

insufficiently managed. As of 2013, around 12,000 tonnes of PCBs are believed to be utilised still in open applications in building materials and construction elements in West Germany, since these are yet to be banned (Weber et al., 2018, p. 16325). Contrastingly, in late 1995 the state and federal governments of Australia recommended a management plan for PCBs, aiming for the safe removal of scheduled PCBs within 13 years. The plan concluded with recommendations for a follow-up review within the following five-year period that focused on sediment and sewerage contamination, further studies of the toxicant in breast milk, assessment of the toxicant in food items, analysis of landfill and monitoring of wildlife (National Advisory Body on Scheduled Waste, 1998). Interestingly, there was no specific recommendation relating to PCB management in pre-existing building materials. At the time of writing this, the follow-up report could not be found.

7.5 THE WAY FORWARD

DEVELOPING STANDARDS

Many countries appear to have legislation in place to manage and monitor the use and disposal of PCBs. The EPA has the authority to report, record, test and prohibit certain substances, including PCBs, which is government by the *Toxic Substances Control Act of 1976* (United States Environmental Protection Agency, 2020). Although, as noted above, most regulations and recommendations appear primarily focused on the overall exposure of PCBs within the environment as well as via food consumption. Advice relating to building materials, especially the exposure and disposal of effected materials from before their prohibition seem deficient. It is recommended that awareness and advice, especially in accessible forms by the general public, be further developed as a priority.

ALTERNATIVES AND RECOMMENDATIONS

Since PCBs primarily exist in existing applications from prior to the prohibitions of the toxic substances, methods to minimise exposure to PCBs need to be focussed management systems. These approaches include:

a. Correct *removal and disposal of contaminated* materials is critical. Construction waste from buildings constructed between 1950 and 1970 can be contaminated by PCBs in conjunction with the building materials themselves in situ. Paints, coatings, sealants, textiles, flooring and the like that contain PCBs must be removed appropriately and according to the recommended local regulations to reduce any impact of the toxicant being released into the environment. Abrasive removal processes, such as sand blasting, must be restricted. Similarly, the reuse of certain building materials in applications like landscaping can also

lead to widespread contamination, and further subsequent exposure through the life cycle processes. While closed applications of PCBs are managed through national inventories on a country-by-country basis, it is acknowledged that this level of monitoring is deficient for open applications owing to limited recommendations or governing advice (Weber et al., 2018, pp. 16325–16343), and it is a vital requirement globally.

b. While *minimising disturbances to existing PCB-containing materials* seems valid, studies have shown that the toxicant has the ability to disperse. The connection between PCB-containing materials and the interior environment, as well as soil surrounding buildings, suggests the toxicant can leach and be transported through routes that may include dissolving organic matter (Herrick et al., 2007). The US EPA compiled initial recommendations directed towards managers of schools and similar facilities in 2009, and a subsequent update three years later. The recommendations include the classification of either "PCB remediation waste" (products produced with PCBS of 50 ppm or above) or "PCB bulk product waste" (PCBs that are no longer associated with their primary source). The advice then includes recommendations for removal and disposal of the toxicants. Although interestingly, enduring PCB levels from the remediation are permitted to remain in the space, with the governing amount determined by the occupancy rate, bulk quantities in comparison to surface loading quantities, and whether the land use is unrestricted or governed by a deed (Brown et al., 2016, pp. 1986–1997). Given the evident toxicity of the PCBs, it would be important that these recommendations are revised with greater rigour to minimise exposure – especially to those most at risk, like children in schools.

c. Owing to the pre-existing nature and prevalence of PCBs within buildings since their prohibition decades ago, the *correct treatment* of affected building materials can offer risk abatement that can be more cost-effective than demolition and disposal practices. Assembling a physical barrier around the toxic materials, including encompassing caulk, can reduce exposure to PCBs while also restricting physical contact with the materials. Similarly, providing a sufficient source or method of fresh air ventilation can also assist. However, remarkably, short-term measurements do not necessarily offer accurate readings of PCB contaminants within an interior space since ambient temperature can affect the levels. It is recommended that assessments are carried out over a longer duration to offer more insightful results. An example (D. L. MacIntosh et al., 2012) of managing existing PCB-containing materials insitu was conducted by encapsulating the caulked materials. The installation of a false wall acted as a barrier between occupants and the affected materials. The false wall incorporated fibreglass insulation board that had aluminium backing, which was installed over the internal side of a transite panel and subsequently sealed with silicone-

based caulk. Gypsum sheeting was then applied over this and the corresponding aluminium stud framing; this too was sealed with silicon caulk and then painted with a latex-based treatment (D. L. MacIntosh et al., 2012, p. 24).

d. Alternatively, *chemical remediation* offers a valuable option for existing materials contaminated with PCBs. While paint is not considered one of the most toxic materials that can contain PCBs, it is the extensive surface area related to paint and its ability to leach the toxicant into the surrounding space that is of concern. A team in Canada (Saitta et al., 2015) assessed the plausibility of utilising a non-metal treatment system (NMTS) and a bimetallic treatment system (BTS) to remediate PCB levels on industrial surfaces, including concrete walls. PCB levels were reduced by between 60 to 97% on painted metal surfaces and by 95% on painted concrete surfaces. Further, an activated metal treatment system was then applied after treatment, which displayed a further reduction by 82% for the BTS tests and 99% for the NMTS tests. The team concluded that this method offered a non-destructive alternative that alleviated the need for expensive removal and disposal of such building materials (pp. 40–47).

8. TOXIC SUBSTANCE FIVE: POLYBROMINATED DIPHENYL ETHERS

8.1 INTRODUCTION

PBDEs are a type of brominated flame retardant (BFR**) –** an assorted family of organic compounds used in a large variety of building materials and interior furnishings to reduce their flammability. These can include carpet, textiles and upholstery, furniture foam and padding, insulators and electrical devices, to name a few (Besis et al., 2017, pp. 871–881). Similarly, they can also be found in polyurethane foams, such as mattresses, and also computer and television surrounds (Y. Lim et al., 2014). They have been widely employed since the 1970's, with some forms prohibited over the last few decades owing to increasing concerns about their impact on human health and the environment (Office of Land and Emergency Management, 2017). While prohibitions and restrictions now apply to some PBDEs, there are many products still on the market or in existence that contain PBDEs, causing health and environmental concerns (Li et al., 2015, p. 25).

8.2 THE TOXIC SUBSTANCE

BFRs have accounted for a large proportion of flame retardants on the market. The utilisation of flame retardants in Korea was increasing by approximately 10% per year between 1997 and 2007. The use of decabrominated diphenyl ethers (decaBDE) a commercial mixture of PBDEs, in Korea occupied almost 50% of the decaBDE in Asia at the start of the 21st first century (Moon et al., 2007, pp. 1402–1412). Their popularity rose because of their cost-effectiveness, and also their ability to withstand extreme temperatures when compared with inorganic, organophosphorus and nitrogen-comprising fire retardants. Fortunately, the BDE industry has decreased since then, following an increase in health and environmental concerns and the consequential regulation of some types (Rahman et al., 2001, pp. 1–17).

Structurally, BDEs are similar to PCBs. They are hydrophobic and fat-soluble (Office of Land and Emergency Management, 2017), although, unfortunately, they are not bonded to the polymetric matrix or structure, which means they can leach out of the products into which they had been integrated. Even through everyday use of the product, they can leach into the surrounding environment (Gevao et al., 2016, pp. 106–113).

Pentabromodiphenyl ether (PentaBDE) and octabromodiphenyl (octaBDE) are two other mixtures of the toxicant, and along with decaBDE are the three prevalent commercial forms of PBDE products. However, these three types can contain more than 200 possible BDE chemical constitutes.

These constitutes belong in 1 of 10 groups, from mono-BDE through to deca-BDE. PentaBDE and octaBDE became regulated and classified as persistent organic pollutants (POPs) in 2009 and hexabromocyclododecanes in 2013 (Lee et al., 2015, pp. 18–25). However, it is the tetraBDE, pentaBDE and hexaBDE constitutes that can be the most ubiquitous in human tissue, constituting about 90% of total body toxicity (Y. Lim et al., 2014, pp. 1376–1389). Interestingly, penta-BDE is mostly utilised in polyurethane foam, like in furniture, whereas octaBDE is mostly used in smaller appliances and some other electronic equipment. DecaBDE is the most commonly utilised form nowadays globally, and is prevalent in the backs of textiles and carpets, plastics, electronic surrounds and wire insulation. (Gevao et al., 2016, pp. 106–113). Significantly, for the three decades that followed 1970, it is estimated that between 3,000 and 5,000 tonnes of pentaBDE were manufactured throughout Europe, with an additional 9,000 to 10,000 tonnes imported through finished products. These figures could be broken down into approximately 30% usage in polyurethane foams in vehicles, 10% in furniture foam and the remainder in textiles, construction materials, packaging and solid applications (Prevedouros et al., 2004, pp. 3224–3231).

The below image demonstrates common locations in which phthalates may exist within a typical interior environment (refer to Image 8.1).

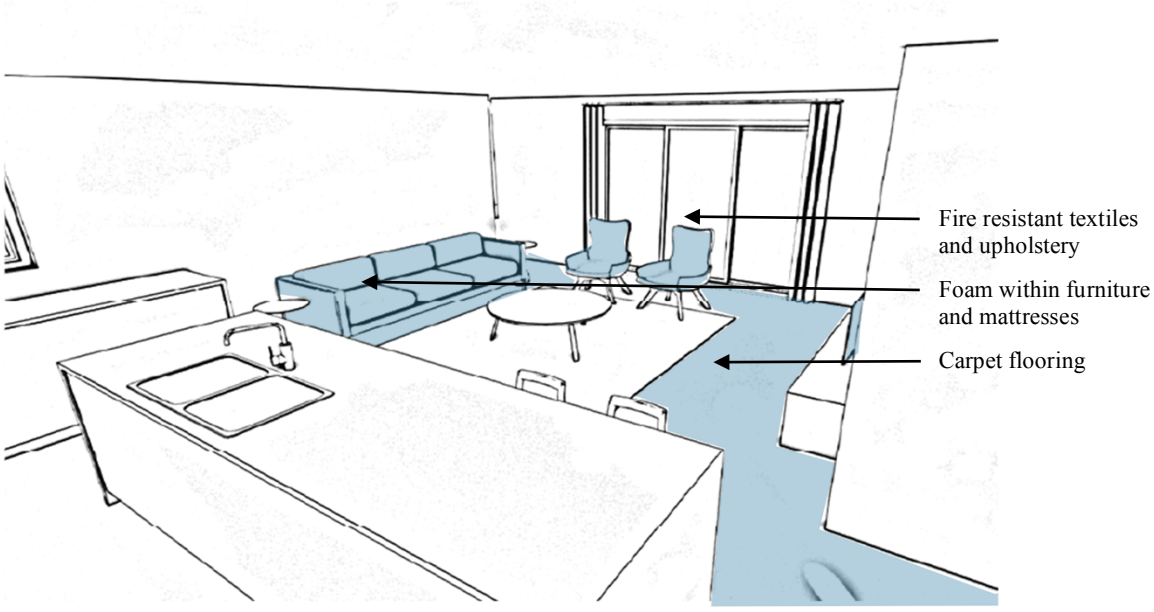

Image 8.1: Perspective of typical interior environment and prevalence of brominated diphenyl ethers

8.3 HEALTH IMPLICATIONS OF THE TOXIC SUBSTANCE

HOW DOES EXPOSURE OCCUR?

In 2007, previous reports of exposure to the toxicants via inhalation were limited. A team in Boston (Allen et al., 2007) sought to evaluate limitations in assessing personal exposure to PBDE that had existed until that time. Such limitations in question included, under-sampling of particulates owing to the reliance of passive sampling processes; underestimations of personal air sampling with the focus of samples being on areas; the assessments were primarily not conducted from within the US; and/or, did not evaluate BDE 209 concentrations within average homes. Allen et al. analysed 20 homes in Boston, Massachusetts, through three samples each – 1) area sample of the bedroom; 2) area sample of a secondary space that would be considered most inhabited during the day; and 3) personal sample, which was a hip-mounted pump device worn by the participant that had the sampling mechanism mounted to their collar within 300 mm of their breathing zone. Interestingly, the personal test exhibited higher results than did the two area tests, and especially regarding the degree of bromination, which had increased. It is understood that the "personal cloud", or the atmospheric space immediately surrounding a person, is heavily influenced by activity patterns of the individual. Other factors that can influence the difference between area and personal air are the size of the particulate, resuspension of dust and physical activity (e.g. dry dusting, walking and folding linen) when compared with more inactive areas within the interior environment. The team concluded with advice for future research to assess the PBDE levels of building contents such as furniture and electrical equipment in order to improve understanding of the relationship between consumer products and PBDE exposure in internal environments. It was also noted that, at that time, the health implications of PBDEs were not greatly understood, so the team were unable to empirically conclude which route of exposure was most critical (pp. 4574–4579).

During the seven years that followed that study, the leading BFR pathway to human exposure was further recognised to be through indoor dust, typically via inhalation and ingestion, with ingested exposure also reported from dietary means. Significantly, exposure from indoor dust has been reported to account for more than 80% of all PBDE intake across North America (Qi et al., 2014). BDE-209 are decabrominated diphenyl ethers that form the primary component of deca-BDE mixtures commercially, and while they were added to the list of POPs in 2017, numerous developing countries, plus China, still manufacture and utilise deca-BDEs. The wide use of these in electronic products as a flame retardant additive can lead to volatilisation or leaching into the surrounding environment (T. Chen et al., 2018, pp. 505–506).

Dust ingestion is considered the most dominant form of exposure to the toxicants within the United Kingdom (UK) and the US, owing to the prevalent existence of PBDEs within those countries. Following a study (Li et al., 2015) that assessed 9 characteristic office spaces in Shanghai, China, samples were collected of settled dust within personal computer cases, surface dust on workstations and floor dust, as well as air samples. PBDEs within the dust readings ranged from 247 to 3.3 x 10^4 ng/g, whereas the air samples varied from 93 to 322 pg/m^3. The team concluded that there are many factors that can affect the results, including the extent of electronic device usage, the air exchange and possible degradation, transport and partitioning techniques upon release, which may correspond to the difficulty in assessing the true health implications of PBDE exposure as the physical conditions in interior spaces can vary immensely (pp. 25–31).

More recently, dietary ingestion has been reported (Kim et al., 2016) to be the most critical contributor to exposure of all PBDEs. In 2016, the gross domestic product globally was discovered to relate to the levels of PBDEs in internal dust of houses. Kim et al.'s study aimed to evaluate PBDE exposure levels through house dust ingestion across various transnational communities represented by 22 countries located over five continents. Dietary ingestion was considered more significant than dust ingestion in all assessed countries except for the US and the UK. In the US, of the total PBDE intake, adults were exposed to 82% through house dust ingestion, with breast milk, umbilical cord and maternal plasma, human serum and hair displaying evidence of the toxicant. It is understood that higher brominated congeners can be leached into the environment through abrasion, and lower-brominated congeners through volatilisation, with relationships evident between human exposure and the quantity or extent of PBDE-containing items within interior environments. Significantly, and noting that Allen et al.'s 2007 study, mentioned above under *How Does Exposure Occur*, did not assess BDE-209, BDE-209 was the most prevalent congener discovered in dust samples from most of the countries assessed, with portions ranging between 31.8% and 99% of all the tested PBDEs of all countries. Since penta-BDE and octa-BDE had been voluntarily phased out from 2004 across many countries, the utilisation of deca-BDE was expected to increase (noting this too was to be phased out by 2014, as instigated by the EPA). Although, resulting levels of BDE-209 in the North American test subjects indicated that the prohibition of the congener was not evident until the late 2000's, which could be consequential to items still being used that were produced before the mid-2000's. Importantly, the team also noted that a reduced quantity of samples was manufactured between 2004 and 2008. However, the percentage of BDE-209 against the total PBDEs tested across the Asian and European subjects signified the prevalence of deca-BDE. It is likely the lack of regulation in some countries like Korea have led the higher results within house dust that the North American counterpart where the production and use of PBDE have levels of

restrictions imposed. To summarise, the results of BDE-209 in comparison with the total PBDE globally suggest worldwide human exposure to and contamination by PBDE (pp. 82–91).

While some academics believe that PBDE exposure primarily arises from non-dietary sources in levels of about 80% of all associated exposure, dietary ingestion has also been reported as a predominate pathway and, in some cases, as even more prevalent than dust ingestion and inhalation. For example, a study (Ni et al., 2012) conducted exploring total PBDEs in Shenzhen concluded that dietary and soil exposure accounted for 65% and 23%, respectively, with a greater prevalence of lower-brominated congeners. The dust samples in Shenzhen only exhibited 2% of total PBDEs, although they team noted that PBDE-containing dust samples were low in comparison to areas such as various cities within China, the US, the UK and Singapore. Conclusions were drawn that exposure to the toxicant is directly affected by regional and individual characteristics, such as lifestyle and the usage of such products (pp. 10–14).

WHAT ARE THE HEALTH EFFECTS?

Exposure to BDEs has been linked to various medical conditions and symptoms, including, hormonal thyroid dysfunction, developmental and cognitive deficiencies, behavioural changes, hearing abnormalities and potentially even cancer (Kumari et al., 2014, pp. 3001–3009). Some studies have been undertaken in the past in relation to thyroid implications following exposure to BFRs, although results have been both varied and limited. BDE-209 has recently been demonstrated to bioaccumulate, despite a previous belief that its minimal bioavailability, high hydrophobicity and large molecular size implied it would not. Further to its prevalence within indoor environments and dietary sources, thyroid assessments have proven difficult. A study revealed that triiodothyronine (T3) levels had considerably increased in rats that had been treated with BDE-209 compared with the control group. But contrastingly, two alternative studies demonstrated T3 and thyroxine (T4) levels had reduced in rats treated with the same toxicant, suggesting the homeostasis of thyroid hormones had been effected owing to the similarity in structure between those particular hormones and the toxicant. Results of previous human-based tests have also shown varied results, with tests to date focusing on either non-occupational exposure or on community groups in proximity to e-waste facilities associated with BDE-209. However, the e-waste facilities are also known to off-gas other toxic substances, including PCBs, so it is very difficult to determine the isolated effect of BDEs (T. Chen et al., 2018, pp. 505–515). This clearly indicates that further research is required quickly to determine the health implications that BDEs can have on humans, as well as the environment and other species. But importantly, the accumulated toxic effects of multiple toxic substances should not always be studied in isolation of other toxics substances since this is not generally a realistic situation.

Relationships between BDEs and foetal and development complications have been hypothesised. One particular study in 2011 (Stapleton et al.) outlined the possibility of PBDEs affecting thyroid hormones during pregnancy, especially during the first trimester when a foetus relies exclusively on the maternal hormones. However, the study concluded that more research was required (pp. 1454–1459). A more recent study Müller et al. (2016) focusing on BFR levels associated with pregnancy in Northern Tanzania displayed similar results to those within US reports, which are considered to have the highest levels globally. The size of newborn babies was reported to be negatively affected as a result to PBDE exposure, and further, 20% of the infants involved in the study reported PBDE levels above the RfD for toxicity associated with neurodevelopment. Exposure to the toxicants is understood to be primarily from the consumption of pemba – a clay-based mineral supplement obtained from local markets that is consumed to relieve pregnancy-associated nausea. It is the clay soil that evidently can contain high levels of PCDFs and PCDD, and an estimated 64% of pregnant Tanzanian women are believed to consume the product, with an associated increase in BDE 47, 99, 100 and 153 within their bodies (pp. 38–47).

While most studies seem to focus on the health implications associated with BDE exposure to rodents, with human-based research appearing limited, the toxicant can evidently can lead to cognitive complications. With PCBs structurally being similar to PBDEs, PCBs have been highlighted as a health concern for quite some time, whereas, unfortunately, there is less evidence available to date on the health implications of PBDE exposure. However, it is understood that both toxicants can traverse through the placenta and are shared with a newborn child through lactation. This process has led research to assess the evidential relationship between PBDE exposure and similar health issues from PCB exposure, which may include neurochemical insufficiencies, as well as motor, sensory and cognitive complications. In addition, thyroxine concentrations can reduce following exposure to PCBs, which is believed to be a cause of hearing loss associated with cochlear development. In a study in 2011 on cochlear function in rats following exposure to both PCBs and PBDEs, serum thyroxine concentrations had reduced less following PBDE exposure than following PCB exposure. Importantly, the study noted that an accumulated negative effect can result in the serum concentrations upon exposure to both toxicants (Poon et al., 2011, pp. 161–168).

Li et al.'s Shanghai workplace study mentioned above, under 'How Does Exposure Occur,' concluded that the inhalation and ingested dust mechanisms for PBDE exposure displayed a non-cancer risk, with PBDE quantities being below the acceptable threshold level (Li et al., 2015, pp. 25-31). Similarly, a study that assessed the PBDE serum levels of women in a case-control group in California also could not link BDE-47, BDE-100 and BDE-153 to an increased risk of breast cancer. However, the team was clear that there were limitations associated with the study, including

PBDE measurements that may not epitomise pre-diagnostic, personal history or chronic exposure, along with factors that may influence endogenous estrogen levels, such as genetic polymorphism (Hurley et al., 2019). While diet apparently is considered the primary exposure route, the cancer risk for adults consequential to the toxic substance is considered low (Ni et al., 2012, pp. 10–14). In contrast, tumours have been reported in liver tissue due to physiological changes to kidney and liver structure (O'Driscoll et al., 2016, p. 13223). These studies do not seem to offer definitive reassurance that the cancer risk does not exist, just that it is either low or has limitations. Therefore, it is suggested that further, updated research is required. In addition, clarification is needed regarding whether prohibited, but legacy, PBDEs still pose a cancer risk, or whether there might be a possibility that PBDEs are still available on the market.

8.4 LIFE CYCLE IMPLICATIONS OF THE TOXIC SUBSTANCE

PBDEs are highly ubiquitous and bioaccumulative, and despite the restrictions placed upon some congeners over the past few decades, a series of critical phases can affect the life cycle of the toxicant. These include the release phase during the production process of PBDEs and PBDE-containing products, the bioaccumulation phase within the environment and, also, the leaching phase within interior environments. Specifically, in relation to this field of research, PBDEs with a lower-brominated congener are understood to volatise within internal spaces or form dust particles during average dissipation and degradation from associated products or materials. Interestingly, some would argue that indoor exposure to the toxic substance is the most significant to society, subsequent to high levels found in human milk (Y. Lim et al., 2014, pp. 1376–1389).

Even though prohibitions have been placed on the use of some PBDEs, the toxicant can still be detected in breast milk. Korea, for instance, has a sizeable BFR market and decaBDE account for a quarter of that (Moon et al., 2012). Although, in 2008, the introduction of regulations of the toxicant restricted its content within electronic materials and devices to < 0.1%, and it was then listed on Korea’s Persistent Organic Pollutants Control Act three years later – the Act has progressively been updated, with the last update being 2017 (Statutes of the Republic of Korea, 2019). Nearly two tonnes of obsolete plastics from new televisions and nearly a third of a tonne from computer monitors were then recycled in 2011. Significantly, while PBDE exposure from dietary sources was understood to have subsequently decreased, little decrease in PBDE contained in breast milk was evident, (Kim et al., 2016, pp. 82–91), suggesting that environmental accumulation of the toxicant still persists but requires further ongoing studies.

Geographically, it is believed that PBDE levels differ owing to the industrialisation or commercialisation of various countries, for example, Canada and the US indicate higher levels of

PBDEs than do Asia and Europe (Daniels et al., 2010). Contrastingly, a study of the same year indicated a reduction in PBDE levels in humans because of the prohibitions in pentaBDE and octaBDE compounds. The team aimed to assess the incidence of BFRs in breast milk in Tanzania following a surge in imported consumer products, population growth and industrial progression. Levels of PBDEs in the breast milk of Tanzanian women were significant and on a par with findings from North American studies; in addition, 20% of the nursing infants had an estimated daily intake of BDE 47 and BDE 99 above the US EPA reference doses for neurodevelopment toxicity. It is believed that such elevated levels of PBDEs in Tanzania were associated with the burning and burying of PBDE-containing products rather than with environmentally and health appropriate waste methods. In addition, many pregnant women in the community consume a clay-based product, pemba, to relieve nausea and provide mineral supplementation. But importantly, elevated concentrations of PCDF and PCDD have been reported within some clay soils, with 64% of women involved in this particular study who consumed pemba exhibiting considerably higher concentrations of BDE 47, 99, 100 and 153 than the women who did not. However, it was noted that PBDEs in pemba had not been investigated, but the substantial correlation indicates a plausible PBDE-content in pemba and should lead to further investigation (Müller et al., 2016, pp. 38–47).

Interestingly, it has been reported that PBDEs can be transformed into dibenzofurans (PBDFs) and polybrominated dibenzo-p-dioxins (PBDDs**)** when thermal stress is applied to fire-resistant plastics, and subsequently, these compounds been discovered in commercial PBDE compounds as well as in lipid-dense connective tissue. Further, the metabolism of PBDEs has been known to create hydroxylated BDEs that can also cause harm (Costa & Giordano, 2007, p. 1062), which suggests that metabolic processes and even by-products of PBDEs when affected by various influential factors can potentially create accumulative issues throughout its life cycle.

An Australian study by Weijs et al. (2019) assessed the impact of various toxic substances, including PCBs, PBDEs and DDT, on dugongs in the Moreton Bay area, on the Queensland coast between the barrier islands and the entry of the Brisbane River. The study noted that Brisbane had around 2.4 million people occupying the third largest Australian city in 2018, through which the Brisbane River runs, consequently leading to excessive pollutants being washed into the bay primarily from agricultural and urban sources. While PBDEs returned low levels within Moreton Bay sediment, they were calculated to be higher in the Brisbane River. The study also identified higher levels detectable internationally (p. 504).

The Stockholm Convention have written a report (United Nations Industrial Development Organisation, 2021) to provide guidance on reducing and preventing the release of PBDEs into the

environment and atmosphere. Methods aim to employ practices of recycling, reuse and even responsible production process to minimise wasteful and toxic landfill. However, these practices do outline technical guidelines for ethical waste management systems when products or materials are contaminated by or contain toxic PBDEs for when recycling and reuse is not appropriate (pp. 24–29). The diagram below (refer to Figure 8.1), outlines the recommended waste disposal process of POPs containing PBDEs.

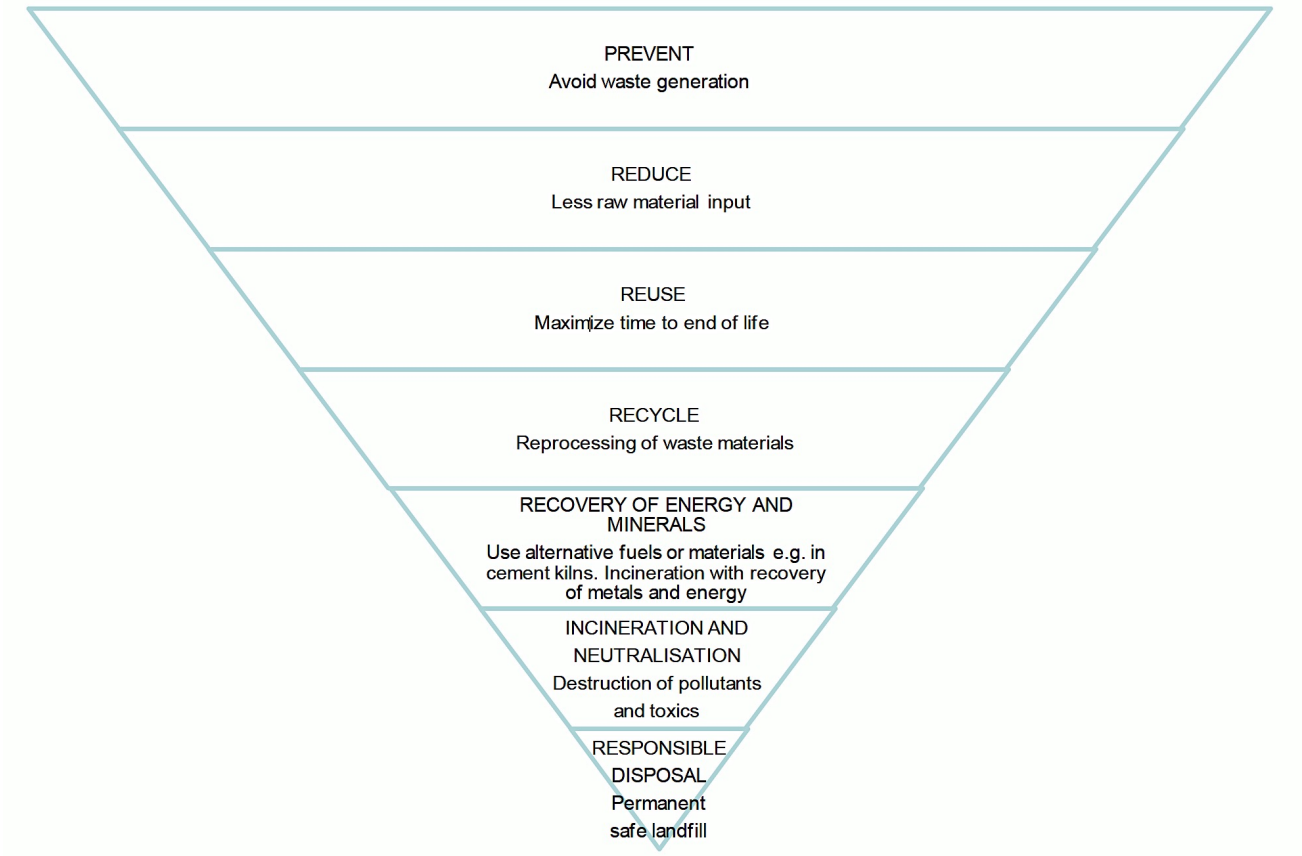

Figure 8.1: Preferred PBDE waste disposal of the Stockholm Convention

Note. (United Nations Industrial Development Organisation, 2021, p. 25)

Very few studies have conducted a LCA of the waste process for PBDEs, but typically have noted that landfill is the least favourable approach. Polyurethane is recommended to be recycled and repurposed to minimise its inclusion in landfill, for example, as carpet cushioning; however, unfortunately, this is yet to become a worldwide practice (United Nations Industrial Development Organisation, 2021).

It is difficult to assess the true lifespan of PBDEs because each congener acts differently. PBDEs with a lower congener are considered more toxic, whereas decaBDE may accumulate at a greater rate because of its lower molecular weight. However, more toxic congeners can be produced by the

bromination of larger congeners, and, significantly, smaller concentrations of BDE-209 can subsist within an organism's lipid cells, although the substance does not bioaccumulate as efficiently as some other congeners, and it has a greater molecular weight. It also has the ability to degrade into more harmful and absorbable congeners, such as pentaBDE and octaBDE (O'Driscoll et al., 2016, pp. 13222–13234).

8.5 THE WAY FORWARD

DEVELOPING STANDARDS

Since early 2007, the importation and production of pentaBDE and octaBDE were prohibited in Australia. OctaBDE is only permitted in the country for laboratory use for "analytical determination". However, the database of the government regulator of such chemicals in Australia, the National Industrial Chemicals Notification and Assessment Scheme (NICNAS), states that pentaBDE and octaBDE was no longer being imported around mid-2005 (Australian Government, 2007). At the time of writing this, more current information from the same source could not be found, although strangely, in 2019, an online report stated (Kempanion Ptd Ltd) that the Minister for Health and Ageing had restricted the production and importation of pentaBDE only two years prior, which is a little conflicting. The report outlined that the complete NICNAS risk assessment was not finished, even though the greatest concentrations of PBDEs in Australia were detected in children and lower levels in young women, according to studies commissioned by the Australian Government Department of the Environment and Water Resources. The director of the NICNAS was requesting more current information to assess the chemical in greater detail, including quantities that had been or were to be imported, and likewise for any products using pentaBDE as a mixture, as well as the use of such chemicals and the contact details of the companies involved (Kempanion Ptd Ltd, 2019).

ALTERNATIVES AND RECOMMENDATIONS

The use of BFRs have displayed a vast increase in the last 20 years and have consequentially been the subject or restrictions and prohibitions to minimise implications to the environment and human health due to the volatility of the toxic substances (Liagkouridis et al., 2015, p. 416). Some alternatives and recommendations include:

a. *Ensuring that new BFR alternatives are accompanied by a high level of scrutiny* to determine they will not carry long-term consequences like the former BFRs. With more rigorous fire regulations currently implemented by government bodies, the use of fire retardants is increasing. However, some of these emerging alternatives are displaying

similar environmental characteristics to those that were previously prohibited or restricted; hence, they may also carry similar health consequences (Eljarrat, 2011, p. 279).

b. *Reducing exposure to PBDE in association with other toxic substances.* As identified previously and discussed in greater detail in *Chapter 10: Discussion*, PBDEs should not be assessed as an isolated toxicant. With its chemical structure being very similar to that of PCBs, in particular, studies to date have already identified an accumulated toxicity discovered in adult rats, who were exposed to both toxic families. Low doses of PCB-52 and BDE-99 did not display behavioural implications when medicated individually. But significantly, when the two toxic substances were combined, although at the same quantity, behavioural changes were evident. In conjunction with the accumulation of PBDEs and PCBs, many other substances may contribute to such a toxic concoction that urgently require investigation – with those of note including dioxins, methylmercury, lead and perchlorate (Costa & Giordano, 2007, p. 1062).

c. *Redesigning common PBDE-containing products* to minimise exposure to the toxicant, while also removing the need for them to be manufactured. While potentially less cost-efficient to produce or supply, opting for materials that are naturally fire resistant wherever possible negates the need to integrate such toxic substances within them. Some examples of fire-resistant and/or retardant materials are provided below –

    a. *Wool* is a natural fibre that can replace synthetic textiles such as polyester. It has a high nitrogen and moisture content, with an ignition threshold of 600 °C and low heat of release and combustion. It is also hypoallergenic and naturally resistant to mildew, mould and dust mites (Sharieff, 2018).

    b. *Fibreglass* is predominantly formed from sand and glass, with additional raw materials sometimes including limestone, silica sand and soda ash. The sand and recycled glass composition offers fire-resistant properties, and it has therefore been utilised in applications such as textiles, thermal insulation and reinforcement (Moultry, 2021).

    c. *Clay* is considered a natural product also, offering limited environmental complications and disposal issues. Traditionally, it has been used for roof tiles owing to its fire-resistant characteristics (Future Content, 2014).

    d. *Some general building products*, including stone, concrete and brick, can be fire resistant. Although, care needs to be taken (as with any material) that flammable additives have not been incorporated into them. It is believed that, in the case of a fire, smoke inhalation and fire itself increases and spreads so rapidly nowadays in our homes that around 40% of fire victims are killed in their sleep (Kayo, 2021). It

should be noted, new and alternative BFRs should undergo extensive assessments to ensure there are no short-term or long-term implications for human health or the environment, to avoid the same problems being produced that the traditional PBDEs have created.

It should be noted that fire-resistant and fire-retardant materials cannot remove the risk of flammability within the interior built environment alone. There are significant standards and regulations, that may differ per state, territory and country that govern fire safety within buildings. These regulations are designed to minimise the risk for occupants, but also the damage to building. Critical factors include allowing sufficient egress paths and emergency exits, smoke warning systems, smoke and flame suppressions systems including sprinklers, installing fire protection equipment to name a few. In Australia, the NCC govern many of these factors based on Building Class. While fire retardant materials and construction systems are vital, they form only a portion of an overall systemic fire protection approach, and this chapter has focussed on the minimisation of specific toxic substances that can be found within building materials.

9. TOXIC SUBSTANCE SIX: ORGANOCHLORINES, ORGANOPHOSPHATES & PYRETHROIDS

9.1 INTRODUCTION

The manufacture of textiles accounts for approximately 43 million tonnes of chemicals on a yearly basis (Chen, 2019, pp. 1–41) The protection of agriculture and human health from diseases and pests has led to widespread use of pesticides around the world, more significantly throughout the last half a century (Jallow et al., 2017, p. 491). While most existing data (Fenske et al., 2002, pp. 549–553) focuses on agricultural exposure to such toxic substances, some studies (Lewis et al., 1994, pp. 37–46; Simcox et al., 1995, pp. 1126–1134) confirm that the greatest non-dietary exposure to pesticides does, in fact, occur within interior environments. Further, almost three-quarters of households use pesticides, leading to potentially significant exposure to their occupants (Julien et al., 2007, pp. 167–174).

Despite being one of the world's most significant non-dietary supplies, cotton is associated with approximately A$2.7 billion's worth of chemical pesticides. Critically, almost a quarter of these have been classified by the WHO as hazardous, with cotton being responsible for around 16% of worldwide insecticide releases (Environmental Justice Foundation, 2007).

The most common pyrethroids currently used include permethrin, cypermethrin, deltamethrin, fenvalerate and cyfluthrin, and the most commonly utilised organophosphate pesticides (OPPs) include chlorpyrifos, malathion, dichlorvos and parathion. Significantly, permethrin is unfortunately incorporated into many textiles (Heudorf et al., 2004, p. 68). Dichlorodiphenyltrichloroethane (DDT**)** and other organochlorines were utilised extensively until its prohibition in many countries from the 1970's, owing to its bioaccumulation in humans and the environment. This led to an increase in pyrethroids, organophosphates and carbates instead, with the former two of the three being the most exploited pesticides globally, primarily in the public health, forestry, horticulture and agricultural industries, but, more importantly, also within interior environments (Aslam et al., 2020, pp. 807–817; Heudorf et al., 2004, p. 67). For the purposes of this thesis, the following sections focus on DDT, chlorpyrifos and pyrethroids.

The below image demonstrates common locations in which textile pesticides may exist within a typical interior environment (refer to Image 9.1).

9.2 THE TOXIC SUBSTANCES

DDT – DICHLORODIPHENYLTRICHLOROETHANE

DDT was introduced as a substitute for toxic lead arsenic (Hu, 2018). The industrial manufacture of DDT began around 1943, after its discovery as an effective insecticide only four years prior. Surprisingly, it flew under the radar for such practices from its invention in 1874 until 1939. The global deployment of commercial DDT over the following decades was driven largely by cost – its value was reported to have dropped from US$1 per pound in 1945 to only US$0.25 per pound by the mid-1950's. After World War II, large quantities of the toxic substance were used agriculturally for the control of pests. However, it was also employed at this time for the prevention and management of typhus and malaria. By the 1960's, 400,000 tonnes were utilised globally per year, with around three-quarters being used agriculturally (Turusov et al., 2002, pp. 125–128).

DDT has the substantial ability to be absorbed into soils that have similar compositions of organic matter, and it can therefore be lethal to marine and bird life specifically. Its ubiquity led to its first prohibition in Sweden from 1970. The Ministry of Health in the Union of Soviet Socialist Republics (USSR**)** followed that same year, banning DDT in pesticides, based on its carcinogenic and bioaccumulate properties. The USSR furthered the prohibition of DDT in agricultural practices in 1981 but allowed continued use of it for the following six or so years for the management of public health, for example, for malaria. From 1989, it was also barred for medical and disinfecting reasons, with only limited state administration–approved use following epidemiologic alarms. Most countries followed suit and also prohibited DDT from 1972, but interestingly, this was primarily due to more prominent studies at the time raising environmental concerns, which outweighed the detrimental health effects. Despite DDT's rapid decline from the 1970's, the US proceeded to export around 300 tonnes of the toxicant in the mid-1980's. One company each within Indonesia, Italy and India were known to continue manufacturing DDT in 1990, accounting for approximately 30 million pounds (Turusov et al., 2002, pp. 125–128). The toxicant is still produced today in India for vector control (Cano-Sancho et al., 2017, p. 096002).

CHLORPYRIFOS

Chlorpyrifos was introduced as a substitute to DDT in 1965 by a company called Dow Chemical, and while it had since been the highest-utilised pesticide on agricultural crops, it was evident in more than 90% of homes tested in a study on the US population (Hu, 2018). It is classified as a broad-spectrum organic insecticide and an organophosphorus pesticide, is greatly toxic, can rapidly degrade in the environment and has long-range transport (Kutluyer et al., 2017, pp. 71–76).

Chlorpyrifos was prohibited for use residentially in 2001 owing to serious health concerns, with pyrethroids subsequently increasing thereafter (Julien et al., 2007).

PYRETHROIDS

Synthetic pyrethroids are more prevalent throughout the environment than their natural pyrethrin counterparts, leading to an increased use of the toxic substances within internal spaces like homes, as well as agriculturally. They operate similarly to pyrethrins, in that they too act as a neurotoxin to affect their target promptly. Pyrethroids interfere with the nervous system, potentially causing a lack of coordination and even tremors (Julien et al., 2007, pp. 167–168). Some pyrethroids, including permethrin, are utilised for the protection of textiles and other interior building materials, like carpet (Heudorf et al., 2004, pp. 67–72).

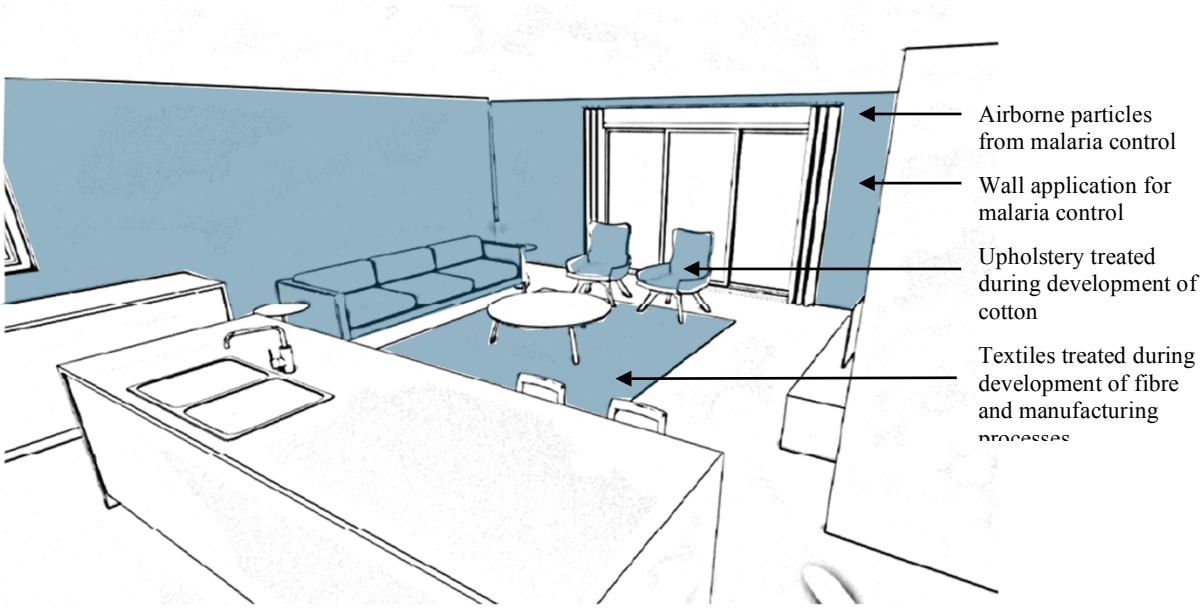

Image 9.1: Perspective of typical interior environment and prevalence of textile pesticides

9.3 HEALTH IMPLICATIONS OF THE TOXIC SUBSTANCES

HOW DOES EXPOSURE OCCUR?

The rising global population has led to a need for a greater agricultural yield to meet demands, but unfortunately that can correspond with an increase in subsequent pesticide use, in some countries more than others, such as Africa. However, the use of such toxic substances can result in detrimental environmental and health issues (Hinson et al., 2017, pp. 1–10). Awareness has been

increasing over the past four decades on the requisite to understand pesticide exposure to employees applying the substances in situ, for example, field personnel in direct contact with the treated produce, but also the general community who come into contact with the toxic substances in their own home and work environments. Sadly, to attain risk assessments for certain toxic pesticides, various doses are administered into animals via inhalation, dermal or oral pathways, with variations to both the duration of exposure as well as the life stage of the subject, to understand consequently the relationship to humans (Franklin & Worgan, 2004, pp. 1–10).

During application to crops, agricultural pesticides can disperse and become airborne simply from drifting and post-application volatilisation. The toxic substances have also been identified in ambient and indoor air, with the latter even being at concentrated levels. A study (Sharon et al., 2002) assessing various agricultural pesticides and their impact on the Californian community in 2002 resulted in a number of pesticides being analysed. Among the significant and present toxic substances, short-term or acute exposure to chlorpyrifos surpassed the associated reference value for 50% of children of the population, which has been linked to serious neurological issues (Sharon et al., 2002, pp. 1175–1184). Various pesticides, as indicated via gas chromatography, have been evident in cotton produce, including aldrin, dieldrin, hexachlorobenzene and, importantly, DDT. These toxic substances have been known to disperse as the fibres degrade (O Ecotextiles, 2010).

Disturbingly, some countries have continued to utilise DDT to prevent malaria since its banning in the 1970's. The Ministry of Health in Uganda aimed to apply the toxicant for enduring spraying of walls to terminate insects upon contact. Deltamethrin was used by the Ugandan Malaria Control Programme to assist in the management of associated epidemics, but it was costly. The Ministry of Health indicated that "if DDT is used it will only be for residual spraying. There are no intentions to use it in the environment" (Wendo, 2004, p. 1376). This opinion seems based on the loss of approximately A$480 million yearly from malaria and the belief that the use of DDT could reduce this, in conjunction with a cost to Africa of more than A$16.5 billion, with an African child passing away almost every 30 seconds from the disease (p. 1376).

Chlorpyrifos functions by attacking the nervous system of targeted insects; hence, it is not surprising that developmental impairments can occur in children subsequent to chronic exposure to the substance (Hu, 2018). The US and the EU have developed different allowable limits to exposure of chlorpyrifos. The US allows between 0.0012 and 0.002 μg/kg body weight daily (age dependent also), with children between the ages of one and two being the most at risk owing to dietary exposure of 0.027 μg/kg body weight daily in the median range, but as high as 0.242 μg/kg body weight daily for the 99.9th percentile. These measurements are based on recommendations from the

US EPA, which are in turn based on a risk assessment subsequent to epidemiological studies specific to development neurotoxicity, reinforced by autonomous research. Whereas the EU's allowable limit is 1 μg/kg body weight daily based on an industry-funded study that focuses on inhibition of erythrocyte acetylcholinesterase, with little regard to supported evidence from the US (Mie et al., 2018, pp. 1–5).

Dermal pathways to pesticide exposure are believed to be the primary route for workers during manufacture, with secondary exposure from contaminated non-agricultural surfaces also being of concern. The potential exposure sources have been linked to furniture, flooring and other materials that have been close to pesticide-applied areas. In addition, cotton textiles and clothing for consumer have exhibited the same toxic pesticides that were applied to the crops during production (Tsakirakis et al., 2018, pp. 2858–2863). The criticality of dermal absorption of the pesticides can be determined by the pathway. These include:

1. the transcellular path, which is the moving of chemicals between the extracellular zone and the cell membrane
2. the intercellular path, which is the movement of chemicals through the extracellular matrix within ingoing or outgoing the cells
3. the appendageal path, which is when chemicals can track deeper into the layers of the skin via appendages such as hair follicles (Eleftheriadou et al., 2019, pp. 561–562).

Ingestion has also been a concern regarding organochlorine insecticides and other toxic substances, and it can be persistent within interior environments where the substance's dilapidation can be protected from the natural elements and predatory microorganisms. Such chemicals have been commonly detected in homes that were constructed (or renovated) prior to the banning of these substances like DDT and PCB, with young children being at high risk of ingestion through direct contact of house dust (Ward et al., 2009).

WHAT ARE THE HEALTH EFFECTS?

While most pesticide exposure studies focus on agricultural perspectives, it is evident that many pesticides prevail in interior environments, especially homes, with a toxic range that can even include banned and restricted products. Older homes can have more excessive concentrations, along with pest-infested dwellings or those lacking general maintenance. Childhood cancers, implicated foetal development and asthma are just some of the serious health consequences, with the latter being alarming given that the application of pesticides within homes is generally to minimise allergens (Julien et al., 2007, p. 167). It should be noted, however, that organophosphorus pesticides and pyrethroids are believed to metabolise quickly in humans. But importantly, the metabolic

process to remove the toxic substances from the body vary depending on the type of exposure and the substance itself. A study (Heudorf et al., 2004) indicated that detoxification from dermal exposure to organophosphorus pesticides can take almost twice as long than from oral or ingested exposure. Similarly, detoxification from pyrethroids require less than half of the time it takes for oral exposure from the organophosphorus pesticides (pp. 67–72)

DDT has been known to produce deleterious health effects from its profound production period during the twentieth century, and it was as early as 1962 that a report by Rachel Carson warned of the bioaccumulation DDT can have, as well as the amplification further up the food chain, with long-term implications for animals, with potential implications for humans as well (Eskenazi et al., 2009, pp. 1359–1367).

Interestingly, the reported effects that chlorpyrifos can have on society vary between industry-funded and independent studies (Mie et al., 2018, pp. 1–5). Various animal studies have demonstrated toxic consequences from exposure to bodily biochemistry; blood parameters and also individual organs can in turn then impact the urinary, reproductive, immune, liver and nervous systems. One particular study (Barzi et al., 2020) assessed 30 female mice, which were divided into three groups. One group received an injection of intraperitoneal chlorpyrifos, another was injected with dimethylsulfoxide and the control group received no injection. Chronic exposure to chlorpyrifos was found to impact foetuses potentially by increasing the apoptosis pathway in developing embryos, which can lead to abortions or developmental disorders in the newborn mice (pp. 794-803). Low concentrations of the substance have also been linked to histopathological, oxidative, autoimmune, endocrine and behavioural changes. Chlorpyrifos can also reduce the survival and motility of sperm cells in males through oxidative stress (Kutluyer et al., 2017, pp. 71–76).

Like chlorpyrifos, exposure to OPPs can lead to significant implications for the nervous system by disturbing the enzyme that controls a neurotransmitter, acetylcholine; similarly, so can exposure to some synthetic pyrethroids (Dikshith, 2011, p. 8). Moreover, pyrethroids also affect insects and mammals by interrupting the sodium channels of the axons, or nervous fibres.

Pyrethroids have been linked to complications associated with prenatal development and fertility, specifically reproductive hormones, sperm concentration and pregnancy impediments. Both non-occupational and office environments have been associated with the ill health of occupants as a result of pyrethroid exposure. It is believed this is generally caused by the application of the pesticide itself within an enclosed space and common negligence – spillages, excessive use,

minimal ventilation, drifting of the chemicals, irresponsible storage and the like (Saillenfait et al., 2015, pp. 281-292).

Further, irritations and dermatitis have been associated with common textiles, owing to dermal exposure. While a recent study (Rovira et al., 2017) aimed to investigate a large series of trace elements in common textiles, more so than pesticides and insecticides, the authors, interestingly, identified that the usage and duration associated with textiles can contribute to health implications following exposure. For example, clothing and linen is typically in direct contact with the skin for extended periods of time daily; in addition, physical stress and sweat can influence the release of certain toxic substances. While a bath towel is not in direct contact with the skin for extended periods of time, the friction associated with rubbing a towel against skin for drying may also release toxic elements (pp. 1966-1974).

9.4 LIFE CYCLE IMPLICATIONS OF THE TOXIC SUBSTANCE

Prior to its prohibition, DDT was primarily used residentially for termite control and agricultural reasons (Yeo et al., 2015, p. 141). Additionally, with the ongoing use of DDT for the prevention of malaria in affected countries, there have been concerns about exposure to both occupants and the applicators of the toxicants. The reapplication of DDT to internal wall faces of homes annually suggests that it will remain residual for the prevention of mosquitoes for up to a year (Bouwman et al., 2011). Although, with reference to section 9.2 Health Implications of The Toxic Substance above, this is still extremely concerning, with arguments continuing regarding the health implications of DDT directly in comparison to alleged lives saved from malaria. Put into perspective, it has been reported that 7,300 deaths and 2.8 million cases of the disease have been reduced to no deaths and only 17 cases regarding the use of DDT in Sri Lanka over two decades (Radcliffe, 2002, p. 2).

Textile production specifically conventionally concerns an extensive and chemical-laden process. Beginning with cotton seeds for propagation, the seeds are treated with insecticides and fungicides. Weeds are repressed with treatments of herbicides, and pest control involves further use of insecticides and pesticides. DDT, aldrin, dieldrin and hexachlorobenzene are a few of the more prevalent pesticides exhibited in gas chromatography. However, unfortunately, the chemical process does not stop there. While some of these chemicals are designed to have a half-life of up to four days before degradation, and some can eventually be washed out of the textile over time, other additives are designed not to wash out in order to contribute to certain characteristics of the fabric or they can be residual from the manufacturing processes. Heavy metals, formaldehyde, dyes and dioxin are some of the more notable toxic substances. To put this in context, genetically modified

cotton can produce around 450 grams of traditionally grown cotton with around 28 grams of pesticides, which is about a 58% reduction in pesticides since the 1990s. Significantly, to develop those cotton fibres into a textile can require an additional 1–11 kilograms of chemicals (O Ecotextiles, 2010).

Textiles have also been considered for burial practices, with a green burial movement expanding primarily in the UK during the past 20 years. The movement aims to minimise contaminants to the ecosystem, including the soil and waterways, while utilising non-toxic and sustainable textiles that support bodily decomposition in burials, using cradle-to-cradle principles (Michel & Lee, 2017, pp. 1-18).

An article from 1994 indicated that DDT residues were still existent in the New Zealand environment as a result of former applications of the pesticide between the 1940's and 1970's, suggesting that the toxicant can remain ubiquitous for decades (Boul, 1995, pp. 257-277). Irrigation pathways are widely acknowledged as a predominant DDT-contaminated zone, from which the DDT can then spread to aquatic areas (Cagnazzi et al., 2019, p. 268). Yeo et al. (2015) analysed a worldwide monitoring program of POPs, International Pellet Watch, monitors and assesses pollution in the oceans. The program discovered the existence of DDD and DDE, which are metabolites of DDT, in sediment and soil. This suggests the presence of legacy substances from previous decades of usage. However, at Port Phillip Bay off the coast of the Victorian coast of Australia, DDT itself accounted for 80% of measurements, indicating recent applications or usage of the toxicant. Yeo et al. suggested that the recent discovery may be related to dicofol pollution, given the area is a harbour zone. Dicofol was noted to be an innovative version of DDT, apparently utilised in anti-fouling paints (pp. 137-145). Although interestingly, the Australia Government's Australian Pesticides and Veterinary Medicines Authority list dicofol as an organochlorine for treating red spiders and mites on vegetables, fruit, cotton and ornamentals; but significantly, an initial approximation states that chronic dietary consumption of residues actually exceeds the acceptable daily intake. However, the authority also states that no acute reference dose (ARfD) has been determined in Australia, and in addition, acute dietary consumption raises trepidations based on international ARfD levels (Australian Pesticides and Veterinary Medicines Authority, 2017).

The study of dugongs (Weijs et al., 2019) in the Moreton Bay and Brisbane River areas of Australia outlined that organochlorine pesticide concentrations in these zones declined between 1998 and 2008, although levels of DDT were around 25 times greater around the Brisbane River entry in comparison to upstream areas. However, the extreme weather events that caused floods in 2011 and 2013 are believed to have increased levels of many organochlorine pesticides in sediment,

suggesting that the flooding may have relocated bioaccumulated toxicants into more concentrated areas. Such POPs in this bay area are understood to be legacy substances, and include agricultural and urban toxic substances (pp. 500–508). However, are remnants or portions of such substances that are evident along the path of relocation spreading the toxicity to affect the food chain further?

9.5 THE WAY FORWARD

DEVELOPING STANDARDS

DDT, along with other POPs, like PCBs and hexachlorobenzenes (HCB), has been prohibited in certain countries since the 1970's. Although evident concentrations have been declining, it is still persistent in the environment. It was formally prohibited for general us in Australia in 1987 and the US in 1972 (Australian & New Zealand Guidelines for Fresh & Marine Water Quality, 2018).

In 2019, Australia cancelled the registration and suspended the labels of chlorpyrifos-containing products, and the remaining registrations and label approvals were cancelled in 2020 in an attempt to reduce its use in the country owing to progressive research and scientific evidence associated with its health implications (Australian Pesticides and Veterinary Medicines Authority, 2021). No restrictions or prohibitions could be discovered at the time of writing this thesis for the use of synthetic pyrethroids within Australia; however, this is recommended to ensure it is safe for use in and around the built environment – and subsequently, the environment.

ALTERNATIVES AND RECOMMENDATIONS

Seeking alternative approaches to pesticides, including DDT, is desperately needed. With more developed countries not requiring DDT, it has been recorded that 23 countries in tropical zones still utilise it. Even though the use of DDT is restricted through the Stockholm Convention on POPs, exemptions are included for disease vector management for specified countries (Radcliffe, 2002, p. 2). However, instead of allowing such exclusions, are there alternatives that can be offered by the United Nations to minimise the impact on the occupants of treated homes? Are there alternative methods that less-developed countries can adopt to follow those countries that have seem to have successful mosquito management process in place? Some alternative approaches to minimise the use of pesticides include:

a. *Seeking less toxic treatments for mosquito prone areas* is necessary to reduce the enduring toxic effects of DDT. However, financial support may be required by countries, with the cost effectiveness of DDT is one of the primary reasons for its continued use to minimise malaria case (Turusov et al., 2002)

b. *More alternatives should be endorsed that offer resistance to building materials, crops* and the like. Hemp, for instance, is considered a cost-effective, sustainable and ecological plant that offers a plethora of uses. Its wide variety of applications, from building materials to textiles, agriculture, food and even skincare, renders it a minimal wastage product also. It is resistant to moisture, biodegradable and flexible but strong. Hemp textiles can also offer UV resistant properties and are hypoallergenic and antimicrobial (Crini et al., 2020, pp. 1451-1476).
c. *Ensuring textiles are certified to the Global Organic Textile Standard.* This third-party standard ensures that the associated textile not only is made from organic fibres but also has been organically manufactured. Further to section 9.3 Life Cycle Implications above, this certifies that toxic chemicals have not been incorporated into, or processed with, harmful substances (O Ecotextiles, 2010).
d. Synthetic materials are commonly produced from petroleum and large quantities of chemicals, which can lead to bioaccumulation within the environment and long-term health implications. These principles are paving the way for an awareness to opt for natural fibres when it comes to textiles. The *Leadership in Energy and Environmental Design, or LEED, certification declares an ethical life cycle assessment* for the end product, with the cycle branching from production processes through to waste, emissions and transport. While the certification is pivotal to sustainability, it does address the LCA processes in minimising long-term harm to the environment, and in turn, to human health (Gam et al., 2020).

10. DISCUSSION

10.0 SUMMARY & DISCUSSION

This research sought to investigate the connection between the presence of six selected toxic substances in the interior built environment, and the effect these toxic substances can have on human health. Ultimately, this research pushed beyond building products themselves, to expose factors within the built environment that influence toxicity.

Six toxic substances were identified as common during the initial background searches finding studies that identified these substances within residential and workplace dust samples. The literature review then revealed their prevalence in a range of interior spaces within today's communities. Having established the selected substances, and having drawn data from the Literature, a template was drafted with the intention of structuring an inquiry into each toxic substance so as to explore what exactly each substance is, how it is produced, and what, if any, value does the substance have. Why is the toxic substance used, or in the case of mould, why does it occur? Are there regulations governing the use of these substances? What are the health implications? And what are the consequences of their ongoing presence? Further, what is the life cycle of each substance, how long can it persist within the interior and exterior environment and can materials containing these toxic substances be recycled or reused? Finally, the template allows for consideration as to whether there are any actions or recommendations that can be offered to minimise the use of each substance thereby reducing any associated health consequences?

The nature of this inquiry revolved primarily around *how* and *why* questions, which led to the methodology being a collective case study. Each toxic substance became a case in its own right, allowing for a concluding cross-case analysis. While case study research methods are commonly criticised (Tsang, 2014), this multi-case study method offers insight into this contemporary phenomenon, allowing deep investigation and theory building that can exceed the more exploratory and analytical research methods so as to really delve into how and why certain decisions have been made in relation to the phenomenon (Zach, 2006, pp. 4–21). The primary findings can be summarised as follows:

Table 10.1: Comparison of cases

The Toxic Substance(s), **The Case**	***What* is it?**	***How* do the toxic substances exist within our environment?** ***How* are we being exposed?**	***Why* does it occur or *why* were the decisions made to utilise it?**
Mould	Naturally occurring microscopic fungi that feed on nutrient-dense and plant-fibre and cellulose products, including some building materials and interior (E.g. timber products, paper-lined plasterboard; Money, 2004, pp. 3–22). Some variants of mould have the ability to create mycotoxins – highly toxic compounds (Marquardt, 1996, p. 77).	A two-fold issue that relates to the progression of the fungi: 1. Selection of building materials susceptible to mould growth. 2. Building defects that encourage mould growth. Exposure can occur through dermal absorption, ingestion and inhalation, and importantly, the genera and concentration of the mould species effects the toxicity (Heyman, 2018).	1. Plant-based building materials can offer a high level of sustainability and renewability. And also, items like paper-lined plasterboard offer cost effective, simple installation and streamlined building processes to meet the current construction demand (Gradeci, Labonnote, Time, & Kohler, 2017). 2. Building defects encourage fungal growth (Singh et al., 2010) and are commonly linked back to a lack of expertise or hasty construction methods to increase profit margins and save time.
Phthalates	Phthalates are a family of industrial chemicals, and the esters of 1,2-dibenzene dicarboxylic acid (Api, 2001, p. 98). This thesis briefly outlines the most common phthalate congeners, but delves into the toxicity of di(2-ethylhexyl) phthalate (DEHP) as its primary focus due to its prevalence within the interior built environment. DEHP is a minimal-odour, colourless liquid that can form up to 40% of the composition of some vinyls (Agency for Toxic Substances and Disease Registry, 2002).	Phthalates are incorporated into the manufacturing process of the numerous building materials, for use as a plasticiser. Though their integration within the product cannot be removed from the final product, the esters are not actually chemically bound, which allows them to disburse into the surrounding environment (Clausen et al., 2010, p. 2760). Humans can be exposed primarily through dermal absorption, ingestion and inhalation (Lin et al., 2011, p. 217).	Phthalate esters were introduced in the early 20th century to supersede the former volatile and pungent camphor, as a plasterciser (International Agency for Research on Cancer, 1989, p. 337). It can offer various characteristics including flexibility, longevity, durability and transparency in products such as polychloride vinyl, and as fragrance and solvents in personal care items (Wynters & Goldberg, 2012). The cost effectiveness associated with PVC products overrides the selection of alternatives such as linoleum (Scott, 2009, p. 15).

Formaldehyde	Formaldehyde is a natural substance created through the oxidation of hydrocarbons and occurs in some organisms through metabolic processes. Though commercially, it is primarily a synthetic compound. It is flammable, highly reactive at room temperature, and a colourless gas (WHO Regional Office for Europe, 2010, p. 103).	Multiple polymers within common building buildings contain formaldehyde resins, like urea-formaldehyde and phenol-formaldehyde, and are incorporated into the production processes of these items. Materials constructed with these two compounds in particular, generally then omit formaldehyde gas into the surrounding atmosphere, with urea-formaldehyde typically off-gassing higher levels. Humans can be exposed primarily through dermal absorption and inhalation (Franks, 2005, pp. 309–320).	Formaldehyde is still used today for its high versatility in building materials, as well as its ability to preserve biological specimens and disinfect bacteria within the medical and mortuary industries (Beheshtian et al., 2013, p. 1331). Following concerns of toxicity, alternative approaches to adhesives have recently assessed food stuffs, including corn flour and sodium hydroxide and also soybeans, as options. Similarly, lignon-epoxy resins and latex-based paints have been investigated for use within common building products instead.
Polychlorinated Biphenyls (PCBs)	PCBs are a family of synthetic chemicals that were primarily used between the 1930's and 1970's, but have since been phased out due to health and environmental concerns. The organochlorine compound mixtures can account for up to 209 individual congeners (Spengler et al., 2001).	Materials containing PCBs have generally been regulated to minimise further exposure. Approaches formulated by the EPA suggest management practices initially, and removal and repair of PCB-containing materials and products (United States Environmental Protection Agency, 2015), suggesting that the PCB-integral component of such materials are safer when removed in their entirety. Humans can be exposed primarily through dermal absorption, ingestion and inhalation (Herrick et al., 2004, p. 1051).	PCBs offered a vast array of beneficial characteristics, including chemical stability, non-flammability, insulating properties, and high boiling point. This diversity offers promising benefits as a plasterciser in plastics, rubber and paints, as well as equipment requiring hydraulic, fire and electrical resistance. This has led to extraordinary manufacturing and application rates (Spengler et al., 2001).
Polybrominated Diphenyl Ethers	PBDEs are a group of organobromine compounds, and like PCBs, include 209 congeners. They are structurally similar to PCBs. PBDEs have less bromine atoms per molecule and are	PBDEs are incorporated into products and materials during their manufacture to add fire resistance properties. Some studies have identified various BDE-removal practices to	PBDEs began to be introduced into general consumer products and building materials from the 1970's to reduce the ignition and growth rate of fire for the safety of the occupants, prior to the understanding of the

	considered more toxic due to their rate of bioaccumulation.	reduce the toxicity, including photodegration, anaerobic biodegration, adsorption and photochemical degradation have been reported as potential remediation practices, though these have primarily focused on remediating solutions or environmental contamination (Chang et al., 2017, pp. 1–11), rather than BDEs integrated into building materials from their manufacture. Further research is required in this regard. Humans can be exposed predominantly through dust ingestion, as well as dietary ingestion and inhalation.	bioaccumulation effects to the surrounding environments as well as human health. DecaBDE has been classified as a probable carcinogen but is still used in many countries. Whereas PentaBDE and OctaBDE have been restricted since the early 2000's following widespread concerns (National Ocean Service, 2021).
Organochlorines, Organophosphates & Pyrethroids	Organochlorines are a family of chlorinated mixtures used commonly as pesticides, whereas organophosphates are chemical compounds made from phosphoric acids and alcohols. Pyrethroids are also synthetic group of insecticides. They are a chemical family that do not all have similar chemical structures but are classified based on their biological function. Their additives, acids and ethers can all vary, but all act as axonic excitotoxins to paralyse their target (Heudorf et al., 2004, p. 68).	All of these insecticides and pesticides are created with the intent to kill such pests. Though they impact pests in different ways: The fat-solubility of organochlorines allow the synthetic pesticides to bioaccumulate and persist in the environment. They target insects by causing spasms by exposing the sodium ion channels and forcing them to fire unexpectedly. Organophosphates damage the acetylcholinesterase enzyme in insects, which is vital for the control of nerve signals. Pyrethroids operate by impacting the nervous system of insects, causing a debilitated state and then death. They are created as chemical formulas with additional additives or petroleum to allow them to have a greater resistance	Organochlorines were created for insect control, with specific types like DDT being used for their cost efficiency and effectiveness. The neurotoxic nature of them though has led them to be banned in many countries, despite their widespread use still in developing countries. Organophosphates were, too, developed as insecticides, though the German military created them as neurotoxins during WWII. More than 25,000 pesticide brands are utilised in the USA alone, but are being monitored by the EPA (Dyro, 2020). Pyrethroids were developed as an alternative to pyrethrins, which are the natural oil originating from chrysanthemum flowers, to address the shortages in the natural approach (Julien et al., 2007, pp. 167–168).

		to light and longevity when compared to the natural version, which allows them to breakdown much more slowly also. Dermal absorption and ingestion have been considered the primary exposure routes for humans (Franklin & Worgan, 2004, pp. 1-10).	

The cases investigated in the research, while diverse in type, have uncovered similarities between the toxics substances. Whether the substances are toxins or toxicants, the harmfulness is instigated by the concentrations and/or the length of time that exposure occurs. Similarly, inhalation, dermal absorption and ingestion are common exposure routes of most, though formaldehyde did not have much evidence to indicate ingested exposure and chemical pesticides did not portray much known evidence of inhalation as an exposure pathway. Surprisingly, extensive research papers and peer-reviewed journal articles identified the critical nature of these prevalent toxic substances and how they may even effect human health and the environment long-term.

Despite some toxic substances being prohibited already, this research has identified that the ubiquitous and currently widely-used toxic substances, to which we are continually being exposed to date, both naturally occurring (like mould) and synthetic (like phthalates, formaldehyde, BDEs and certain pesticides), need much more regulation and public awareness of the toxic substances existence – what they are, where they exist, how we are being exposed to them, and what we can do about it. There is also the abiding issue, that when developing potentially healthier alternatives, we need to be cautious to investigate potential consequential issues to both human health and the environment, as much as humanly possible.

Importantly, throughout each case a series of factors including decisions and actions were identified as ways in which harmfulness within the interior built environment may be effected, and ways in which exposure to these toxic substances may be reduced. These factors include:

a) *Selecting low-VOC building materials and finishes*. Indoor air pollution is understood to primarily be from building materials and their associated VOCs. Harmful emissions can be reduced by careful practices that control the source of these toxic substances and minimise the volume of VOCs in an interior space. Opting for truly natural products is ideal. However, selecting materials that contain minimal additives, from adhesives through to lacquers, can reduce substances like formaldehyde. Research (Chen et al., 2013) also

indicates that ensuring a minimum of 30% of building materials be composed of green building materials with ultra-low emission can still significantly reduce the VOC content in the surrounding environment (pp. 126–130). Worth noting here is the deceptive practice known as 'greenwashing' that has increased since environmental awareness began to rise from the 1970's. Essentially, "greenwashing" is the assertion of misleading environmental claims, pitched for marketing purposes at unsuspecting consumers. Information, regulation and awareness are required to ensure consumers are not being misled and that we are able to determine the true "green" principles when formulating decisions (Rotman et al., 2020). Similarly, exercising attention with materials marketed as "natural" is important, as these may still contain various toxic substances. For example, a vinyl may stipulate being phthalate-free. But is there an alternative plasterciser incorporated into the product, and does that carry any similar hazardous properties?

b) *Enhancing IAQ – reducing moisture, mould, airborne toxic substances*. Indoor pollution can be categorised within one of the five following groups:

Table 10.2: Typical Groups of Pollution

Group	**Pollution Source**	**Location**
1	Contaminated urban air entering the building	External
2	Anthropotoxins (toxic human waste)	Internal
3	Household activity-based sources	Internal
4	Microbial air contamination	Internal
5	Building material toxins and toxicants	Internal

Group 1 is primarily beyond the control of an individual, excluding an individual's contribution to the grander scale of issues at a global level, it includes pollution from transport, industrial plants and the overall off-gassing of the urban context. Groups 2 through to 4 can be influenced by an individual residing within the premises. Significantly, Group 5 is believed to account for up to 80% of chemical toxins and toxicants within indoor air. These have been noted as originating from both the materials themselves, as well as degradation process during its use, whether it be microbial destruction or wear and tear (Zhigulina & Chumachenko, 2017, pp. 1–6). Notably, air exchange rates, humidity and temperature are just some of the chemical and physical elements that can contribute to the IAQ (Gunschera et al., 2013, p. 138). An increase building impermeability and air tightness, for the purpose of energy

efficiency, may also lead to compounded contaminants within the interior built environment that include particulate matter, VOCs and biological organisms (Vardoulakis et al., 2015, p. 299). This energy conservation has been linked to an increase in health detriments, as many sustainability-focussed buildings focus on the cradle to grave approach of the building itself and its constituents, but do not factor in the health of the occupants (Sundell, 2010, pp. 185–186). While various certification schemes exist, including Green Star in Australia, Leadership in Energy and Environmental Design (LEED) in the US, the Building Research Establishment Environmental Assessment Methodology (BREEAM) in the UK, many do include consideration of IAQ. Though IAQ measures typically only contribute to 3% to 11% to of overall certification credits, which may not offer much incentive. In addition, the increase in air tightness within some 'green buildings' in order to increase energy efficiency can also have deleterious effects on occupants, due to a lack of ventilation and even the use of recycled materials that may contain toxic substances from their previous form (Steinemann et al., 2017, pp. 351–358). Contrastingly, Passivhaus practices utilise mechanical ventilation methods without recirculating air to assist with the optimisation of IAQ (Moreno-Rangel et al., 2020, pp. 1–16).

c) *Reducing microbial growth*, especially one of its predominant precursors – water damage – is another critical factor that can affect the toxicity of the interior built environment. Air purifiers can assist in improving IAQ by filtering out microbiological contaminants, as well as other airborne pollutants. A study that assessed the concentrations of culturable bacterial aerosol in residences in Poland found an improvement of 50% in homes that utilised sufficient air purifiers compared with those that did not (Brągoszewska et al., 2019, p. 7). With moisture typically occurring from rising damp or absorption from the surrounding land, infiltration, condensation, significant weather events and even just simple building occupation and functions (Beckett et al., 2020, p. 2), current building practices need to adhere to the latest building regulations and standards to ensure the utmost quality of the interior environment. For Australia, this relates to the NCC and the Australian Standards, along with any local and state regulations, as required. The NCC has recently reviewed condensation requirements, including the new Deemed to Satisfy Provisions in Part F6 (Volume One) and Part 3.8.7 (Volume Two) of the NCC (Australian Building Codes Board, 2019). Notably, however, designers and interior architects are required to comply with all locally-required regulation, including the NCC, Australian Standards and locally-based regulations. Building occupants, tenants and home-owners need to minimise the risk of water ingress internally. These include maintaining satisfactory RH levels. RH is also critical, with 75–95% RH required to minimise indoor moisture. While nutrient composition of building materials is also an important factor

(Gradeci, Labonnote, Time, & Köhler, 2017). If moisture levels are maintained within acceptable levels, there is little risk of them being affected by microbial growth.

d) Further to section 10.1 Cross-case Analysis and 'Reducing microbial growth' in section 10.4 above, *maintaining ideal conditions* in interior environments is critical in reducing the volatility of some toxic substances. The rate of emissions of VOCs and formaldehyde from building materials, for example, has been reported to be affected by RH and temperature. Historically, this too has been assessed with only a single factor – although a reasonably recent study (Xiong et al., 2016) identified a relationship between the emission rates of such substances when both the temperature and RH change simultaneously. A former reference room model was initiated into Xiong et al.'s study to assess the human carcinogenic potential, with an RH of 70% at 25°, which unfortunately resulted in an elevated cancer risk (pp. 734–741). This, too, signifies the importance of future studies focusing on the accumulative effects of toxic exposure to a number of toxins and toxicants, not just a single substance in designated circumstances. Realistically, exposure to multiple toxic substances is common in our current interior environments where we are rarely exposed to a singular toxic substance in perfect conditions. Therefore, research studying the effects on human health of exposure to multiple toxic substances is recommended, though of course understanding that there are so many drivers that impact internal spaces.

e) *Sustainability and the overall life cycle* should be considered during the specification and design processes, and even when purchasing products. Brzyski et al. explained the importance of sustainability throughout the holistic building process, with life cycle, utilisation and disposal being as critical as procuring constituents, the manufacture of materials and appropriate processes after demolition (2019, p. 020005).

The snapshot images on the following page (refer to 10.1 to 10.6) are those provided in the previous chapters of the 6 toxic substances. The lower image (refer to 10.7) is an overlay of all 6 sketches provided to each individual case, to further emphasise the importance of acknowledging the accumulated effects that toxic substances may have in any one interior space. It is vital to understand that, while analysing the toxicity of individual contaminants, there also must be an appreciation that the concentration and the accumulated effect can create the toxicity.

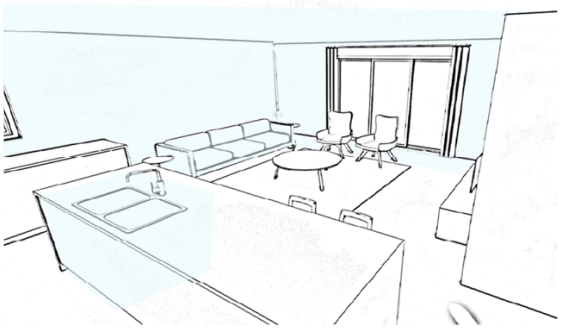

Image 10.1: Common locations of mould

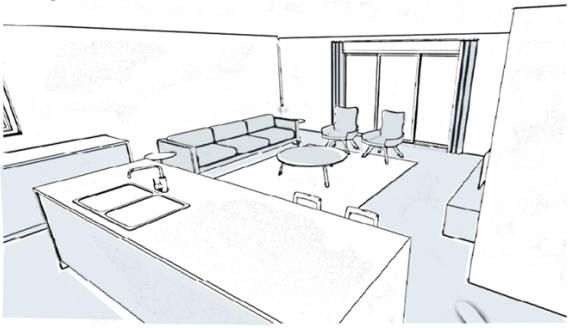

Image 10.2: Common locations of formaldehyde

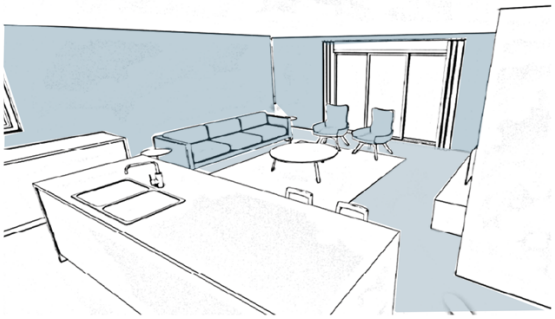

Image 10.3: Common locations of phthalates

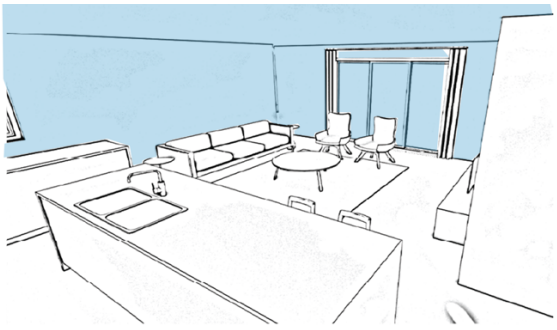

Image 10.4: Common locations of PCBs

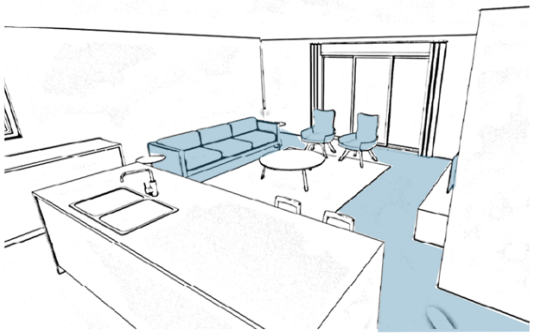

Image 10.5: Common locations of PBDEs

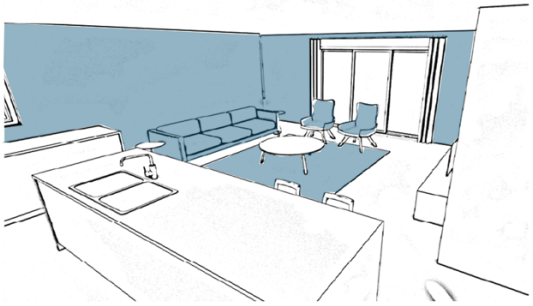

Image 10.6: Common locations of textile pesticides

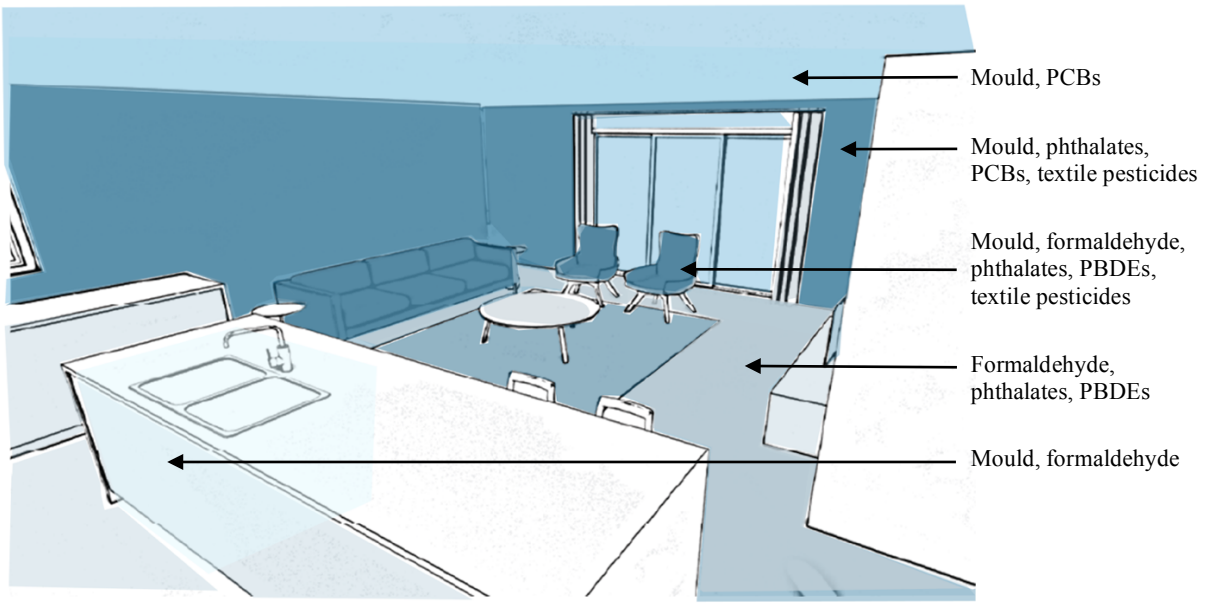

Image 10.7: Overlay of the six above images, indicating our toxic environments and common locations of such toxic substances

10.1 COMMON BUILDING MATERIALS TO AVOID

Buildings today contain a concoction of toxic chemicals that pollute the interior environment, and in turn, negatively affect our health. These thousands of chemicals must also be considered contextually during the manufacturing practices – that being, harmful toxic substances are produced during these production processes that off-gas into the surrounding environment and also pose health implications to workers within such chemical factories (Pacheco-Torgal & Jalali, 2011). Though contrastingly, with a contemporary focus on sustainable practices including recyclability, toxicity needs to be assessed through the entire life cycle of a material or product. That said, recycling products that may contain hazardous substances can lead to toxic exposure to both humans and the environment once a materials is redeveloped into a new form (Janssen et al., 2016, p. 5). And with society being exposed to an accumulating number of new chemicals, providing high levels of exposure and toxic concoctions (Sundell, 2010, pp. 185–186), it is critical that sources of toxic exposure are reduced wherever possible. Appreciating that technologies are always advancing, the following table indicates some possible alternatives to common building materials:

Table 10.3: Common building materials to avoid

Common Building Materials	**Toxic Substances Commonly Associated with Such Materials**	**Possible Health Implications**	**Possible Alternatives**
Timber products, including furniture, composite timbers, medium density fibreboard, chip board	Formaldehyde Mould	DNA damage Cancer Reproductive Issues Asthma & Allergies Mycotoxicosis Cancer Allergies & Asthma Hepatotoxicity Sensitivity Multi-symptom Illnesses	Silver nanoparticles supported by titanium dioxide, incorporated into plasterboard Natural biocides Chitosan-copper complex-treated bamboo Hemp Materials incapable to tolerate changes in conditions Reducing paper-lined and nutrient-dense materials, noting the key is in the management of moisture and humidity, in reducing mould prevalence
Vinyl-containing materials, including wallpaper, flooring, upholstery	Phthalates Formaldehyde	Foetal and reproductive issues Asthma & Allergies Endocrine & hormonal disruptions Neurotoxicity Vision impairment Cardiovascular Issues	Products free of phthalate, especially di-(2-ethylhexyl)-free phthalate (DEHP) Potentially Di(isononyl) cyclohexane-1,2-dicarboxylate-containing plastics instead of DEHP, pending further research

Common Building Materials	Toxic Substances Commonly Associated with Such Materials	Possible Health Implications	Possible Alternatives
		Numbness & pain to limbs	Bamboo, cork and linoleum products, subject to chemical content associated with adhesives, plastercisers and binders Tiles offer a cost-effective alternative to vinyl products Natural stone and timber, subject to chemical content of possible adhesives or sealers
Adhesive or preservative-containing products, including composite timber products, plywood, textiles, carpet, panelling	Formaldehyde	DNA damage Cancer Reproductive Issues Asthma & Allergies	Lignon-epoxy resin Products containing corn flour and sodium hydroxide based adhesives, subject to allergies Products containing soybeans, subject to allergies and associated curing agents Latex-based paints, subject to allergies Natural wool carpet rather than solution-dyed nylon Low-VOC paints Phthalate-free wallpapers, vinyls and textiles
Paints Electrical equipment Sealants Wall coverings Fluorescent light ballasts Caulking	Polychlorinated biphenyls (PCBS) Polychlorinated dibenzo-p-dioxins (PCDDs) Polychlorinated dibenzofurans (PCDFs)	Type II Diabetes Neurotoxicity Endocrine and hormonal disruption Reproductive and development complications	Correct demolition of former buildings that contain such toxic substances since their prohibition Chemical remediation
Fire resistant and retardant materials, including carpet, mattresses, polyurethane upholstery foam, computer and television surrounds	Brominated Diphenyl Ethers Brominated Flame Retardants	Endocrine and hormonal disruption Developmental and cognitive deficiencies Behavioural changes Hearing abnormalities Cancer	Materials with natural fire retardant and resistant properties, such as wool Fibreglass Clay Stone, concrete, brick
Textiles Cotton	Organochlorines Organophosphates Pyrethroids	Cancer Developmental and reproduction complications Asthma Nervous system and behavioural issues	GOTS-certified products, not just organic cotton Hemp

10.2 PRINCIPAL CONSIDERATIONS

On reflection of this thesis, there are a number of significant overarching principles. A synthesis of these include:

1. We are being exposed to a poisonous concoction of toxic substances within the interior built environment, some naturally-forming and many others synthetic, but it is the dose that is critical.
2. We are primarily being exposed to toxic substances through dermal absorption, inhalation, and ingestion of dust and food and water sources.
3. With decisions seemingly being formulated to allow for efficiency in mass production of building materials (and associated substances), for example, cost effectiveness, ease of access, and ease of replacement (Marshall-Baker, 2010, p. 515), continual innovation is needed to find alternatives to these toxic substances so as to reduce the negative effect on human health.
4. The life cycle of such chemicals is also a critical factor that needs consideration when developing new alternatives, being sure to consider how it is made, whether the production process creates toxic emissions, what processes are involved in integrating chemicals into building materials, are there additional health implications from installation methods, what is the life cycle of such materials, can it be reused or recycled without contributing to further issues, does it need disposing of into land fill at the end of its life? What is the true cradle to cradle process?
5. Some toxic substances can remain in existence within the environment for decades after their purpose ceases, so as a society we need to rectify the use of harmful chemicals immediately and source healthier alternatives.
6. There is a risk in creating new alternative chemicals that pose less risk to human health. It can often be impossible to distinguish the long-term effects of chemicals and substances without having the ability to research and examine them over long term periods.

10.3 LIMITATIONS

This research has been limited by 3 main factors. The first limitation is the lack of Australian regulations to date, that is directly associated with the health implications of the 6 selected toxic substances. The majority of the governmental research and regulations was from the US, which suggests that further research and action is required by the Australian government to monitor and/or restrict the use these selected toxic substances, in the interest of the health of Australians. And similarly, while the research was evidence-based, peer reviewed, relevant and recent, most was

published by researchers internationally. However, this insight into global evidence did assist in understanding how pervasive these 6 toxic substances are and how their health implications don't discriminate between cultures.

Secondly, the toxic substances selected to be each individual case are considered some of the most prevalent contaminants in the modern-day interior built environment (Brambilla & Sangiorgio, 2020, pp. 1–14; Fang et al., 2019, pp. 309–318; Hwang et al., 2008, pp. 26–35), but given their chemical structure and subsistence it is very difficult to make common connections between them. For example, mould is a natural fungus that is a result of its ideal conditions, formaldehyde is both a synthetic and natural chemical, and polybrominated diphenyl ethers are primarily a manmade compound. To try to formulate the research into a somewhat comparable structure, objectives were created to ensure the same information was being sought across each case, refer to 'Appendix A: Chapter Template.'

And thirdly, as the researcher's expertise lies within the field of design. While my practice frequently concerns healthcare design, my expertise is not that of a trained health practitioner. Therefore, the connections that are conveyed are formulated from my interdisciplinary research so as to build the necessary familiarity and understanding as much as possible. Over the years, the researcher has studied various short courses and achieved various Statements of Attainments that covered topics such as physiology, anatomy and chemistry, which have definitely assisted in the understanding the potential connections between health and the interior built environment, though experience beyond that within the medical and health industries has not occurred at the time of writing this. The nature of this study has been exclusively an extensive literature review of pre-existing data. So, while the aim was to theoretically formulate connections between the interior built environment and the deleterious impact that the presence and concentrations of toxic substances can have on human health, it has posed gaps in research or evidence required to definitively assess the impact that such substances can have on human health, not just an individual substances in designated conditions, but a realistic accumulative concoction of contaminants in an array of conditions. And of course, given our diverse environment, may be something that can never truly be examined.

10.4 FUTURE RESEARCH & RECOMMENDATIONS FOR PRACTICE

From this research, there are a number of considerations that require further research, including additional extensive analysis of what toxic substances we are continually being exposed to, what is their individual toxicity as well as an accumulated concoction toxic effect on human health, and

more importantly, why are such contaminants still in existence. Critical items that need to have greater bearing over our decisions include:

a) *Sustainability and the overall life cycle* of materials and products from raw material, to manufacture, to transportation, to construction, to occupation, to demolition, to recycle upcycle or disposal. These concerns for the overall life cycle should carry greater weight and be considered during the specification and design processes, and even when purchasing products. Brzyski et al. explains the importance of sustainability throughout the holistic building process, with the life cycle, utilisation and disposal being as critical as procuring constituents, the manufacture of the materials and appropriate processes after demolition (2019, p. 020005).
b) *Public knowledge of environmental issues* is critical to raise awareness of such dangers within our surrounding environment. A methodical understanding of how indoor environmental quality can affect health is vital for designers, building managers and even occupants. During extreme situations such as this current pandemic, systematic guidance is will provide a critical level of education on minimising risks within the internal built environment. The collaboration of designers, architects, building scientists, biologists, medical professionals, engineers and data analysts are required to form a largely interdisciplinary approach, in combination with large-scale and long-term assessments, experiments and interventions. Though notably, the extensive progression and investigation of regulations and standards is essential in developing this framework, in conjunction with a more holistic focus to also include the health of the building occupants (Awada et al., 2021, p. 107480). Some media outlets are considered to carry a level of bias, although these outlets are advantageous for expressing the development of social issues. Yet, despite ambiguities and information streams, decisions are subsequently created, and science is filtered down to the public through mainstream media (Comby et al., 2014, pp. 100–115). People have a right to personal choice, and with awareness, it can allow consumers to make their own informed decisions.
c) *Progressive governmental regulations* are required to continually stay up to date with current and emerging research, to ensure the best interests of society are being considered. Policies associated to building regulations often do not address public health evidence, which leads to a deficiency in the management of urban development. Building regulations must acknowledge health implications in future policies, it must incorporate and manage the key policies to protect all scales and phases of development within the construction industry, and also, building occupants need to better understand technological advancements in

> energy efficiency (Carmichael et al., 2020, pp. 137146–137146). While long-term implications are typically not identifiable initially, it is critical that these issues remain at the forefront. National building codes and State standards need to be continually reviewed to minimise the impact that the construction industry is having on human health, but also on the surrounding environment and future condition and diversity of the earth at a grander scale. Countries with government regulatory bodies that have enforced limitation on the use of certain toxic substances, thereby stimulating the advancement of safer alternatives should step up and assist countries who may not yet have regulated the use of toxic materials.

A concept in Toxicology first suggested by a Swiss physician, Paracelus (1493-1541) is that, "all things are poisons and nothing is without poison; only the dose makes a thing not a poison," (Chemical Safety Facts, 2021). And with a typical apartment dwelling in Australia in 2017 registering toxic levels within indoor air being 6 to 8 times worse than outdoors and indoor air pollution 4 to 6 times worse (Zhigulina & Chumachenko, 2017, p. 2), further research needs to be conducted to assess the harm being caused by common toxic substances and chemicals that are prevalent within the interior built environment. This gained insight then requires well informed, evidence-based decisions to be made at governmental levels to prohibit, regulate and monitor any use of toxic materials, whilst paving the way for healthier alternatives for both human health as well as the wider environment.

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APPENDIX A: CHAPTER TEMPLATE

- ***Introduction*** *of toxin:*
    a) Briefly introduce and identify the toxin (approx. 100 words)
- ***Objective 1 –*** *To examine the* ***history of the toxin or chemical*** *in relation to the interior purposes: (Approx. 1,200 words)*
    a) Interrogate the history of the toxin – How long has it been around, or been problematic?
    b) Why was it introduced, what is its purpose?
    c) Where does it exist, where is it commonly found?
- ***Objective 2*** *– Health implications of the toxin (approx. 2,000 words)*
        i. **How** does exposure occur?
    a) Is it achieved through the subsequent application of the toxin, or is it more in depth during its manufacture?
        i. **What** are the health effects?
- ***Objective 3*** *–* ***What*** *are the life cycle implications?* (approx. 1,000 words)
    a) *Investigate the life cycle of the toxin*, from manufacture to end of life, to determine the detriments of reusing and recycling its related products
- ***Conclusion****: To comprise recommendations or actions* (approx. 1,000–1,200 words)
    a) Investigate if there are potential healthier alternatives for future use, or for reducing exposure to it, to enhance wellbeing as opposed to harming it.

Chapter	Word Count
Preliminary Sections	2000
Introduction	6500
Literature Review	4500
Methodology	3000
Toxin 1	5000
Toxin 2	5000
Toxin 3	5000
Toxin 4	5000
Toxin 5	5000
Toxin 6	5000
Discussion/Conclusion	5000
References	9000
TOTAL WORD COUNT	60000